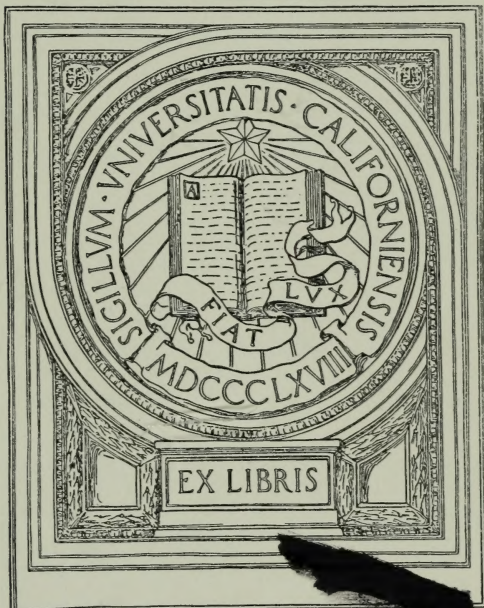
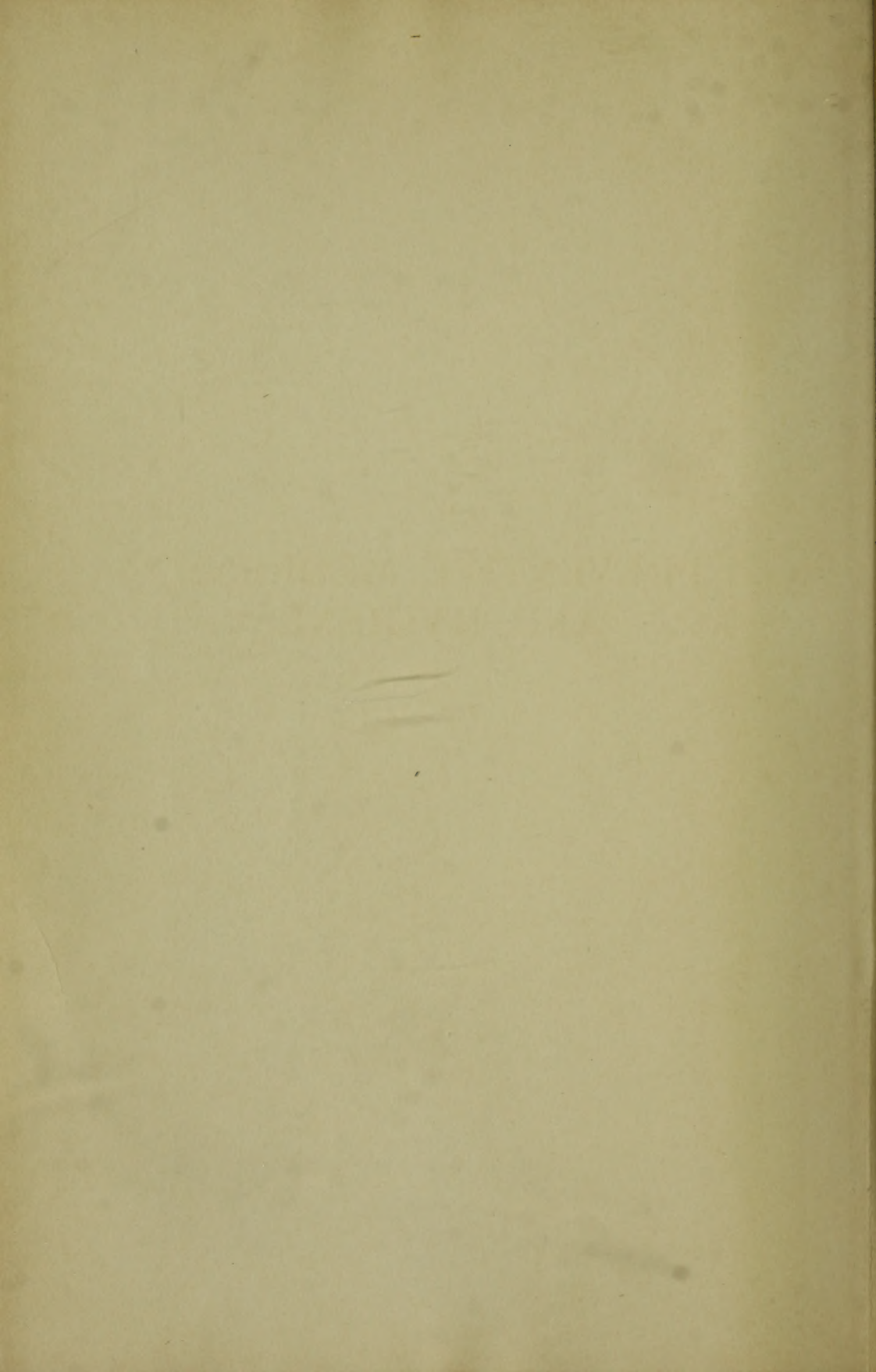


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PREVENTIVE MEDICINE AND HYGIENE

MILTON J. ROSEN

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CHICAGO, ILLINOIS
REPRINTED FROM THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION
PUBLISHED WEEKLY
CHICAGO, ILLINOIS
MAY 1925

PREVENTIVE MEDICINE AND HYGIENE

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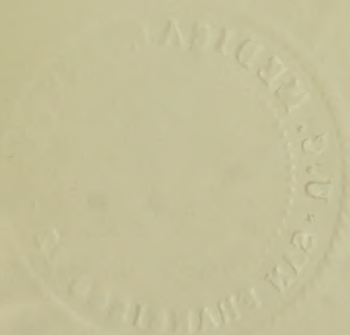
NEW YORK: THE LANCET
D. APPLETON AND COMPANY

PREVENTIVE MEDICINE AND HYGIENE

MILTON J. ROSENBAUM

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PRINTED IN THE UNITED STATES OF AMERICA

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PREFACE TO THE FIRST EDITION

This book is a collection of papers by a number of authors who have been working in the field of public health work. The book is intended to be a guide to the student and the worker in the field of public health work. The book is intended to be a guide to the student and the worker in the field of public health work. The book is intended to be a guide to the student and the worker in the field of public health work.

TO
MY WIFE

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NEW YORK

PREFACE TO THE FIRST EDITION

This book has been written in response to a demand for a treatise based upon modern progress in hygiene and sanitation. The work is planned to include those fields of the medical and related sciences which form the foundation of public health work. So far as I know, no other book on the subject covers the broad field considered in this volume. The progress in hygiene and sanitation has been so rapid that the subject of preventive medicine has become a specialty, and its scope has become so broad that the question throughout the making of this book has been rather what to leave out than what to include. The facts here brought together are widely scattered in the literature and many of them are difficult of access; they have been collected for the convenience of the student of medicine and the physician, as well as those engaged in sanitary engineering or public health work.

During twenty-three years of varied experience in public health work it has been my good fortune to have served as quarantine officer, in epidemic campaigns, in epidemiological investigations, and in public health laboratories, at home, on the continent, and in the tropics. The fruits of these experiences are reflected in this book, which may be taken as representing my personal views gained in the field, in the laboratory, in the classroom, and in administrative offices.

It is well-nigh impossible to prevent or suppress a communicable disease without a knowledge of its mode of transmission. This is the most important single fact for successful personal prophylaxis, as well as in the general warfare against infection; therefore the communicable diseases have been grouped in accordance with their modes of transference. Each one of the important communicable diseases is discussed separately in order to bring out the salient points upon which prevention is based. The classification adopted is believed to be unique and should prove helpful to those who are especially concerned in the prevention of infection.

The book may be considered in two parts, namely, that which deals with the person (hygiene) and that which deals with the environment (sanitation). The first part includes the prevention of the communicable diseases, venereal prophylaxis, heredity, immunity, eugenics, and similar subjects. The second part deals with our environment in its relation to health and disease and includes a discussion of food, water, air, soil, disposal of wastes, vital statistics, diseases of occupation, industrial hygiene, school hygiene, disinfection, quarantine, isolation, and other topics of sanitary importance, as well as subjects of interest to health officers. All the important methods used in public health laboratories are described.

To have made this book in monographic style with references to authorities for every statement would have resulted in an unwieldy work of impractical size and form. The textbook style has therefore been adopted and citation of authorities for facts that are now well established has been regarded as unnecessary. In this respect it may seem that I have given scant credit to many workers from whose writings I have borrowed results, thoughts, and sometimes words or even sentences. At the end of each chapter will be found a list of references to articles or books that I have especially drawn upon, and I desire to acknowledge my obligations to these sources as well as to refer the reader to them for further study of particular subjects. I have also drawn freely upon my own previous writings and those of my coworkers in compiling this book. The chapter on Disinfection is based upon my book entitled *Disinfection and Disinfections*, published by P. Blakiston's Sons & Co., Philadelphia, 1902.

I have received generous help from a number of friends and it is a pleasure here to acknowledge especially my obligation to Dr. David L. Edsall for reading and correcting the chapter on Diseases of Occupations, to Dr. John F. Anderson and Dr. Joseph Goldberger for revising the chapters upon Measles and Typhus Fever, to Prof. George C. Whipple for reading and improving the chapter upon Water, to Charles T. Brues for many suggestions in the section upon Insect-borne Diseases, and to Prof. W. E. Castle for a similar service with the section on Heredity. Dr. Charles Wardell Stiles has kindly furnished information concerning the relation of parasites to soil. I also desire to express my obligations to Prof. Arthur I. Kendall, Dr. Harold L. Amoss, Dr. Lewis W. Hackett, Prof. William D. Frost, and Miss Emily G. Philpotts.

It has been my object to give in this volume the scientific basis upon which the prevention of disease and the maintenance of health must rest. Exact knowledge has taken the place of fads and fancies in hygiene and sanitation; the capable health officer now possesses facts concerning infections which permit their prevention and even their suppression in some instances. Many of these problems are complicated with economic and social difficulties, which are given due consideration, for preventive medicine has become a basic factor in sociology.

BOSTON

M. J. ROSENAU.

PREFACE TO THE FIFTH EDITION

During the five years that have elapsed since the last edition, no epochal discoveries have been made to revolutionize the principles and practice of preventive medicine and hygiene. Nevertheless, so many advances have taken place in every subject that it became necessary largely to rewrite the text and entirely to reset the book; in fact, changes have been made on practically every page. On the other hand, several hundred pages in the former edition have been omitted in this one.

The new subjects considered comprehensively for the first time in this book are the psychoanalytic approach to sex hygiene, the prevention of cancer, the conservation of vision, periodic physical examinations, flukes and their relation to disease, granuloma inguinale, balantidial dysentery; also resuscitation, gas masks, statistical methods and practical points in public health administration.

The following topics have been largely rewritten: amebic dysentery, scarlet fever, sleeping-sickness, tularemia, pappataci fever, scurvy, rickets, pellagra; also maritime quarantine, sewage, garbage, pasteurization, personal hygiene, oral prophylaxis and the teeth. The sections on venereal diseases, sex hygiene, the social problem, heredity and eugenics have been expanded and revised. The section on mental hygiene is entirely new.

I am indebted to many friends for expert help in many subjects in which they are acknowledged authorities. This coöperation has been of material service in helping to make the text authoritative and in keeping it up to date. Individual acknowledgments of special assistance are given on page xi.

M. J. ROSENAU.

HARVARD MEDICAL SCHOOL
BOSTON



ACKNOWLEDGMENTS

I have had the generous coöperation of many friends who have helped make the book authoritative and bring the text up to date. I am especially indebted to the following for help in the subjects named:

MR. M. C. WHIPPLE, Water
MR. PHILIP DRINKER, Air
DR. A. C. REDFIELD, Mountain Sickness, Physiology of Respiration
DR. J. D. LONG, Maritime Quarantine
DR. J. H. WAITE, Conservation of Vision
DR. L. R. THOMPSON, Industrial Hygiene
DR. ALICE HAMILTON, Industrial Toxicology
DR. I. CORIAT, Psychoanalysis and Sex Hygiene
DR. J. R. MOHLER, Meat, Meat Inspection
DR. B. H. RANSOM, Animal Parasites
DR. C. A. BROWNE, Food Preservation
DR. TALIAFERRO CLARK, School Sanitation
DR. W. B. CASTLE, Heredity and Eugenics
DR. J. P. LEAKE, Smallpox and Vaccination
DR. E. FRANCIS, Tularemia
DR. JOSEPH GOLDBERGER, Pellagra
DR. G. W. MCCOY, Leprosy, Rats, Fleas and Plague
DR. S. B. WOLBACH, Typhus Fever, Fleas
DR. W. C. BOECK, Dysentery
DR. A. S. POPE, Scarlet Fever
DR. C. W. STILES, Hookworm Disease
DR. W. G. SMILLIE, Hookworm Disease, Malaria, Mosquitoes
DR. W. H. FROST, Pneumonia, Influenza, Common Colds
DR. A. F. HESS, Scurvy, Rickets
DR. E. R. BALDWIN, Tuberculosis
DR. M. J. WHITE, DR. C. M. SMITH, Venereal Diseases
DR. L. O. HOWARD, DR. C. T. BRUES, Insect-borne Diseases
DR. W. L. AYCOCK, Infantile Paralysis
DR. ROBERT OLESEN, Goiter

Dr. Reginald M. Atwater gave me material help throughout the book, and I am also grateful for particular assistance from Dr. D. L. Augustine, Dr. K. D. Blackfan, Dr. L. D. Felton, Mr. H. C. Lythgoe, Dr. C. E. North, Dr. F. Simpson, Dr. J. W. Schereschewsky, and Dr. Benjamin White.

The author is again indebted to Miss Mae C. Moran, his efficient secretary, who looked after the book through the press and also made the index.

M. J. ROSENAU.

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PREVENTIVE MEDICINE AND HYGIENE

SECTION I

PREVENTION OF THE COMMUNICABLE DISEASES

CHAPTER I

DISEASES HAVING SPECIFIC OR SPECIAL PROPHYLACTIC MEASURES

VACCINATION AND SMALLPOX

The prevention of smallpox depends upon vaccination. Vaccination was the first specific prophylactic measure given to man; it produces an active immunity to smallpox. Despite its importance and great practical value, there is much antivaccination sentiment due to ignorance or misconstruction of the facts.

Smallpox was once the most prevalent and dreaded disease in the world. Before the days of vaccination scarcely five persons out of a hundred escaped it, and about a quarter of those who took it died. Many of those who recovered were mutilated or maimed for life. It is still unduly prevalent. During the ten years 1913-1922 there were 579,610 cases of smallpox reported in the United States. This means that one in about two hundred had this readily preventable disease during this period. In 1923 the rate for smallpox in the United States was 27.1 per 100,000. This was exceeded by only three countries in the world of which we have record; Switzerland (55.0), Russia (42.9) and Greece (32.9), practically all among the unvaccinated.

Important Historical Facts.—The credit of giving vaccination to the world is due to Jenner, who proved through carefully planned experiments that cowpox protects against smallpox. This fact had been familiar to the farmers and folk of England as a vague tradition for a long time. Jenner, while at Sudbury, heard a milkmaid say, "I cannot take smallpox because I have had cowpox." This remark made a strong impression upon the young medical student.

Benjamin Jesty, a Dorchestershire farmer, in 1774 successfully vaccinated his wife and two sons. Plett, in Holstein, in 1791 also successfully vaccinated three children. It was Jenner, however, who through logical and scientific

methods proved that a person who has had the mild disease, cowpox, enjoys protection against the serious and often fatal disease, smallpox. Waterhouse and others soon repeated and corroborated Jenner's experiments and helped to establish the soundness of his conclusions.

Jenner made his crucial experiment May 14, 1796, when he transferred vaccine matter from the hand of a dairy maid (Sarah Nelms), who had scratched her hand with a thorn and "was infected with the cowpox from her master's cows," to the arm of a boy about eight years old (James Phipps). A typical take followed. "In order to ascertain whether the boy, after feeling so slight an affection of the system from the cowpox virus, was secure from the contagion of the smallpox, he was inoculated the first of July following with variolous matter, immediately taken from a pustule. Several slight punctures and incisions were made on both arms, and the matter was carefully inserted, but no disease followed. The same appearances were observable on the arm as we commonly see when a patient has had variolous matter applied, after having either the cowpox or the smallpox.¹ Several months afterward he was again inoculated with variolous matter, but no sensible effect was produced on the constitution."

In addition to such direct experimental proof, Jenner inoculated smallpox matter into ten persons who had at some previous time contracted cowpox and found that they were resistant to smallpox. In justification of such human experimentation it should be remembered that at that time the inoculation of smallpox matter into healthy individuals was an acknowledged method of preventing that disease. Jenner himself was inoculated when a boy. The question of "inoculation" (with smallpox) as contrasted with "vaccination" (with cowpox) will be discussed presently.

With such proof as this Jenner put a vague belief upon a scientific basis. He demonstrated that cowpox is a local and mild disease in man, that it may be readily transferred from man to man, and that it protects against smallpox. The chain of evidence was complete, but he first proved his thesis to his own satisfaction before he gave it to the world. He said: "I placed it on a rock where I knew it would be immovable before I invited the public to take a look at it." Jenner in 1796 presented the results of his observations to the Royal Society, of which he was a Fellow, but the paper was refused. He then published it in 1798 as a book, modestly entitled, "An Inquiry into the Causes and Effects of the Variolæ Vaccinæ, a Disease Discovered in Some of the Western Counties of England, Particularly Gloucestershire, and Known by the Name of the Cowpox." Every student of preventive medicine should read this brief "inquiry" in the original. It is one of the medical classics. Time has proved it to be a model of careful observation and logical presentation, showing self-restraint and moderation of an observant, imaginative, and judicial mind.

¹ This keen observation shows that Jenner was familiar with the form of modified take known as the immediate reaction, recently rediscovered and now explained in terms of anaphylaxis (page 7).

Benjamin Waterhouse, the first professor of Theory and Practice of Physic in the Harvard Medical School, early became convinced of the value of Jenner's demonstration and obtained some vaccine virus on threads from abroad. On July 8, 1800, he vaccinated his son, Daniel Oliver Waterhouse, then five years old. This was the first person vaccinated in America. After his son and two slaves were successfully vaccinated with cowpox, they were inoculated with smallpox with negative results.

In Boston on August 16, 1802, nineteen boys were vaccinated with the cowpox. On November 9th twelve of them were inoculated with smallpox; nothing followed. A control experiment was made by inoculating two unvaccinated boys with the same smallpox virus; both took the disease. The nineteen children of August 16th were again inoculated with fresh virus from these two boys with negative results. This is one of the most crucial experiments in the history of vaccination, and fully justified the conclusion of the Board of Health: "*Cowpox is a complete security against the smallpox.*"

Thomas Jefferson helped materially to spread the new doctrine in this country, and, in 1806, in writing to Jenner, said: "Future nations will know by history only that the loathsome smallpox has existed and by you has been extirpated." This prophecy has by no means been fulfilled—though eminently possible.

VACCINATION

Definition.—Vaccination consists in introducing vaccine virus into the skin with the object of inducing cowpox (*vaccinia*) in order to prevent smallpox (*variola*). Vaccine virus contains the active living principle of cowpox. The vaccination or "take" should be regarded as successful only when the manifestations run a characteristic course. Primary takes occur in persons who are susceptible; modified reactions are indicative of immunity and occur in persons who have previously been vaccinated or who have had smallpox.

For over eighty years vaccination (from *vacca*, a cow) was a specific term limited to the introduction of the virus of cowpox into the skin in order to induce vaccinia and prevent variola. Since the time of Pasteur,² however, the term has been used in a generic sense to include the introduction of many different substances in many different ways and for many different purposes.

Course of the Eruption.—*First Vaccination (Primary Take).*—The period of incubation is about three days, when a papule appears upon the skin where the vaccine virus was inserted. The papule develops into a vesicle and this in turn into a pustule, which dries, crusts and rapidly heals. The papular, vesicular and pustular stages follow each other in orderly succession.

The *papule* is small, round, flat, bright red and hard, but superficial.

² Pasteur said of his immunization against anthrax and chicken cholera: "I have given to the word vaccination an extension which I hope science will consecrate as an homage to the merit and immense services rendered by Jenner." Valery-Radot, *Life of Pasteur*, American edition, p. 332.

About the fifth day the summit of the papule becomes vesicular. By the sixth day the papule has changed into a *vesicle*, which is at first clear and pearl-like. It is surrounded by a narrow, deep red and swollen ring, which about the end of the seventh day begins rapidly to grow wider, and is called the areola. This gives the picture of the "pearl upon the rose leaf," which constitutes the true Jennerian vesicle. On the seventh day the vesicle is typical and charac-

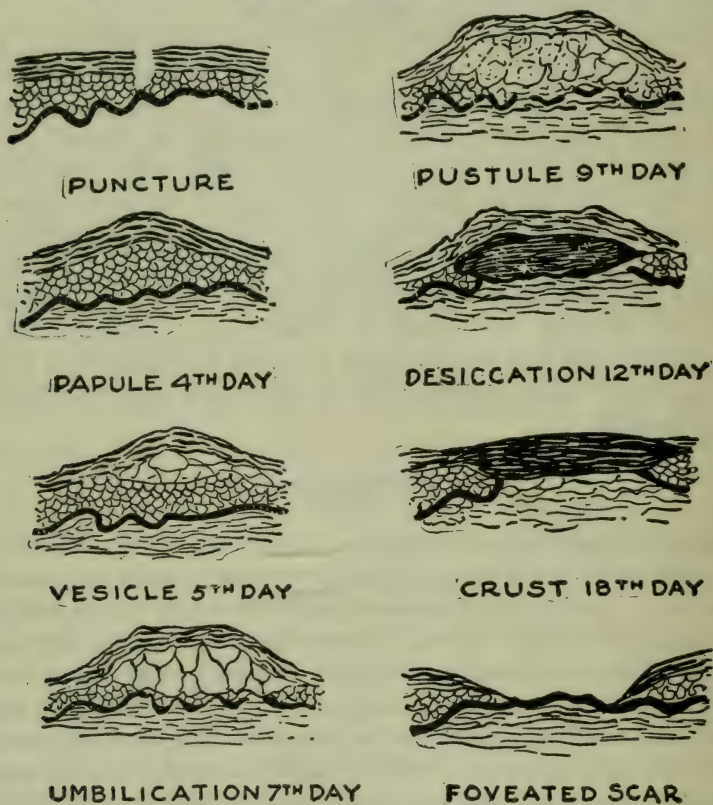


FIG. 1.—THE COURSE OF THE ERUPTION (Diagrammatic).

teristic, round or oval, flat on top, umbilicated, and clear or whitish. It is multilocular; if pricked with a pin or accidentally opened, it collapses slowly. By the eighth day it turns yellowish and the middle is fuller, following which the so-called second umbilication develops. The vesicle gradually turns into a *pustule* by the ninth day. Meanwhile, the areola deepens, widens and may be swollen. The skin feels hot, is painful and the axillary glands become enlarged and tender. By the twelfth day the local eruption has reached its height, following which the areola rapidly fades and the swelling subsides. The pustule soon dries, leaving a brown, wrinkled scab, which finally drops off about the twentieth day. It should never be removed, as it forms the best dressing. The scar is at first red, and finally turns white with the pits or foveations so characteristic of the true pock mark.

The course of vaccinia develops in four periods of about three days each: (1) The period of incubation; (2) the papule requires about three days to develop into the vesicle; (3) the vesicle in turn requires about three days to change into a pustule; and (4) the pustule finally requires about three days to reach full maturity.

The course of the eruption is singularly constant. Small variations, how-

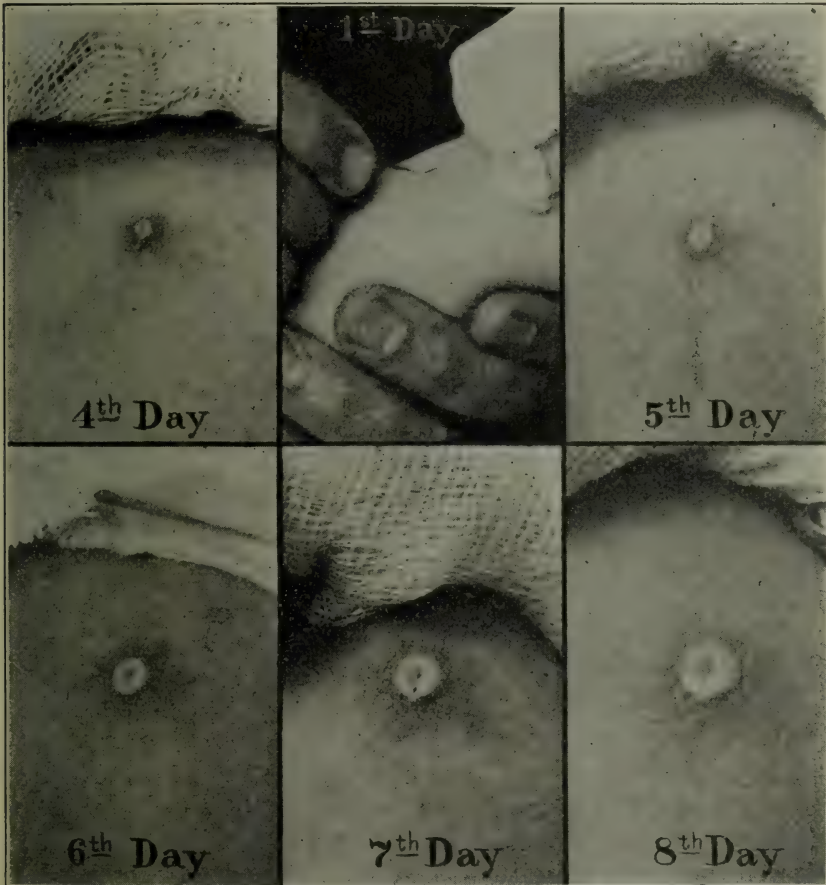


FIG. 2.—VACCINIA.
Course of the Eruption.

ever, occur. Thus, sometimes the period of incubation is delayed, or the height of the take may come before, but rarely after the twelfth day. The best protection is afforded by typical takes, and no reliance whatever should be placed upon atypical manifestations which are occasionally observed.

General Symptoms.—These vary. Malaise, loss of appetite, sometimes nausea and vomiting, headache, pain in the muscles of the back, and other indications of a mild febrile reaction appear about the seventh day, and soon cease. The temperature may go to 38° or 38.5° C. as the vesicle ripens. The

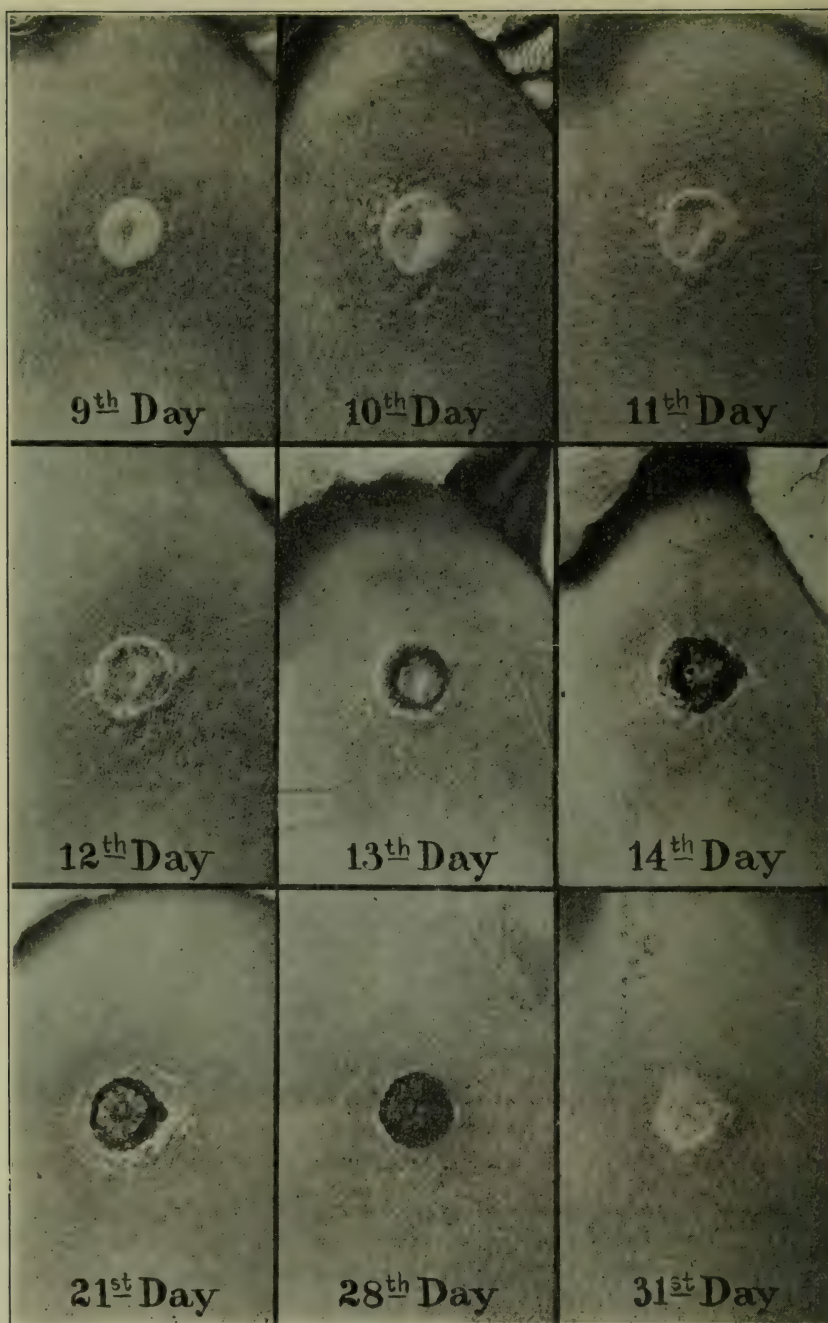


FIG. 3.—VACCINIA.
Course of the Eruption.

febrile reaction bears no special relation to the size and number of the vesicles or to the areola. The regional lymph-nodes become enlarged and tender about the time the pustule forms. The nitrogen elimination increases about the tenth day for a short time. The blood changes resemble those of smallpox, an early leukopenia and secondary leukocytosis.

REVACCINATION

Modified Takes.—The clinical picture of secondary vaccinations may be quite different from the typical take following a primary vaccination. By secondary vaccinations is meant vaccinations performed at any time after a successful take. Secondary vaccinations often run an accelerated, milder or modified course with shortened periods of incubation. These altered reactions were known in the time of Jenner, but were lost sight of until recently rediscovered and their significance realized from studies in anaphylaxis.

The reactions after revaccination are (1) primary; (2) immediate; (3) accelerated.

The Primary Reaction.—A revaccination may run an unaltered course resembling a primary take in all respects, showing that immunity to cowpox has disappeared. The period of incubation is about three days, and the eruption reaches its height about the twelfth day.

The Immediate Reaction.—The immediate reaction is characterized by a papule which appears in about twenty-four hours. This modification is also called the reaction of immunity. While the reaction is in fact probably immediate, it does not become apparent as a papule until about twenty-four hours. The papule remains a papule and does not develop into a vesicle. It grows slightly in size for two or three days and very gradually fades within two or three weeks. The immediate reaction resembles a skin tuberculin test in many respects. Virus of moderate potency may give small takes which should not be confused with the immediate reaction, as they have longer periods of incubation. A control scratch is helpful in interpretation.

The Accelerated Reaction.—The period of incubation is shorter than in the primary take. The papule usually appears in about thirty-six hours. It develops much more quickly than in primary takes, the height being reached before the eighth day, and it rapidly subsides. The accelerated reaction indicates partial protection.

Interpretation.—The difference between primary, accelerated and immediate reactions is determined by the period of incubation and also by the time at which the areola reaches its largest diameter. Every gradation is seen between an immediate reaction and a primary take.

Modified takes are expressions of immunity. They are an index of protection and its degree. They also signify that the prior vaccination was successful. Modified reactions are positive takes and should be recorded on vaccination certificates as such, and the nature of the modification indicated, whether immediate or accelerated. Similar modified takes are also seen in those who have had smallpox.

The nature of the modification varies with the interval of time between the first and second vaccinations and with the capacity of the individual to retain immunity. If the interval is less than two years, revaccination is apt to give an immediate reaction; between two and ten years there is usually an accelerated reaction; after longer periods of time revaccination is apt to run the course of a primary take. This correlation applies only in the aggregate, for individual cases vary greatly. These modified reactions may be explained in terms of anaphylaxis. They not only indicate protection, but increase it. Vaccination not only produces immunity, but is also a measure of it. Therefore, persons may be released from quarantine as soon as they show a modified take, especially the immediate reaction indicating protection.

METHODS OF VACCINATION

The operation of vaccination consists in introducing vaccine virus *into* the skin. Under no circumstances must the vaccine virus be placed under the skin, or subcutaneously. The technic of vaccination may be compared to the transfer of a culture in a bacteriologic laboratory. Precisely similar precautions to prevent contamination must be used in both cases. Vaccination is a minor surgical operation. No person unfamiliar with surgical cleanliness should be permitted to perform this "little" operation. The vaccine may be introduced in many ways: the best for general use is the scratch method.

Jenner used punctures or short incisions. Later, blisters were raised upon the skin and the virus rubbed into the abraded surface. The incisions were increased in number, and then cross scratchings or scarifications were made. Finally, the scratch method came into general use.

Puncture (*The Tattoo Method*).—Puncture consists in pricking the skin with the point of a needle moistened with the virus. This method of vaccination is theoretically the best, but one puncture is apt to miss fire, and even six or eight punctures cannot be depended upon. It has therefore been necessary to adopt the method of multiple punctures, which is done as follows:

Place a drop of vaccine virus upon the prepared site. Hold the arm horizontal with the skin taut by grasping the arm from below with the left hand. A sharp, sterile needle is now held between the fingers of the right hand so that the needle is parallel to the skin and pointing to the operator's left. The side of the needle point is then pressed into the drop of vaccine virus about fifteen or thirty times, the needle being lifted clear of the skin each time. The motion should be quite perpendicular to the skin and needle and not in the direction of the needle. In this way the elasticity of the skin will pull a minute portion of the epidermis over the point of the needle at each pressure, so that the virus is carried into the layer of epithelial cells. The total area of punctures should never be greater than one-eighth inch in diameter, and should not be deep enough to bleed. Immediately after these superficial pricks have been made, the remaining virus is wiped off the skin with sterile gauze and the sleeve pulled down. When properly done the per-

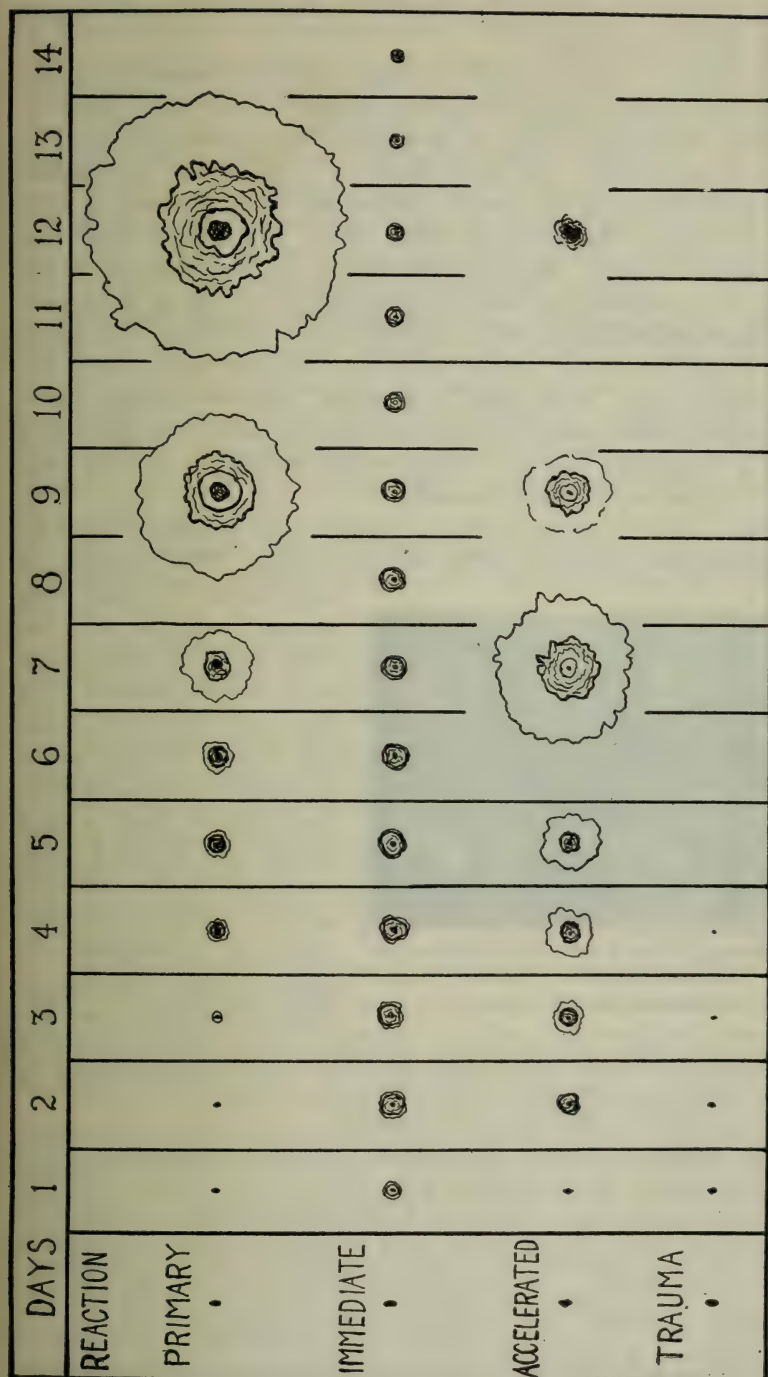


FIG. 4.—VACCINATION WITH COWPOX. PRIMARY AND MODIFIED REACTIONS.

Primary Vaccination.—Course from day to day. Incubation three days. Reaches its height about the twelfth day.

Immediate Reaction.—Incubation twenty-four hours, or less. The papule does not develop into a vesicle and pustule, but remains a papule and disappears slowly—several weeks.

Accelerated Reaction.—Incubation about thirty-six hours. Reaction reaches its height about the seventh day. Intermediate grades occur.

centage of takes is equal to that of the scratch method and a single vesicle appears at the point of insertion.

The disadvantages of this method are that without demonstration and practice the technic of applying just the proper pressure is not easily acquired, and further that without due care an area larger than one-eighth inch in diameter may be punctured. The advantages are its mildness, painlessness and rapidity, and when properly done a control site is not necessary, since the evidence of trauma disappears in about twelve hours, well before the immediate reaction appears. Another advantage is the fact that the excess virus is wiped off immediately so that the uselessness of a dressing is obvious.

Intradermal vaccination consists in injecting the virus between the layers of the skin. About 0.1 c.c. of a 1:40 dilution is injected with a very fine needle quite superficially. It is really a modified puncture, except that a much larger amount of the virus is introduced into the skin. This method is not commended for general use, for it requires special skill to prevent some or all of the virus getting in subcutaneously.

Scarification.—Scarification consists in producing an abraded surface by multiple scratches close together, or by cross scratching. The objection to

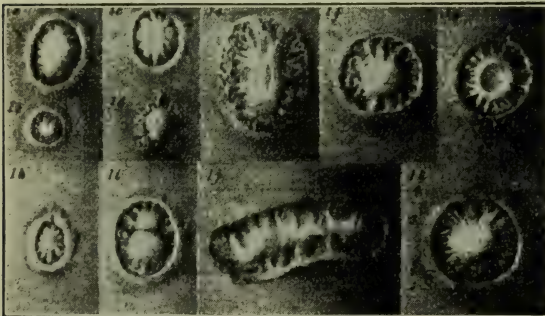


FIG. 5.—VACCINATION SCARS PRODUCED BY METHOD OF SCARIFICATION.

Note unnecessary central scar.

scarification is that this method produces a relatively large abraded surface which is soon covered by a dry, hard crust of serum and blood through which the eruption cannot pierce. The vesicles form a ring around the scarified area, leaving a central irritated wound, inviting infection. Anaërobic conditions are produced under the crust or scab which forms over the

scarified surface, favoring infection with tetanus. This scarified area also leaves an unnecessary and unsightly scar (Fig. 5). The method should be prohibited. It is actually forbidden in Germany.

Scratch or Incision.—This is the method I use and recommend. Incision is the only method of vaccination permitted by the laws of Germany and recommended by the Local Government Board of England. A scratch or incision, if properly done, consists really of a continuous series of punctures and serves the same purpose. Incision may be made with the point of any sharp instrument. It should not be deep enough to draw blood, but a few drops do no harm. I prefer to scratch with a sterile needle. The method has manifest advantages. It is quick, easy and produces typical takes with no scarring except one desirable pock mark for each vesicle. It has all the advantages of puncture and none of the disadvantages.

Number and Length of Incisions.—This has a bearing upon the probability of the take as well as the protection. A relationship probably exists between the number of vesicles and the degree and length of immunity (see page 15). The German regulations of 1899 require at least four incisions, each one centimeter long and two centimeters apart. The Local Government Board of England directs that four vesicles should be produced, and that the total area of the vesicle formation shall not be less than one-half a square inch.

My own practice now is to make one incision about one-eighth inch long. A control scratch is not necessary, but is useful for purposes of teaching, especially for demonstrating the immediate reaction.

The Best Place to Vaccinate.—The outer surface of the left arm at about the insertion of the deltoid is the most convenient for the operator and the patient. This is the original site selected by Jenner. It is easily kept cool and dry, and is less likely to have severe glandular reactions than other points. The skin here is easily made taut during the operation by grasping the under side of the arm. Inspection of the course of the eruption is also facilitated.

Any part of the skin or exposed mucous membrane is susceptible; a take will follow the accidental insertion of the virus into the conjunctiva, the lip, etc.

The leg is sometimes selected to avoid disfigurement. With small insertions on the arm by the scratch or puncture method, disfiguring scars do not result. To the sanitarian the typical foveated pock of vaccinia is a sanitary dimple. The leg is more exposed than the arm to moisture, street dust and traumatism and therefore to complications. Dock refuses to vaccinate on the leg unless the patient will stay in bed until the vesicle heals. With babies in diapers and with young children it is exceedingly difficult to keep these parts clean. If the leg is selected, the vaccination should be done on the outer surface of the calf below the head of the fibula, and not on the thigh.

The Operation.—Wash the skin with soap and water, follow with acetone, ether or alcohol, and allow to dry. Do not use denatured alcohol, iodine or other germicides, for they are apt to injure the virus and prevent successful takes. Acetone is an efficient cleanser, is cheap and evaporates rapidly.

Handle the capillary tube with strict aseptic precautions and place its contents upon the skin in one small droplet. With a sterile needle make a scratch starting in the droplet. Rub the vaccine virus gently into the scratch with the side of the needle about five or six times. Do this at once before the serum in the scratch dries. If a second droplet is applied, the two scratches should be about an inch apart. The excess vaccine virus may then be wiped off with sterile gauze, and the sleeve pulled down.

General Principles.—The modern tendency is towards smaller insertions and more frequent vaccinations. This is a sensible and practical trend. The smaller reactions produce a satisfactory immunity, even though not quite as durable as the larger takes. In fact, the immunity retained after many

years by many vesicles is not sufficiently greater than the protection afforded by a single vesicle to justify the difference. The important principle is time between vaccination and exposure to smallpox rather than the size and number of the vesicles; hence, the importance of repeated small vaccinations at intervals of about ten years. Furthermore, there is a minimal risk of complications with the small takes; and finally, there is a minimal inconvenience, not only with the primary vaccination, but especially in the secondary vaccinations, which when oft repeated usually give modified reactions which pass almost unnoticed by the patient. This procedure should do much to allay the fears concerning vaccination and to eliminate its objections. One vesicle is better than no vaccination at all, even though the protection is not quite as strong nor as long.

The present practice is to use no method of insertion larger than one-eighth inch in any direction. With fully potent virus on first vaccination this gives a vesicle at least three times as large as the insertion. The take following a scratch one-eighth inch long corresponds approximately in size to the tattoo method.

When to Vaccinate.—The best time to vaccinate is in infancy at about the sixth month, but avoiding the summer. There are no contra-indications to vaccinating babies immediately after birth, but takes are somewhat less likely than at six months. Vaccination at about the age of six months avoids the periods of teething and of artificial feeding, and usually proceeds with little or no fever or discomfort. Moreover, in infants the general symptoms from first vaccination are usually much milder than in older children and adults, and complications are practically eliminated. This alone is an important reason for advising parents that the first vaccination be performed early in life.

The fact that the immunity wears off after a number of years makes it necessary to practice revaccination in order to afford a continuous protection. There is some difference of opinion as to just when it is best to vaccinate the second time. It may be done before entering school. After this vaccinations may be performed every ten years, and whenever there is particular danger of exposure to smallpox. If revaccination is frequent enough, the individual will be protected throughout life without ever having a severe take. Where vaccination is compulsory and practically everybody is vaccinated, experience indicates that two vaccinations are enough for all practical purposes to control smallpox.

Summer is not so favorable a time to vaccinate as spring, fall or winter. Not only does the virus deteriorate quickly on exposure to warm temperatures, but there is greater danger from softening the vesicle and crust by perspiration or swimming.

All persons exposed directly or indirectly to smallpox should at once be vaccinated and revaccinated until only an immediate reaction is obtained unless they have had smallpox.

Precautions.—Keep the vaccine tubes protected and cold until the mo-

ment of using. Avoid the action of direct sunlight during or immediately after vaccination. Keep the vaccinated area dry, cool and clean. Bathing need not be omitted nor any of the ordinary occupations, but care should be taken not to soften the crust with water or sweat. Unnecessary use of the arm must be guarded against, as this increases the congestion, inflammation and chances of infection.

No dressing is necessary as long as the vesicle remains unbroken, but several layers of dry sterile gauze, attached to the inside of the sleeve rather than to the skin, do no special harm provided they are renewed frequently. Shields and pads of any sort are unwise because they act like a poultice and favor softening and breaking down of the vesicle.

The best dressing is the unbroken skin, and then the crust or scab which naturally forms. If the pustule breaks, or the crust comes off, or the take shows indications of secondary infection, frequent dressing with an active germicide is indicated. It is not good surgical practice to bind up any actively suppurating wound for more than twenty-four hours.

The site should be inspected at least on the second, seventh and eleventh days after vaccination to determine whether the take is running a characteristic course.

The Immunity.—The immunity appears about the eighth day of the vaccination. Layet puts the point of safety at the ninth day, Burckhard at the eleventh. These data are based upon the early work with variolation, when persons were inoculated with smallpox at various periods following vaccination. Sacco got only a local eruption by inoculating smallpox on the eighth to the eleventh days, and none after that.

Vaccinia protects not only against smallpox, but also against cowpox. Curiously enough, the degree and length of immunity appear to be greater against smallpox than against itself. Persons who have had smallpox may often be vaccinated successfully, although sometimes they give accelerated rather than immediate reactions. It is irrational to attempt to fix a definite time for the duration of the immunity. This varies as in other infectious processes, and in this case shows considerable individual variation. It is known through experiment and experience that the immunity gradually wears off. The degree of protection is usually absolute for a few years, then slowly fades. Judged by the reactions following revaccinations, it begins to disappear in two years and in ten years may be completely gone. In many individuals one primary take protects for life; usually, however, only when supplemented by revaccinations which yield modified reactions. In this, as in other diseases, immunity is a relative term. Smallpox itself does not always protect against smallpox. Some people have two and even three attacks of smallpox.³ Such cases, however, are exceptional, and it is also exceptional to have smallpox occur in an individual who has been recently vaccinated, that is, within about seven years before the exposure to smallpox.

³ Jenner mentions "the lady of Mr. Gwinnett, who has had the smallpox five times."
—Baron, *Life of Jenner*, Vol. II, p. 265.

Careful statistics collected in Japan since 1879 show quite definitely the gradual diminution of the immunity, beginning with the second year after vaccination. Kitasato's table,⁴ based on 951 cases, is as follows:

<i>Successful Revaccination After</i>	<i>Per Cent</i>
1 year	13.6
2 years	32.9
3 years	46.6
4 years	57.3
5 years	51.1
6 years	63.8

Weil, in 1899, reported 72.5 per cent of successful revaccinations after seven years, 80 per cent after eight years, 85 per cent after nine years, and 88.6 per cent after ten years. German government reports show 90 to 93 per cent successful revaccinations after ten years. These figures need critical study, for they were for the most part collected before the early modified reactions in secondary vaccination were understood or even recognized. The successful revaccinations for the most part probably consisted of accelerated reactions. Judging by the high percentage of takes reported in first vaccinations, the revaccinations adjudged unsuccessful were mainly immediate reactions. Furthermore, it should be remembered that successful revaccination indicates loss of immunity to cowpox, not necessarily to smallpox, for vaccinia gives a stronger protection against variola than against itself.

It is commonly asserted that, if a revaccination takes, the subject was therefore susceptible to smallpox. While this is usually true with a primary take and even with an accelerated reaction, it does not necessarily follow. It is a still greater fallacy to state that, if a vaccination fails, the subject is therefore immune. This view may result in real harm. Vaccination may fail for many reasons—the operation may not have been properly done, or the virus may have been inert. Sometimes persons are unsuccessfully vaccinated three, four, or more times before a typical take is obtained.⁵

A modified reaction must be regarded as evidence of immunity. Thus, the immediate reaction is indicative of a high degree of immunity if fully potent virus has been properly applied, and the accelerated reaction indicates a partial immunity. No instance of natural immunity to cowpox has been conclusively demonstrated. Practically everyone is susceptible and shows a reaction when properly vaccinated.

The nature of the changes in the body which produce the immunity are not understood. In this sense vaccination is still an empiric procedure. We now know of many analogous instances, however, where an active acquired immunity is induced by means of an attenuated virus. The immunity produced by vaccine virus does not depend upon an antitoxin. The blood, however, contains specific antibodies, shown by the fact that the activity of vac-

⁴*J. Am. M. Ass.*, 1911, 56: 889.

⁵One of my cases gave a history of having been unsuccessfully vaccinated five times. The sixth attempt produced a typical primary take with twenty-one vesicles.

cine virus is destroyed when mixed with equal parts of blood serum from a calf two weeks after successful vaccination.

There appears to be a relation between the immunity conferred and the number of vaccination scars. There is also some evidence that the protection is directly proportional to the area of the local eruption. The data contained in the final Report of the Royal Commission on Vaccination is summarized in the following table:

MORTALITY OF POSTVACCINAL SMALLPOX IN RELATION TO THE NUMBER OF SCARS

Number of Scars	3,094 Cases* (1836-1851) Per Cent	10,661 Cases* (1852-1867) Per Cent	6,839 Cases † Per Cent
none	21.7	39.4	..
1	7.6	13.8	6.2
2	4.3	7.7	5.8
3	1.8	3.0	3.7
4	0.7	0.9	2.2

* *Final Report of the Royal Commission on Vaccination*, 1896, paragraph 291. Thorne, from data collected by Marson.

† Same Report, paragraph 293. Summary of cases apart from those of Marson.

This varicidal property of the blood is believed to be a lytic process. The blood also has other antibody activity, producing agglutination, precipitation and complement fixation.⁶

VACCINIA AND VACCINE VIRUS

Vaccinia, or *cowpox*, is an acute specific disease which runs practically the same clinical course in all susceptible species. After an incubation period of three days the local eruption begins as a papule which soon develops into a vesicle, and later into an umbilicated pustule. Surrounding the vesicle is a reddened, inflamed, and tender areola. The neighboring lymph glands are swollen and tender, and there may be slight fever lasting several days. The pustule dries, leaving a crust or scab, which comes away, disclosing a typical foveated or pitted scar.

The eruption is always local and confined to the site of the vaccinated area; the constitutional symptoms are always mild. *Vaccinia* or *cowpox* is a benign disease; when uncomplicated, it has never been known to cause death or leave any unpleasant sequelæ.

Is Cowpox Modified Smallpox?—The unity or duality of these two diseases has been the subject of much contention. Jenner considered cowpox to be modified smallpox. The successful experiments in Germany, England, and this country, in which smallpox has actually been modified by passing variolous matter through calves, has demonstrated that we are dealing with two forms of one disease. When smallpox is thus converted into cowpox, it remains fixed as such, and never reverts to smallpox. Twenty-nine separate

* M. H. Gordon, Medical Research Council, Spec. Rep. Series, No. 98, 1925.

records of successful modification of smallpox virus into cowpox are found in the literature; also a number of negative attempts. Some of the strains obtained in this way have been used to vaccinate with typical takes and adequate protection.

It seems likely that so-called casual cowpox had its origin from smallpox through accidental inoculation in milking cows by persons having or recovering from smallpox. Once started, the propagation of the modified virus from cow to cow would be comparatively simple.

Immunity as a general rule is quite specific; in fact, there is a general principle in immunity that acquired immunity is usually exquisitely specific. This lends countenance to the assumption that cowpox is a modified form of smallpox.

Spontaneous or Casual Cowpox.—The disease is said to occur “spontaneously” when its origin cannot be traced. Cowpox is usually transferred from cow to cow through milking. It may be started from a vaccinated person or modified from a case of smallpox. It is highly significant that casual cowpox was much more common when smallpox was much more prevalent. In 1866 an instance of casual cowpox was discovered in Beaugency, France, and this virus, known as the Beaugency strain, has since been widely used.

Vaccine virus is the living specific principle in the matter obtained from the skin eruption of animals having “vaccinia” or “cowpox.” Vaccine virus is obtained from calves, but may also be obtained from older cattle, from man, rabbits, buffalo, caribou, camels, and other mammals.

Seed virus may be obtained (1) from casual cowpox, (2) from smallpox modified by passage through calves, and (3) by retrovaccination. Each produces typical takes and adequate protection.

Human virus is no longer used on account of the risk of transmitting syphilis and perhaps other diseases peculiar to man. Another disadvantage is that it is not practicable to obtain the great quantities needed especially at the time of an epidemic.

Bovine virus has been used since the time of Jenner, but especially since 1891 when Copeman showed the possibility of purifying it. Bovine virus has the great advantage of being readily obtainable in any amount and when desired. It may be purified with glycerin and phenol, and it further totally eliminates the danger of conveying syphilis and other diseases peculiar to man.

Vaccine Lymph and Vaccine Pulp.—Vaccine *lymph* is the fluid contents of the vesicle or the serous fluid which exudes after the vesicle is broken. The *pulp* consists of the entire vesicle with its contents, which is scraped from the skin. The active principle of vaccinia is concentrated in the epithelial cells. The pulp is generally used because vaccinia is an epithelial infection and the pulp therefore gives a greater yield and has better potency.

Propagation.—In the propagation of bovine virus calves are preferred, because they are more manageable, the skin is more tender, and the eruption is therefore more abundant and typical. With young animals a milk diet may be used, which simplifies the problem of dust contamination from dry feed.

If hay or fodder is used, it should first be autoclaved. Either heifers or bull calves are suitable, although the former are preferred.

The animals are held in quarantine for seven days, under observation, to determine the absence of infections such as tuberculosis, anthrax, foot-and-mouth disease, tetanus, fever, diarrhea or skin eruptions of any kind.

Before vaccinating the calf it is carefully cleaned, and the site of the vaccination is shaved and prepared surgically, but without the use of strong germicidal solutions, for the reason that they are apt to destroy the activity of the vaccine virus. Cleanliness and asepsis are the watchwords. The area selected is usually the abdominal wall between the tip of the sternum and the groin, sometimes including the inner side of the thigh. The usual method is to make long, superficial incisions in the skin about one centimeter apart, and the seed virus is gently rubbed into these incisions. The calves must then be kept rigidly isolated in a special room, moderately lighted, free from dust, and screened to keep out insects. The temperature of the animal is taken several times daily and the eruption at each stage of the disease is closely watched and recorded.

The virus is usually taken from the animal about the fifth day. It is an advantage to take the virus early, in order to avoid contaminating infections which may occur when the vesicles mature. Vaccine virus taken after the eighth day may be unreliable. Jenner's golden rule was to take the virus before the areola appeared; that is, before the seventh day. Virus taken after the eighth day is apt to produce unduly inflamed or abortive vesicles, called spurious takes by the early vaccinators. Only typical and entirely characteristic vesicles should be removed. The vaccination site is scrupulously cleaned by repeated washing and scrubbing and the pulp is then removed by scraping the vesicles with a sharp spoon-curette. Before the virus is removed, the animal is chloroformed or killed to avoid pain, and an autopsy is done as soon after the virus is removed as practicable. If the autopsy shows any lesions indicating infections other than vaccinia, the virus is discarded.

Purification of Vaccine Virus.—Vaccine virus obtained from the skin always contains bacteria. The initial contamination may be lessened by care in propagation. The virus may be purified so that it contains no bacteria, or at least none pathogenic for man. In the United States vaccine virus is required to have fewer than fifty bacteria per dose, and these must be non-pathogenic, as determined by tests.

In 1891, Monckton Copeman⁷ made the important discovery that the virus may be purified and preserved with glycerin.⁸ The glycerin acts as a differential germicide, that is, it is comparatively harmless to the active principle in the vaccine virus, but destroys the frail non-spore-bearing bacteria. In time the virus itself succumbs. Vaccine virus, therefore, should not be used when too fresh nor when too old. Manufacturers in the United States date

⁷ *Tr. Internat. Cong. Hyg.*, 1891.

⁸ Glycerin also serves as a preservative for other filterable viruses, as foot-and-mouth disease, infantile paralysis, rabies, etc.

their products as potent for not more than three months "if kept below 5° C. (41° F.)."

Fifty per cent glycerin of the best quality is used. I have shown that no growth of bacteria, yeasts, or molds takes place in 60 per cent ⁹ glycerin. Two to four parts of 50 per cent glycerin are added to one part of the pulp by weight. The mixture is then thoroughly ground between sterile glass rollers in a special mill. The pulp should be thoroughly broken up and a uniform suspension obtained. The amount of glycerin added depends upon the consistency and character of the pulp. The only objection to adding more glycerin would be the greater dilution of the virus, and, therefore, a larger proportion of negative takes. A higher percentage than 50 per cent of glycerin soon renders the virus inert. The time required for the virus to ripen depends upon the temperature. Most of the non-spore-bearing bacteria perish in thirty days at 15° to 20° C. Approximately the same effect may be obtained at 37° C. in a few hours. At low temperatures the glycerin has practically no bactericidal effect. The process must always be controlled bacteriologically.

It is now the common practice to add 0.5 per cent phenol to the glycerinated virus. Phenol is also a differential germicide; it destroys non-spore-bearing bacteria, but has comparatively little effect upon the virus of vaccinia. Other substances for the purification of vaccine virus have been tried, such as potassium cyanid, brilliant green and chloroform, but with less success in practice.

Henseval and Convert ¹⁰ showed in 1910 that the virus multiplied in the testicle. Noguchi ¹¹ by painstaking methods obtained a bacteria-free vaccine virus which may be propagated in the testicles of bulls or rabbits. Human beings react to the testicular strain in an entirely typical manner, but this method has not been found practical for propagation on a large scale. Such virus rapidly loses potency. A similar difficulty has interfered with the use of Levaditi's neurovaccine prepared from the brains of rabbits.¹²

Fresh, Dried and Glycerinated Virus.—The fresh and the dried vaccine virus cannot be purified, and thus the glycerinated virus is preferable and the only form now in practical use.

The *fresh* virus was formerly used in arm-to-arm vaccination, and sometimes in direct transfer from calf to arm. This method, once popular and still used in parts of the world, should be abandoned for evident reasons.

Vaccine matter when *dried* remains potent a very long time, especially when dried, frozen, and protected from oxygen and light. Formerly, physicians preserved the dried crust from a typical take. When needed, small portions of this crust were ground, moistened and inserted into the skin.

The old-fashioned dry points were prepared by drying vaccine lymph on splinters of ivory and later on bone or glass. Dry points have no advantage and many disadvantages, especially that the dried virus upon such points

⁹ U. S. Hyg. Lab. Bull., No. 16, 1903.

¹⁰ Bull. Acad. roy. de méd. de Belg., 1912, 26: 251.

¹¹ J. Exper. M., 1915, 21: 339.

¹² Compt. rend. Acad. de sc., 1922, 174: 249.

cannot easily be purified. Further, the points are used as scarifiers and the method of scarification favors irritation and infection of the wound. Dry points practically always contain more bacteria than the glycerinated virus. For these reasons dry points are no longer permitted in interstate traffic in accordance with the federal regulations.

The best container for single doses of vaccine virus is a capillary tube. These are easily sterilized, filled and hermetically sealed. They should be wiped with acetone and broken with sterile gauze, and the contents can then safely be emptied by means of a small rubber bulb. When many vaccinations are to be done, it is preferable to have the virus in bulk.

The Government Control of Vaccine Virus.—By the law of July 1, 1902, the vaccine virus sold in interstate traffic in the United States must come from a licensed manufacturer. These licenses are issued by the Secretary of the Treasury only after a careful inspection of the plant, personnel, and product by a competent officer of the U. S. Public Health Service. The licenses are good for one year, and are reissued only after reinspection. The government regulations require each lot of vaccine virus to be examined carefully by modern bacteriological methods to determine the number of bacteria, and special tests must be made to determine the absence of pathogenic microorganisms. These tests include animal inoculations, as well as cultural methods. Special tests for each lot of vaccine must be made to determine the presence or absence of streptococci, tetanus spores, the gas bacillus, and other pathogenic microorganisms. The government does not guarantee the purity and potency of each package of vaccine virus, but through its inspections and frequent examinations of the virus on the market every confidence may now be had in the vaccine virus propagated by licensed manufacturers in this country.

Care of Vaccine Virus.—Vaccine virus should always be kept ice cold until used. The warmth of the doctor's pocket, the office desk or the druggist's shelf may be enough to seriously impair its potency in a few hours. The heat of the railroad car or post office may soon render it impotent. This explains the difficulty sometimes encountered in obtaining potent virus during the summer months. Ice-box storage is not enough. The packages of virus should be kept in small glass or metal containers in actual contact with a large volume of ice.

Freezing does not injure vaccine virus; in fact, virus kept below freezing may preserve its potency for years. Recent virus keeps better and gives more vigorous takes than old material. It is always advisable to keep a record of the name of the manufacturer and the lot number and expiration date found on the package. The handling of the container and capillary tubes should be governed by the principles of rigid asepsis.

Retrovaccination.—When vaccine virus is passed from calf to calf through a long series of transfers, it has a tendency to lose virulence and to give weak and imperfect takes. From a practical standpoint it is very important in propagating vaccine virus to maintain an active strain that gives characteristic takes. When the virus is persistently grown on one animal species, cer-

tain associated skin bacteria gain increased activity, and these may be repressed and even eliminated by changing the species from time to time. This is known as retrovaccination, and is usually carried on by transferring the virus from calf to child to calf; or, the bovine virus may be passed from man through rabbit, monkey or other susceptible animal back to the calf.

Vaccination of Exposed Persons.—The question frequently arises whether persons exposed to smallpox should be vaccinated. The effect of vaccination during the period of incubation of smallpox is very interesting, and may be summed up as follows:

Vaccination done at the beginning of the incubation period, in time to have the vaccine eruption reach maturity before the smallpox begins, will usually prevent or abort the disease. The best protection is attained when the height of the vaccinal "take" has been reached before the exposure to smallpox. If done about the sixth or eighth day of the period of incubation, the vaccination takes and may modify the severity of the smallpox. If the vaccination is done during the last stage (ninth to fourteenth day) of the period of incubation of smallpox, the two infections run their course side by side without influencing each other. Vaccination just before or during the primary fever of smallpox does not influence the disease.

As we can never be quite sure just what stage in the period of incubation a given case may be in, the rule is to vaccinate exposed persons. There may have been multiple or unknown sources of exposure. Furthermore, little harm will be done if it is too late and the vaccine eruption is added to the smallpox. Indeed, Hanna¹³ presents claims to the effect that there is evidence of mitigation of the severity of smallpox when vaccination is performed at any time after infection up to the day of onset and even afterward.

All persons in the diagnosis of whose cases there could be any doubt whatever should be vaccinated before they are sent to the smallpox hospital.

Contra-Indications.—There are no known contra-indications to vaccination. Infants may be vaccinated immediately after birth, although it is customary to wait until nutrition under breast feeding is well established. Pregnancy is not a contra-indication. During the smallpox epidemic in Detroit, 1924, all patients were vaccinated on admission to the Herman Kiefer Hospital, which is the contagious hospital operated by the Detroit Department of Health. In all, 3,346 persons were vaccinated, including 773 obstetrical cases and 676 newborn babies, 90 cases of erysipelas, 425 cases of diphtheria, 644 cases of scarlet fever, 368 cases of tuberculosis and others with measles, mumps, etc. The takes were typical and there were no untoward results.¹⁴ Denny and Hopkins¹⁵ showed that when lepers are vaccinated the reaction is more pronounced than in normal individuals. At the same time there is a flare-up of the specific lesions of leprosy, which, however, were in no case permanently aggravated; in fact, some showed actual improvement.

¹³ *Pub. Health*, 1910, 23: 351.

¹⁴ Personal communication from Carl E. Buck, Epidemiologist, Detroit Department of Health.

¹⁵ *U. S. Health Rep.*, 1922, 37: 3141.

In community protection, it is reasonable practice to omit vaccinating the enfeebled, the very sick, and those with profuse skin lesions such as eczema, unless there is exposure.

Dangers and Complications.—The danger from vaccination has been grossly magnified. Vaccination must be looked upon as the production of an acute infectious disease, which, although always benign, should not be treated as trifling. The only danger lies in the fact that we have produced an open wound, which is subject to the complications of any wound. Even a pin prick or a razor scratch may result in death. Before the days of asepsis, wound complications were a matter of concern. No figures on the subject are significant before 1902, when the government passed a law looking to the purity and potency of vaccine virus and other biologic products. With proper precautions, the individual risk is now nil. When trouble ensues, it is in instances where the vaccination was done by scarification or other large insertion, or where the site has not been kept dry, cool and clean. Shields and other dressings closely applied to the skin have been the cause of complications; and in some instances infection has been scratched or rubbed into the take. It is now rare to see complications of any sort. In any properly vaccinated case, *the danger is infinitesimal when compared with the benefit conferred.*

Of the millions of vaccinations done in the army and navy during the World War, there is not a single record of serious result. This clearly indicates that with the use of a carefully tested virus and efficient technic, danger has disappeared.

Syphilis was formerly a complication in arm-to-arm vaccination, but cannot take place with bovine virus. Tuberculosis and leprosy were formerly thought of as possibilities when human virus was used, but these also are not to be feared with the use of bovine virus.

Foot-and-mouth disease has in one instance been demonstrated as a contamination of vaccine virus.¹⁶ It is, however, impossible to convey foot-and-mouth disease to man through cutaneous inoculation. While no harm has been done to man, the contamination is undesirable and vaccine virus is tested from time to time to assure its freedom from this infection (see page 19).

Autovaccination is usually due to scratching the virus with the finger into the nose, the mouth, the mucous membranes or any part of the skin. When carried into the eye it may cause blindness. Physicians sometimes vaccinate their lips by blowing into vaccine tubes. In vaccine establishments accidental vaccination of the hand is common.

Generalized vaccination is sometimes reported, but in my experience is a mistaken diagnosis. The eruption is strictly confined to the site of insertion. Satellite vesicles sometimes develop in the immediate neighborhood owing to the spread of the virus into minute nicks in the epithelium. I once

¹⁶ Mohler and Rosenau, U. S. Dept. of Agriculture, B. A. I. Circular 147, June 16, 1909.

produced a generalized eruption in a calf after intravenous injection of a large amount of the virus. In this case there was a prolonged period of incubation.

Wound infections are now rarely seen, and when they occur demand the usual surgical measures.

Tetanus.—Tetanus deserves a special word. This serious infection sometimes complicates a vaccination wound just as it may any wound. When we consider the millions of vaccination wounds, many of which are neglected surgically, it is no surprise to learn that tetanus occasionally occurs as a postvaccinal complication. Most of the reported cases are from the United States, and almost always in children with their first vaccination. Acland is acquainted with only one instance in more than five million consecutive vaccinations in England, and even in this one there was no evidence that the tetanus was in the vaccine virus. Over thirty-one million doses of vaccine were used in the United States from 1904 to 1913 inclusive, yet only 41 authenticated cases of tetanus occurred subsequent to vaccination.¹⁷ The pernicious method of vaccination by scarification was used in almost all cases of tetanus following vaccination. Many of the cases give a history of having the vaccination scab or crust removed in some way, thus permitting infection of the wound with a re-formation of the crust and the establishment of an anaërobic condition. Several fatal cases of tetanus were due to the use of bunion pads as a dressing. Laboratory tests have demonstrated the presence of tetanus spores in bunion pads from the same source as those which were associated with tetanus cases. The use of any kind of a shield as a vaccination dressing is deprecated.

The fact that lack of care is an important factor in postvaccinal tetanus is indicated in the figures from the United States Army and Navy with a record of millions of vaccinations without a single case of tetanus. At the Hygienic Laboratory at Washington vaccine virus representing over two million vaccinations has been examined without finding tetanus. Special tests for tetanus are required by federal regulations of every lot of vaccine virus before it is placed upon the market.

The occurrence of occasional stray tetanus spores in vaccine virus was demonstrated by Carini.¹⁸ Such vaccine, however, has proved entirely harmless in thousands of cases. Francis also showed that vaccine virus purposely contaminated with tetanus spores will not produce tetanus in monkeys, although it will produce typical takes.¹⁹

Glycerin does not destroy the tetanus spore. While the occasional danger cannot be denied, it is plain that postvaccinal tetanus can usually be laid to improper methods of vaccination and to lack of care of the vaccination wound.

Compulsory Vaccination.—Vaccination affords a high degree of immunity to the individual, and a well-nigh perfect protection to the community. To

¹⁷ *U. S. Pub. Health Rep.*, Reprint 289, July 16, 1915.

¹⁸ *Centralbl. f. Bakteriöl.*, Orig. 1904, 37: 1147.

¹⁹ *U. S. Hyg. Lab. Bull.*, No. 95, 1914.

remain unvaccinated is selfish in that by so doing a person steals a certain measure of protection from the community on account of the barrier of vaccinated persons around him.

The laws and regulations ²⁰ concerning vaccination in the United States show a marked lack of uniformity. Massachusetts,²¹ Rhode Island and New Hampshire stand with Hawaii, Porto Rico and Guam in requiring the vaccination of public school children. There has been a tendency to relax vaccination laws in a number of states. Utah even went so far as to specifically forbid compulsory vaccination as a prerequisite to school attendance. In England the conscience clause allows many persons to remain unvaccinated.

Decisions in the various courts in the United States have held compulsory vaccination to be legal. A decision of the Supreme Court of the United States (*Henning Jacobson v. The Commonwealth of Massachusetts*, April 1, 1905) upheld in every respect the statute, the validity of which was questioned under the Constitution:

"The liberty secured by the Constitution of the United States . . . does not impart an absolute right in each person to be, at all times and in all circumstances, wholly freed from restraint. Real liberty for all could not exist under the operation of a principle which recognizes the right of each individual person to use his own, whether in respect to his person or his property, regardless of the injury that may be done to others."

Theoretically it would be ideal if all persons submitted to vaccination and revaccination voluntarily. But experience has shown that this is impractical, and, wherever tried, has failed. The best results have always been obtained where vaccination has been required, and, in my judgment, this is the only present means by which smallpox may be eliminated. (see page 122).

Claims for Vaccination.—1. "Duly and efficiently performed it will protect the constitution from subsequent attacks of smallpox as much as that disease itself will." ²²

2. It protects against smallpox for a period which varies with the individual, but which for practical purposes may be taken to average seven to ten years.

3. The protection may be renewed by repeating the vaccination.

4. Persons vaccinated every ten years will be reasonably protected throughout life.

5. Vaccination and revaccination systematically and generally carried out confer complete protection to a community or nation. In other words, while the individual protection is not always lasting, the communal protection is absolute. Two successful vaccinations are sufficient for adequate communal protection.

6. A person vaccinated once and at a later time contracting smallpox

²⁰ J. W. Kerr, "Vaccination, and Analysis of the Laws and Regulations Relating Thereto in Force in the United States," *Pub. Health Bull.*, No. 52.

²¹ Massachusetts, in 1809, was the first state to enact legislation relative to vaccination.

²² "I never expected that it would do more, and it will not, I believe, do less."—Jenner. Baron, *Life of Jenner*, Vol. II, p. 135.

as a rule has the disease in a less serious form than an unvaccinated person. The degree of favorable modification of smallpox depends upon the length of time elapsing between the vaccination and the attack of smallpox.

7. The beneficial effects of vaccination are most pronounced in those in whom the vaccine affection has run its most typical and perfect course.

The experience of over 125 years offers convincing proof of the pronounced difference in the mortality and morbidity from smallpox in the vaccinated and the unvaccinated.

The most striking proof of the benefits and sure protection of vaccination is shown by the immunity of the personnel of smallpox hospitals during epidemics. The well-vaccinated doctors, nurses, and attendants, although in intimate contact with the infection, are uniformly spared. The patients, on the other hand, almost invariably tell a story of never having been successfully vaccinated. There are many records of families, all members of whom have had intimate and prolonged exposure to smallpox, and practically every unvaccinated member contracts the disease, while the recently vaccinated remain protected.

The control of smallpox is directly proportional to the compulsory requirements of the vaccination laws and their sane and effective enforcement. The amount of the disease in any country is directly proportional to the amount of unvaccinated population. Communities with compulsory vaccination and revaccination have eliminated the disease.

Germany has demonstrated that smallpox can be controlled in a nation by vaccination. April 8, 1874, a general compulsory vaccination and revaccination law was passed. The law requires the vaccination of all infants before the expiration of the calendar year following birth. Since this law went into effect, Germany, in comparison with her previous record, has practically eliminated smallpox, despite the fact that the disease has been frequently introduced from without. In 1897 there were but eight deaths from smallpox in the entire German Empire, population 54,000,000. Since then long periods have passed without a single death from smallpox. From 1901 to 1910 there were only 380 deaths from smallpox in Germany; during the same period there were 4,286 deaths from smallpox in England and Wales, with only about half the population of Germany; furthermore, many of the deaths in Germany were in foreigners. Thus in 1909, out of twenty-six deaths from smallpox, thirteen were foreigners, eleven of whom were Russians. In 1911, there were 288 cases of smallpox in the German Empire. Of these, 119 were in foreigners. In 1912, there were 340 cases, of which 153 were in foreigners. In 1913, there were only ninety cases, of which thirty-nine were in foreigners. In the huge German army there were only two deaths from smallpox from 1874 to 1914. One of these was a reservist who had not been successfully vaccinated. During and immediately following the World War, the large number of Russian prisoners in Germany and the disarrangement of public health control caused a marked increase of smallpox in the civil population, chiefly in those who had been vaccinated more than twenty-five

years previously. Since then smallpox has again been brought down practically to the vanishing point.

Vaccination certificates should show the name and age; the date of application of the vaccine virus; the nature of the take, whether primary, accelerated or immediate; and the signature of a licensed physician. The data should be based upon observation of the course of the take. Accelerated and immediate reactions are to be interpreted as successful takes.

INOCULATION OR VARIOLA INOCULATA

(Variolation)

The earlier practice of *inoculation* or *variolation* must be carefully distinguished from that of vaccination. By inoculation we mean the introduction of *smallpox* virus into the skin of man. The disease thus produced is usually mild, but is nevertheless smallpox, and just as contagious as smallpox.

Inoculation is a very old custom. It was practiced by the Chinese from time immemorial. The method was introduced into western civilization through Lady Mary Wortley Montagu, who learned of the practice at Constantinople and had her own boy "engrafted" with successful result. In 1717 Lady Montagu wrote her now famous letter to her friend Sarah Chiswell, and inoculation soon became popular in England (1721) and spread to America and the Continent.²³ It was introduced into this country by Zabdiel Boylston at Boston. But the dangers were early realized and inoculation was soon replaced by vaccination. According to Plehn, inoculation is still practiced in central Africa.

The method of inoculation is similar to that of vaccination. The matter is obtained from the vesicle or pustule of a case of smallpox. This material is then introduced into the skin by means of a puncture, an incision, or through an abraded surface. The Chinese inoculate usually by plugging the nostrils with cotton previously saturated with a mixture of water and pustular-crustaceous matter taken from the eruption of a smallpox patient; less commonly by blowing the crushed fresh crusts into the nostrils through a bamboo pipe.

Following the inoculation of smallpox virus a local eruption appears on the fourth day at the site of the inoculation. This local eruption resembles vaccinia but develops more rapidly. Constitutional symptoms appear on the evening of the seventh or the morning of the eighth day following the inoculation. These symptoms resemble the onset of true smallpox and are rigor, headache, vomiting, and fever. The local eruption subsides on the appearance of the febrile symptoms but at the same time the general eruption breaks out. The crop is usually discrete, moderate in number, but runs the usual course through papule, vesicle and pustule formation.

²³ The practice of inoculation had been published in England as early as 1714 by Timoni of Constantinople, at Venice in 1715 by Pylarini, and in the same year in London by Kennedy, a surgeon who had been in Turkey. Its adoption and subsequent diffusion, however, were due to Lady Mary Wortley Montagu.

This phase of the subject may be made clearer by considering smallpox as existing in three forms (1) *variola vera* or true smallpox; (2) *variola inoculata* or inoculated smallpox; (3) *vaccinia*, cowpox, or modified smallpox. The differences between these affections are shown in the following table:

VARIOLA VERA	VARIOLA INOCULATA	VACCINIA OR COWPOX
True smallpox	Inoculated smallpox	A derivative of smallpox
Occurs only in man	Occurs in man and monkeys	Man, monkeys, cattle, guinea-pigs, rabbits, rats, camels, and many other mammals
High mortality—25 per cent in some epidemics	Milder; rarely fatal; about 1 in 500	Benign; never fatal
A general eruption, often confluent or hemorrhagic	A local and a general eruption, fewer pustules (rarely over 200); seldom confluent or hemorrhagic	Always local and confined to the site of the vaccination
Highly contagious	Equally highly contagious	Not contagious—contracted only by mechanical transfer of vaccine virus
Period of incubation 12 days	8 days' incubation	3 days' incubation

Emphasis must be placed on the fact that *variola inoculata*, while usually a mild disease, is just as communicable as true smallpox, and those who contract the disease in this way get true smallpox, sometimes in serious or fatal form. *Inoculation, therefore, protects the individual but endangers the community.*

Inoculation has fallen into disuse only because we have vaccination. There are conceivable emergencies in which the practice would be justified. For example, on board ship or on an island or in an out of the way place, in the absence of vaccine virus. Under such circumstances it would be essential to inoculate everybody at the same time.

The inoculation of smallpox will always remain for the student of hygiene one of the most interesting episodes in the development of preventive medicine. It illustrates in the clearest manner some of the fundamental phenomena of infection, susceptibility, and immunity. It was animal experimentation on a huge scale, the like of which we shall never see repeated on man as the subject. It is now a matter of regret that for the sake of science better advantage was not taken of the data.

SMALLPOX

(*Variola*)

Smallpox belongs to the family of acute exanthematous diseases. The eruption is general and occurs on all parts of the skin and exposed mucous membranes. Prominences and surfaces exposed to irritation are particularly

affected, while depressions and protected surfaces tend to be spared. The eruption comes out as papules which develop into vesicles, and these into pustules which dry and crust, following the course of vaccinia. Four clinical types are recognized: (1) discrete, (2) confluent, (3) hemorrhagic (including the highly fatal hemorrhagic pustular form and the uniformly fatal purpuric form), and (4) modified. The period of incubation is quite constant, twelve days, although variations from nine to fifteen days have been noted. Usually fourteen days elapse between exposure and eruption.

Smallpox is a disease subject to mutations. Clinically, cases vary from malignant hemorrhagic types that kill in forty-eight hours to mild cases with half a dozen papules. As a general rule, the severity of the disease is directly proportional to the amount of the eruption. Aberrant forms are noted in most epidemics.

Alastrim is a name given to one of these aberrant forms. I have seen this disease in Jamaica, and believe it to be true smallpox with some clinical variations. For public health purposes it must be considered and handled as smallpox. It is prevented by vaccination.²⁴ Smallpox is also called kaffir pox, cotton pox, milk pox, parasmallpox and many other names.

Prevalence.—It is very difficult for us now to realize that smallpox was once much more common than measles and much more fatal. Many of those who recovered were disfigured for life; left blind, or with some other serious consequence of the disease. For centuries smallpox was one of the greatest scourges. It depopulated cities and exterminated nations. In Europe alone, where its ravages were comparatively slight, it killed hundreds of thousands yearly. In the eighteenth century, of which we have the best records, almost everybody had it before he grew up. Parents often exposed their children to the disease in order to be through with it, just as they now sometimes do with the minor contagious diseases. In China a mother does not number among her children those who have not yet had smallpox, because she well knows how uncertain will be their stay in the family.

Smallpox was formerly a disease of children. It was called *Kinderblattern*. Since vaccination protects the child, smallpox has now become more prevalent among adults.

The distinguished mathematician, Bernouilli, estimated that 15,000,000 people died of smallpox in 25 years in the eighteenth century. It has been estimated that 60,000,000 people died of smallpox during that century. Haygarth gives an account of a smallpox epidemic in Chester, England, population 14,713. At the termination of the epidemic there were but 1,060 persons, or 7 per cent of the population, who had never had smallpox. Many similar instances are cited in the literature. The French physician, De la Condamine (1754), said that "every tenth death was due to smallpox and that one-fourth of mankind was either killed by it or crippled or disfigured for life." Sarcone (1782) estimated the number of persons in Italy who suffered from smallpox as 90 per cent of the population.

²⁴ U. S. Pub. Health Rep., 1921, 36: 1437.

Smallpox was introduced into the Western Hemisphere by the Spaniards about fifteen years after the discovery of America. In Mexico within a short period 3,500,000 persons are said to have died of the disease (Chapman). Catlin (1841) states that of 12,000,000 American Indians 6,000,000 fell victims to smallpox. In Iceland, in 1707, 18,000 perished out of a population of 50,000; that is, smallpox killed 36 per cent of the total population in one year.

A good example is that of Boston in 1752, population at that time 15,684. Of this number 5,998 had previously had smallpox. During the epidemic 5,545 persons contracted the disease in the usual manner, and 2,124 took it by inoculation. One thousand eight hundred and forty-three persons escaped from the town to avoid infection. There were, therefore, left in the city but 174 persons who had never had smallpox.

Smallpox is still as serious as it was in former times. The following table ²⁵ gives the recent prevalence of smallpox in Europe and in the United States:

CASES OF SMALLPOX NOTIFIED IN EUROPE AND THE UNITED STATES, 1919-1923

Countries	1919	1920	1921	1922	1923	Approximate Population, Millions
Europe:						
England and Wales	294	263	315	973	2,485	38
Scotland	9	725	106	7	3	5
Norway	0	0	1	0	0	3
Sweden	7	11	2	0	0	6
Denmark	0	0	7	0	0	3
Germany	5,012	2,042	688	215	17	61
Netherlands	5	50	1	0	2	7
Belgium	*	*	21	23	31	8
Switzerland	3	2	596	1,159	2,145	4
France	572	392	341	172	195	37
Spain (deaths)	3,620	3,280	2,087	1,332	529	20
Italy	34,365	26,453	4,644	534	253	40
Kingdom of Serbs, Croats and Slovenes	*	4,156	2,119	728	1,042	11
Bulgaria	874	527	22	24	20	5
Roumania	20,523	3,467	2,745	865	89	8
Hungary	*	*	131	2	9	6
Austria	411	253	18	4	17	14
Czechoslovakia	11,209	4,529	1,542	70	36	24
Poland	1,864	3,948	5,078	2,399	502	2
Lithuania	*	1,213	1,035	345	25	2
Latvia	*	422	255	160	23	2
Estonia	267	435	141	58	21	3
Finland	1,759	77	27	91	12	3
Russia in Europe (with- out Ukraine)	169,545	121,587	68,503	40,839	31,311	80
Ukraine	*	34,730	28,123	11,095	3,710	46
Total	250,339	208,562	118,548	61,095	42,477	438
United States	56,332	96,684	102,787	32,800	29,968	106

* Data not available.

²⁵ League of Nations, Health Organization, Epidemiological Intelligence No. 8, August, 1924.

The table shows only the cases reported. Many more cases actually occurred, for both the reporting and the records are quite incomplete. Over one million cases of this readily preventable disease occurred during the recent five years in parts of Europe and the United States.

Occurrence.—Few of the acute infectious diseases show such a complete independence of conditions such as race, climate, soil, age, sex and occupation, sanitary surroundings, etc., as does smallpox. It thrives wherever the contagion is carried and wherever it finds susceptible people. Probably no one is naturally immune. The susceptibility of an unvaccinated population varies only when a smallpox outbreak leaves many immune. This is one reason why the disease recurs in irregular waves among an unvaccinated population. The mortality varies greatly in different epidemics: at times it is less than one per cent; it frequently reaches 30 per cent and over.

From 1897 to 1912 the mortality in the United States varied from 0.34 per cent to 6.2 per cent; in 1895 it was 20.84 per cent; and following that 0.5 per cent. These differences occurred in the prevaccination era as well as now. There seem to be two distinct strains in the United States, one the classic smallpox of the textbooks, the other a very mild form. These strains remain rather true to type.

Strains of smallpox differ widely in virulence but probably not in communicability. People have little fear of the relatively non-fatal type of the disease which was prevalent in the United States in 1900-1920, and were unwilling to adopt preventive measures. Our recent experience, however, with the malignant strains has shown the fallacy of this attitude. To be vaccinated only in the face of a severe epidemic is locking the door after the horse is stolen.

The epidemiology of smallpox bears no relation to improved sanitation, which has diminished the prevalence of plague, typhoid and cholera, and has practically subdued typhus and relapsing fever. It is evident that general sanitation could not affect contagious diseases like smallpox and measles. Smallpox spares neither the high nor the low, the rich nor the poor, the clean nor the dirty, the wise nor the foolish, the good nor the bad; before the days of vaccination it counted many kings, queens and princes among its victims. George Washington contracted smallpox in the West Indies.

Modes of Infection.—Smallpox is spread mainly by direct personal contact. We are still ignorant of the precise channel by which the infection enters the body. The view generally held is that it enters the system through the respiratory mucous membranes. The Chinese inoculate the disease by placing variolous matter in the nostrils, but the disease so produced is said to resemble variola inoculata.

The virus of smallpox is contained in the skin lesions. Of this we have experimental evidence. The secretions from the mouth and nose contain the virus.²⁶ These nasopharyngeal secretions are probably the most common

²⁶ Friedemann and Gins, "Experimentelle Untersuchungen über die Übertragung der Pocken," *Deutsch. med. Wchnschr.*, 1917, 43: 1159.

means of infection. The disease is contagious before the eruption appears. It is even believed to be communicable during the period of incubation. Smallpox has always been taken as the type of the contagious diseases; the contagion appears to be the most "volatile" of any of the diseases of man with the possible exception of measles and influenza. This volatility, however, has been overstated. The radius of danger is limited to the immediate vicinity of the patient. English observers have long taken the view that smallpox may be blown for great distances, and they attribute the prevalence of smallpox to the windward of hospitals as an indication that the virus may be carried down the wind. My experience with the disease teaches me that the danger from such a source is practically nil. One may safely live next door to a smallpox hospital that is well screened and properly managed. Sur-reptitious visiting, the interchange of infected materials, or possibly flies and other insects may account for the spread of this disease outside of hospital walls.

In addition to more or less direct contact, smallpox may be spread indirectly in a great variety of ways. The secretions from the mouth and nose contain the infection, and, while suspicion has not particularly fallen upon the feces and urine, it is probable that all the secretions and excretions from the body may be infective at some time throughout the disease, or during convalescence. Toys, pencils, spoons, cups, towels, handkerchiefs, bedding, and objects of the greatest variety that have in any way become contaminated may carry the infection. Under favorable circumstances the active principle may probably live for a considerable time upon fomites, although the practical danger from this source is not very great.

Smallpox is not usually considered an insect-borne disease, but it is highly probable that a fly lighting upon a smallpox patient and getting its proboscis, feet, and other portions of its body smeared with the variolous matter, and then flying to a susceptible person, could thus readily transmit the infection. Other insects may by such mechanical transfer play a similar rôle.

Diagnosis.—*The Paul Reaction.*—The Paul reaction consists in inoculating the suspected material on the cornea of rabbits. The reaction is positive for smallpox, negative for chickenpox. When positive, the reaction consists of tiny, dewdrop-like vesicles, some of them umbilicated, which make their appearance in forty-eight hours. On microscopic examination, Guarneri bodies are present in the epithelial cells and the lesions are characteristic. It takes training to recognize the microscopic picture during life. Simon and Scott²⁷ therefore recommend that the rabbit's cornea be fixed in sublimated alcohol, which brings out intensely white opaque little elevations along the line of inoculation. If, then, these lesions are examined in stained preparations, the characteristic epithelial changes, with the presence of Guarneri bodies, signify smallpox.

Guarneri in 1892 described the bodies which now bear his name²⁸ in

²⁷ *J. Lab. & Clin. M.*, 1925, 10: 7.

²⁸ *Arch. per le sc. med.*, 1892, 26: 403.

the deep epithelial cells of the skin in cowpox and smallpox as well as in the epithelial cells of the cornea of rabbits. He gave them the name of "Cytorrhycles variolæ." Tyzzer²⁹ was also convinced that these vaccine bodies represent the etiological agent of the disease. He found them constantly present in smallpox and absent in chickenpox. Paul, in 1915,³⁰ introduced modifications in the technic.

The differential diagnosis between smallpox and chickenpox is further discussed on page 394.

Resistance of the Virus.—It is generally, and doubtless correctly, assumed that the active principle of variola has approximately the same resistance to external conditions as vaccine virus. This assumption is confirmed by experimental evidence, which shows that the virus of smallpox is somewhat more readily destroyed than the virus of cowpox. Scientific data concerning the viability of variolous matter is meager. Brinckerhoff and Tyzzer³¹ found that variolous virus is less resistant to desiccation than vaccine virus; that variolous virus does not pass a Berkefeld filter and is attenuated by long exposure to 60 per cent glycerin.

In general it may be said that variolous virus is killed by exposure to ordinary germicidal substances, both liquid and gaseous, in the strengths and time commonly employed. It succumbs in fact before the average non-spore-bearing bacteria.

There is an exception to this statement in the case of phenol, cresol and other coal-tar disinfectants. McClintock and Ferry³² have shown that such germicides as carbolic acid, cresols, and the like do not destroy the virulence of vaccine virus in 0.5 per cent solutions in five hours' exposure. Noguchi³³ found that 1 per cent phenol has no injurious effect upon emulsions of the testicular strain of vaccine virus. The inference is allowable that this class of disinfectants cannot be relied upon to prevent the spread of smallpox.

Isolation and Smallpox Hospitals.—Isolation and disinfection are only secondary measures in preventing smallpox. They cannot be regarded as substitutes for vaccination.

Isolation should be carried out with strictness for the reason that smallpox is one of the most contagious of the communicable infections. While the patient should be isolated, it is not necessary to isolate the hospital by banishing it to an inconvenient or undesirable location. There is, in fact, no good reason why a smallpox hospital should not be one of the units of the general hospital for communicable diseases. In any event, there need be no danger from a smallpox hospital situated upon a highroad or near other habitations. *Wherever the hospital is situated, proper precautions must be taken to prevent the spread of the disease.*

The smallpox hospital should not be a pesthouse, but should be as invit-

²⁹ *J. Med. Research*, 1904, 11: 1.

³⁰ *Centralbl. f. Bakteriol*, 1915, Pt. I, Orig., 75: 523.

³¹ *J. Med. Research*, 1906, 14: 223.

³² *J. Am. Pub. Health Ass.*, 1911, 1: 418.

³³ *J. Exper. M.*, 1918, 27: 425.

ing and attractive as economic conditions justify. Smallpox should not be treated in the home. From the standpoint of prophylaxis the hospital is the logical and best place to care for this and most other communicable infections. Skilled nursing and trained attendants must be provided.

The room in which the smallpox patient is isolated should be simply furnished to facilitate cleanliness and to permit purification. It must be well screened and free from insects and vermin of all kinds. The room should be well ventilated.

The nurse attending a case of smallpox should also be segregated, and all visiting should be strictly interdicted. A separate kitchen should be provided and care taken that the dishes be scalded and remnants of food burned.

Disinfection.—Bedding, underwear, towels, and other objects should not leave the sick room unless they are first boiled, steamed, or immersed in a suitable germicidal solution, such as bichlorid of mercury, 1:1,000, or formaldehyd solution, 10 per cent. Carbolic acid should not be trusted.

For terminal disinfection cleansing of surfaces with a germicidal solution is much surer than gaseous fumigation. Objects particularly contaminated or soon to be used by others should be given a separate and special disinfection. Finally, the room should be thoroughly cleansed, aired, and sunned.

The patient must be regarded as the source and fountainhead of the infection, and measures should be used at the bedside to prevent the surroundings from becoming contaminated. Cloths, cotton, and other dressings that become soiled with the contents of the vesicles and pustules after they break should be burned. The urine and feces may be disinfected with chlorinated lime. The sputum and discharges from abscesses should be collected on cheap cloths and burned. As a rule, smallpox patients are not dismissed from quarantine until desquamation has ceased. This may be favored by the use of warm baths and a generous use of glycerin soap, also by anointing the skin with vaselin or a bland oil. Special attention should be given to the hair, which should be well shampooed; to the interdigital spaces, and the fingernails, as well as to all folds of the skin, before the patient is released.

Summary.—*Prevention.*—There is only one way to prevent smallpox, and that is through vaccination. The method is logical, specific, sensible and satisfactory. Isolation and disinfection are only secondary. In a well vaccinated population, smallpox would disappear instead of being a national sanitary disgrace as it now is.

The management of a smallpox epidemic is discussed on page 523. The differential diagnosis between smallpox and chickenpox is discussed on pages 30 and 394. Quarantine for smallpox is considered on page 532.

RABIES

(*Hydrophobia*; *Wasserscheu*; *Wut*; *Tollwut*; *Lyssa*; *La Rage*)

Rabies is an acute, specific, rapidly fatal paralytic infection communicated from a rabid animal to a susceptible animal, through a wound usually pro-

duced by biting. Man always contracts the disease from some lower animal, commonly the dog. The infective agent must be inoculated into the tissues. Contact with the saliva of rabid animals may produce the disease if there are small fissures or abrasions in the skin. The virus is harmless when ingested provided the mucosa is intact. *Rabies is a wound infection.*

The disease is remarkable in several particulars, especially the period of incubation, which is more variable and more prolonged than that of any other acute infection, and its high mortality which is practically 100 per cent. Spontaneous recovery from rabies naturally acquired is very rare if it occurs at all. Pasteur³⁴ was the first to note that recovery occasionally took place in inoculated dogs after the first symptoms were manifest. Högyes,³⁵ Joseph Koch³⁶ and others³⁷ have reported recoveries in experimental animals, dogs, rabbits and guinea-pigs.

Susceptible Animals.—Every mammal is susceptible. Even birds may contract the disease. It is most common in dogs, but it also occurs frequently in wolves, jackals, foxes, and hyenas. Rabies in cats and skunks is comparatively rare and but occasionally transmitted to man. Cattle, sheep, and goats are infected relatively in about the same degree. It is less common in horses. Swine contract the disease less frequently than other domestic animals.

Rabies is perpetuated in civilized communities almost exclusively by the domestic dog and to a small extent by wild animals of the dog family.

Prevalence.—Rabies exists practically all over the world. It has never been in Australia, and has not been known in Denmark, Norway and Sweden for more than fifty years. It can be controlled in other insular or peninsular countries, and then kept out by a quarantine on dogs. Rabies had been eradicated from England, but was reintroduced during the World War by dogs carried in flying machines. It is most common in France, Belgium, and Russia. In France, rabies of a virulent type with a short incubation period has spread since the first of the World War.

Compared to the major plagues of man, rabies is a comparatively rare disease. It is, nevertheless, a serious public health problem and readily controllable. The infection is widespread throughout the United States, and in recent years has been on the increase. In 1890 the United States Census reported 143 deaths in thirty states; in 1908 rabid animals were reported from 535 localities, with 111 human deaths; in 1911 there were 1,381 localities with ninety-eight deaths in man; in the ten years 1910 to 1920 there were an average of sixty-three deaths annually from rabies in the United States Registration Area.

Rabies is commonly supposed to prevail only during the hot months, but it is in fact more prevalent in cold weather. Exposure to cold seems to increase its virulence. More dog bites occur from April to September than

³⁴ Communication to the Academy, 1882, Expositions 9, 10.

³⁵ *Nothnagel's Specielle Path. u. Ther.*, Wien, 1897.

³⁶ *Ztschr. f. Hyg. u. Infektionskr.*, 1909, 64: 258.

³⁷ J. M. Phillips, *et al.*, *J. Infect. Dis.*, 1921, 29: 97, containing a review of the literature.

from October to March in this climate, because dogs run abroad more freely at this season of the year.

Period of Incubation.—From the standpoint of prevention it is fortunate that the period of incubation of this disease is prolonged. This period varies from fourteen days to a year or more. Such prolonged periods of incubation indicate latency. The average period is as follows: Man, forty days (apt to be shorter in children or following bites on the face); dogs, twenty-one to forty days; horses, twenty-eight to fifty-six days; cows, twenty-eight to fifty-six days; pigs, fourteen to twenty-one days; goats and sheep, twenty-one to twenty-eight days; birds, fourteen to forty days.

The period of incubation depends upon the amount and virulence of the virus and the nature and site of the wound, especially with reference to its nerve supply. It requires about fifteen days, counting from the last injection, to induce an active immunity to the disease by means of the Pasteur preventive treatment. There is, therefore, usually sufficient time, if started early, to prevent the development of symptoms.

It is probable that the prolonged and variable period of incubation is due in part to the fact that it takes time for the virus to travel along the nerves to the central nervous system, and that it may there remain dormant (latent) until conditions favor multiplication.

The Virus.—The active principle of rabies occurs principally in the saliva and in the central nervous system. It may be in the saliva five days before the animal shows symptoms (Roux and Nocard). It is, therefore, sufficient to watch a dog that has bitten a person or another animal for ten days. If no symptoms of rabies appear during this time there is no danger of conveying the disease, and the Pasteur prophylactic treatment is unnecessary.

The virus has been found in the adrenals, the tear glands, the pancreas, the vitreous humor, the spermatic fluid, the urine, the lymph, the milk, as well as all parts of the central nervous system and the peripheral nerves. It is also found in the spinal and ventricular fluids. It has not been demonstrated in the liver, spleen, blood, or muscles.

The virus enters the system through the broken skin and follows the nerve trunks from the seat of injury to the spinal cord, thence to the medulla and brain. The route corresponds to that of tetanus toxin. The mode of invasion of the virus may explain why pain, throbbing, tingling, numbness and other nervous disturbances are the first symptoms to occur in parts of the body that have received the virus. It also partly explains the variable period of incubation, which is shorter in wounds of the face than wounds of the extremities. It also explains why the disease is more liable to occur when the wounds are in parts of the body with a rich nerve supply. I have experimental evidence (unpublished) that indicates that the virus readily enters the nerve endings in the skin.

Noguchi³⁸ claimed that he succeeded in growing the virus, which appears in cultures as granular and pleomorphic chromatoid bodies, some of which are

³⁸ *J. Exper. M.*, 1913, 17: 29.

surrounded with membranes. Williams³⁹ and Moon⁴⁰ believe they have evidence of growth in brain tissue, having produced rabies in animals in the fifth transfer of such "cultures." Several workers report transfers upon artificial culture media, with indications of growth. The nature of the virus is not settled. It is filterable (see Negri Bodies, page 48).

Viability.—The virus of rabies in the spinal cord of rabbits dies in about five, surely in eight days when dried at 20°-22° C., if protected from light. Spread in thin layers, it dies in four or five days, and exposed to the sunlight in forty hours. It is quite resistant to putrefaction. In a decomposed carcass it may be recovered by placing some of the central nervous system in glycerin. The glycerin destroys most of the contaminating bacteria, but preserves the virus. Rabic virus is completely destroyed at 50° C. in one hour, and at 60° C. in thirty minutes. It is not injured by extreme cold.

Harris found the virus to be very resistant to dryness at low temperatures. Sawtschence⁴¹ found that it requires from five to seven days to destroy the fixed virus in 5 per cent phenol, and that it is not destroyed by 0.5 per cent phenol in twenty days. Other filterable viruses, notably variola and vaccinia, also show unusual resistance to phenols and cresols. These substances are therefore ineffective germicides for this group of infections.

According to Cumming⁴² most of the aldehyd compounds are very active in destroying the infectivity of the fixed virus. A 0.5 per cent solution of salicylaldehyd, benzaldehyd, or furfurol destroys the virus in less than three hours. The special activity of formaldehyd is shown by the fact that the virus is destroyed when exposed for two hours to 0.08 per cent solution. Bichlorid of mercury, 1:1,000, for one hour, or a saturated solution of iodin in water, completely destroys the virulence, and Wyrskowski has shown that gastric juice has a pronounced deleterious effect upon the virus. It rapidly loses its strength when exposed to air and especially to sunlight, but protected from heat, light and air, retains its virulence for a long period. Practically, there is little danger from saliva-smearred fomites.

The Relative Danger of Bites.—Wolf bites are most dangerous on account of the savage character of the wound, and the virulence of the virus. Cat bites come next, and then dog bites. The relative danger of bites of other animals is as follows: foxes, jackals, horses, asses, cattle, sheep, pigs. There is no authentic instance of the transmission of the disease by the bite of man, though this may be possible. The bites of horses and other herbivora are less dangerous because their blunt teeth usually cause contused wounds without breaking the skin.

Bites on exposed surfaces are more dangerous than through the clothing, because the saliva is wiped from the teeth and little or none enters the wound. Long-haired dogs and sheep often escape infection for the same reason. Bites upon the face are most apt to be followed by rabies. The develop-

³⁹ *J. Am. M. Ass.*, 1913, 61: 1509.

⁴¹ *Ann. de l'Inst. Pasteur*, 1911, 21: 492.

⁴⁰ *J. Infect. Dis.*, 1913, 13: 232.

⁴² *J. Infect. Dis.*, 1914, 14: 33.

ment of the disease depends upon the virulence and quantity of virus introduced into the wound, the site of the wound especially with reference to the nerve supply, and the susceptibility of the animal bitten.

Not every person bitten by a mad animal develops rabies. Leblanc's figures are 16.6 per cent. The statistics are difficult to analyze, and it is almost impossible now to collect sufficient data. According to the most reliable figures, it would seem that rabies develops in not less than one person in ten bitten by mad dogs, and not receiving the Pasteur treatment. Paltauf places the figures at 6 to 9 per cent. From 15 to 20 per cent is a moderate estimate of the death rate for all persons bitten by rabid animals (see also page 43). Some strains are more virulent than others.

Factors Influencing the Development of Rabies.—The development of rabies depends upon a number of factors, such as the origin and nature of the virus, its virulence and amount, the site of inoculation and the character of the wound. The surest way to induce the disease is by injecting the virus under the dura or directly into the brain. Small quantities of either fixed or street virus suffice by this method. Positive results are also quite constant when a mad dog bites the shaved head of another dog. Pasteur used this method of natural infection by preference.

Galtier⁴³ obtained positive results by infecting experimental animals with the virus upon the superficial layers of the skin. I found that rubbing the virus into the scarified skin is one of the most reliable methods of inducing the disease next to cranial inoculations. Remlinger⁴⁴ induced rabies in rabbits and guinea-pigs by bandaging the virus upon the freshly shaven skin. This was confirmed by Galli-Valerio.⁴⁵ These experiments explain the possibility of infection from insignificant bites as well as infection following exposure without biting, as in licking, and emphasize the importance of cauterizing the edges of the skin wound with nitric acid.

The effects of subcutaneous inoculation are very inconstant. Negative results are the rule if the virus is localized subcutaneously in places where the skin is loose with poor nerve supply and minimum injury to tissue. Subcutaneous inoculations are more often positive in the rabbit than in the dog or guinea-pig. Fermi⁴⁶ has shown that rats and mice are the most susceptible of all laboratory animals to subcutaneous injections. Dogs and also rats when injected subcutaneously are much more susceptible to street virus than to fixed virus. Pasteur disclosed the apparent paradox that dogs sometimes succumb to small amounts injected subcutaneously, but resist large amounts. This was confirmed by Kraiouchkine⁴⁷ and Remlinger.⁴⁸ There are many other things about rabies that we do not know.

⁴³ *J. de méd. vét.*, 1890, 622.

⁴⁴ *Compt. rend. Soc. de biol.*, 1905.

⁴⁵ *Centralbl. f. Bakteriöl, Orig.*, 1 Abt., 1906.

⁴⁶ *Centralbl. f. Bakteriöl, Orig.*, 1907, et *Gior. d. r. Soc. Ital. d'ig.*, 1906.

⁴⁷ *Sur les effets de l'injection sous-cutanée du virus fixe de la rage*, Saint Petersburg, 1897.

⁴⁸ *J. de Physiol. et de path. gén.*, 1905, 7: 295.

PREVENTION

The prevention of rabies is considered under three heads: (1) treatment of the wound; (2) the Pasteur prophylactic treatment; and (3) the control of the disease in dogs.

The cauterization of the wound and the Pasteur prophylactic treatment are efficient preventive measures for the individual, but they are not the true and best methods of controlling and preventing the disease. Rabies may be checked, even eliminated, by measures directed towards the dog (see page 45).

THE LOCAL TREATMENT OF THE WOUND

Cauterization with Nitric Acid.—Wounds produced by the bite of an animal, in which there is any possibility of rabies, should at once be cauterized with “fuming” or strong nitric acid. The acid is best applied with a glass rod very thoroughly to all parts of the wound, care being taken that pockets and recesses do not escape. Punctured wounds should be laid open to allow proper cauterization. Experiments in my laboratory indicate the importance also of cauterizing the edges of the skin.

Thorough cauterization with nitric acid reduces the danger of wound complications, and experience demonstrates that wounds promptly and thoroughly cauterized with nitric acid are seldom followed by rabies. Cabot⁴⁹ obtained the best results with nitric acid and was able to save the lives of 91 per cent of guinea-pigs by cauterization with nitric acid at the end of twenty-four hours; Poor⁵⁰ saved 45 per cent at the end of twenty-two hours. Experiments under my supervision (unpublished) indicate that practically all guinea-pigs may be saved by prompt application of nitric acid; that its effectiveness decreases with time, but that it is still partially protective up to forty-eight hours. No other substance gives equally good results. Strong germicides, such as carbolic acid, are not reliable; nitrate of silver is valueless; formalin and the actual cautery are not effective.

Nitric acid, on account of its diffusibility and penetration, is almost specific for rabies. The resultant wound heals well and little if any additional scarring is produced.

THE PASTEUR PROPHYLACTIC TREATMENT

This method was announced December 6, 1883, by Pasteur, at the International Congress at Copenhagen, and on February 24, 1884, he laid before the French Academy the details of his experiments and results. The next year Pasteur, with the help of Roux and Chamberland, worked out the details of the method now in general use.

The principle of the treatment consists in producing an active immunity

⁴⁹ *Med. News*, Mar., 1899.

⁵⁰ *Coll. Stud.*, Research Laboratory, N. Y. C. Dept. of Health, 1911, 6: 25.

by means of a modified virus, which is attenuated by drying. The fixed virus contained in the spinal cord of rabbits dead of hydrophobia is emulsified and injected subcutaneously.

The distinction between street and fixed virus is of fundamental importance with reference to immunity.

Street Virus and Fixed Virus.—Street virus refers to the virus as it is found in nature. It was so called by Pasteur because he obtained it from mad dogs found on the streets of Paris. Street virus, then, is obtained from dogs naturally infected. When this virus is inoculated into a rabbit, the disease is reproduced after a variable and often prolonged incubation period—fourteen to twenty-one days or more. If the virus is then conveyed from rabbit to rabbit through a series of transfers, it becomes progressively more virulent for rabbits. The period of incubation is shortened, until finally the rabbits invariably sicken on the sixth or seventh day and die on the ninth or tenth. When the virus has reached this degree of virulence for rabbits, it is said to be *fixed* for the reason that its virulence remains constant. In its passage through rabbits the modification from street virus to fixed virus is gradual. It is important to note that fixed virus, which has attained a high degree of virulence for rabbits, has lost much of its potency for dogs and seems to be avirulent for man when introduced into the subcutaneous tissue.⁵¹

Fixed virus is an interesting example of mutation of a pathogenic micro-organism by animal passage. The morphology of the Negri bodies is altered, and according to Levaditi⁵² fixed virus has lost the special pansporoblastic cycle of evolution represented by the Negri bodies. Pasteur showed that the virus may be intensified by successive passage through certain animals, as guinea-pigs, rabbits and cats; and weakened by passage through other species, as monkeys, etc.

Is Fixed Virus Pathogenic for Man?—Ferran of Barcelona in 1888 treated 85 persons for dog bites by injecting the fresh fixed virus subcutaneously without ill effects. He used a 1:100 suspension of finely divided fresh fixed virus, injecting 2 c.c. in three separate portions of the body daily for five days. His results were so good that the next year Boreggi⁵³ applied this method, with the result that five of his patients died within a period of ten days. This was an accident which has not been explained. The symptoms, except for paralysis, did not resemble rabies. This unfortunate experience discouraged the further use of fresh fixed virus for many years. Wysokowicz⁵⁴ inoculated fixed virus intravenously into 70 persons without an accident. A number of experimenters (Nitsch,⁵⁵ Proescher and others) inoculated themselves subcutaneously with fixed virus in great amounts without any inconvenience. Högyes, in 1897, revived the use of fresh unaltered fixed virus by the method of dilution.

⁵¹ *New York M. J.*, Oct. 9, 1909; *Arch. Int. Med.*, 1911, 8: 353.

⁵² *Compt. rend. Soc. de biol.*, 1924, 90: 994.

⁵³ *Gazz. med. lomb.*, 1889, 48: 217.

⁵⁴ Cited by Krasnitski, *Ann. de l'Inst. Pasteur*, 1902, 16: 393.

⁵⁵ *Wien. klin. Wchnschr.*, 1904.

The subcutaneous tissue is evidently quite resistant to the rabbit virus; indeed, even when street virus is injected subcutaneously into susceptible animals the results are uncertain. Marx tested the fresh fixed virus upon monkeys in large doses with negative results. Levaditi⁵⁶ reproduced the disease in a chimpanzee intracerebrally. Over 100,000 persons have been injected subcutaneously with the fixed virus since 1884, when Pasteur announced his discovery, with satisfactory protection against rabies and without untoward effects. It is suggested, however, that the fixed virus may occasionally be pathogenic for man when injected subcutaneously in view of the paralysis which sometimes complicates the Pasteur prophylactic treatment, but the interpretation of these cases is not settled (see page 42). Schweinburg⁵⁷ thinks that the paralysis is due to lipoids contained in the material injected and that Högyes' method, which requires the least amount of material, causes the smallest percentage of paralyzes.

Preparation of the Virus.—Rabbits are injected under the dura mater with a few drops of an emulsion of fresh fixed virus. Strict aseptic precautions are necessary in order to keep out other infections. The rabbit should begin to show symptoms on the sixth or seventh day, and die on the ninth or tenth. Usually the rabbit is not allowed to die, but is chloroformed in order to avoid terminal infections and unnecessary suffering. The spinal cord is removed and hung in a bottle containing potassium hydroxid, and kept in the dark at a temperature of 22° C. Under these conditions the cord generally desiccates, and at the same time the virulence of the virus diminishes until it is no longer infective.

The Pasteur Method.—Pasteur was well on the safe side in starting treatment with cord fourteen days old; in fact, the virus dies long before the fourteenth day: five-day cord usually fails to infect, and eight-day cord has lost its power of producing the disease.

About one-half a centimeter of the cord constitutes a dose. This is ground in about 2.5 c.c. of sterile salt solution so as to produce a uniform emulsion, and strained through gauze. This suspension is injected into the subcutaneous tissue of the abdominal wall. In many institutes the small segments cut each day from the drying cord are placed in pure glycerin. The virulence of the cord in glycerin is not altered for at least twenty days, if kept in the dark and at 15° C. This method, introduced by Calmette⁵⁸ in 1891, based upon observation made by Roux in 1887,⁵⁹ is very convenient, especially where comparatively few patients are treated.

Bacteriologic examinations are made of parts of the spinal cord in order to insure the absence of bacteria, and the rabbit is carefully autopsied as a guarantee that no other disease is present.

⁵⁶ *Compt. rend. Soc. de biol.*, 1924, 91: 56.

⁵⁷ *Wien. klin. Wchnschr.*, 1924, 37: 797.

⁵⁸ *Ann. de l'Inst. Pasteur*, 1891, 5: 633.

⁵⁹ *Ann. de l'Inst. Pasteur*, 1887, 1, 87.

The scheme of treatment is shown in the following table:

DAY OF TREAT- MENT	SCHEME USED AT PASTEUR INSTITUTE, PARIS			SCHEME USED AT U. S. HYGIENIC LABORATORY, N. Y. C. DEPART- MENT OF HEALTH AND BERLIN INSTITUTE, ETC.	
	Pasteur's Original Method		Modified Scheme 1912-1922		
	Mild	Intensive			
1	14, 13*	14, 13, 12, 11	7	5	6, 6
2	12, 11	10, 9, 8, 7	6	4	5, 5
3	10, 9	6, 6	6	3	4, 4
4	8, 7	5	5	2	3
5	6, 6	5	5	4	3
6	5	4	4	3	2
7	5	3	4	2	2
8	4	4	3	4	1
9	3	3	5	3	5
10	5	5	4	2	4
11	5	5	3	4	4
12	4	4	4	3	3
13	4	4	4	2	3
14	3	3	3	2	2
15	3	3	2	2	2
16	5	5	4†	4†	4†
17	4	4	3†	3†	3†
18	3	3	2†	2†	2†
19	5	3‡	4‡	3‡
20	4	2‡	3‡	2‡
21	3	2‡	2‡	1‡
22	3§	4§	...
23	3§	3§	...
24	2§	2§	...
25	2§	2§	...

* The numbers represent the age of the dried cord in days.

† Multiple wounds.

‡ Severe wounds.

§ Wounds of the head and face and very severe wounds.

The above table shows the trend towards more intensive methods. Pasteur started with cord fourteen days old: now most of the institutes start with five- or six-day cord. The dose is usually about 0.5 cm. of rabbit cord emulsified in 2.5 to 3 c.c. of salt solution. Somewhat smaller amounts are used for children. The dosage is approximate and the scheme of injections somewhat elastic.

Other Methods.—The classic scheme of Pasteur has been further modified in various ways, depending upon the method used to attenuate the virus. Thus Pasteur attenuated the virus by drying; Babes by heating; Frantzer by the use of bile; Tizzoni and Cattani attenuated the virus in gastric juice. Högyes used fresh material in a diluted suspension; Ferran fresh material and in increasing doses. Cumming altered the virus by dialysis. Harris dried the fresh virus at low temperature, which is used in diluted suspension. Semple used fixed virus (in the brain, medulla and cord) killed by heat and carbolic acid. Other methods have been used to attenuate the virus, such as glycerin, carbolic acid, mechanical disintegration, and, lastly, antirabic serum.

The methods in more or less general use in this country are: The *hygienic laboratory method*, based upon Pasteur's scheme (table, page 40); *Hogyes' method*, a dilution of attenuated fresh fixed virus; the *Harris method*, desiccated fresh fixed altered by dialysis in collodion sacs.

The *Semple method*⁶⁰ is fast becoming the method of choice on account of its simplicity, effectiveness and comparative freedom from paralytic complications. The material for the prophylactic injections is prepared from the fresh fixed virus in the brain, medulla and spinal cord of rabbits. This is ground in 8 per cent sterile salt solution containing 1 per cent carbolic acid, strained through fine muslin, and kept at 37° C for 24 hours. At the end of this period the virus is dead—at least it is not infective when injected into susceptible animals. The material is now diluted with an equal volume of sterile normal saline solution. This final dilution contains 4 per cent of the dead virus in 0.5 per cent carbolic acid normal saline solution. The dose is 2.5 cc. injected into the subcutaneous tissues of the abdominal walls once a day for 14 days.

The claim for the Semple method is that the suspension retains its maximum potency and powers of immunization for a period of at least 3 months. The material injected contains the smallest amount of nerve tissue commensurate with efficient treatment and thereby avoids the so-called post-paralytic paralyses. The dosage is more accurate than with the attenuated cord method, since the cords vary very much in size. The Semple method is economical and further very convenient as the vaccine may be produced in quantity and the whole treatment sent in one shipment. All doses of the vaccine are the same, regardless of age, sex, severity of the bite or location of the wound. Experience demonstrates that the 14-dose treatment is sufficient for all types of cases. Finally, brain matter is said by Nitsch to be ten times more virulent than spinal cord, therefore in using brain we are giving a larger proportion of specific antibody-producing substance and a minimum dose of the useless and probably harmful nervous tissue. There are recorded 5,035 cases treated by the Semple method with 8 deaths, only one of which is described as a failure.⁶¹

Comparison of Methods.—Comparative statements of the different methods deserve critical analysis, for the figures are not statistically comparable: they are obtained in different countries, at different times; recorded and edited in accordance with different plans. In some localities and at certain times rabies is much more virulent than in other localities and at other times. Even when all the factors are considered, the results of the Semple method remain favorable. Antirabic serum has been prepared by Marie's⁶² method but is weak and has feeble protective and no curative value.

Treatment at a Distance.—It is no longer necessary to go to a Pasteur

⁶⁰ No. 44, *Scientific Memoirs*, Govt. of India, 1911.

⁶¹ *Internat. Conf. on Rabies*, Health Sec., League of Nations, Suppl. to *Ann. de l'Inst. Pasteur*, 1928.

⁶² *Bull. de l'Inst. Pasteur*, 1908, 6: 705.

institute, for the virus may be sent by mail. In view of the stability of the material used in the Semple method, this is entirely practical.

Care during the Treatment.—During the treatment the patient may go about his usual business. It is not necessary to stay in bed. The patient should, however, avoid fatigue, cold, emotional stress, trauma, and alcohol. It has been shown that these are important predisposing factors to the disease. It was found that customs' officers returning to the Siberian borders after prophylactic treatment for wolf bites showed an unusual mortality, which seemed to be due to exposure to cold. The disease has been observed to occur after the shock of falling into water, or depressing influences.

Complications of the Treatment.—The Pasteur prophylactic treatment may be complicated by (1) local reactions or (2) paralysis.

Local reactions at the site of the wound are usually trivial. Abscesses do not occur. The local reactions consist of redness and induration. It is not necessarily the last injection, but rather the site of some previous injection that flares up, but soon subsides without further trouble. This occurrence increases with the progress of the treatment; it is most frequent in the second week. As the treatment involves the introduction of foreign proteins into the body, it is probable that these reactions represent a phase of anaphylaxis.

Paralysis.—Paralysis occurs occasionally and may be fatal. This complication seems to be a mild or modified type of rabies, but there is doubt concerning its cause. It is suggested that it may be due to infection with the fixed virus or possibly toxin, or to both. Paralysis may follow injections of nerve substances derived from a normal animal.⁶³ Serious paralysis is a rare complication; in mild form it doubtless occurs more often than is indicated by the records. It affects adults chiefly, young children almost never.

Paralysis, when it occurs, comes on during or shortly after the treatment. It usually consists of brief weakness of limited groups of muscles affecting the extremities or the face. Sometimes the complication is neuritic and occasionally paraplegic. Exceptionally, the paralysis is of the ascending or Landry's type and ends fatally when it affects the muscles of respiration. It is not easy to differentiate between this complication and true paralytic rabies which may occur during the course of treatment.

The frequency with which paralysis is reported varies very much. Simon collected data up to and including 1911 from various institutes showing that among 211,774 persons treated there were one hundred cases of paralysis, a rate of 0.48 per thousand, with nineteen deaths. The frequency of paralysis was found by Remlinger to be one in 1230, or 0.8 per thousand. Babes' figures are somewhat higher, 1.3 per thousand. Mejio,⁶⁴ in 1917, reported 19,800 cases treated at the Pasteur Institute, Buenos Aires, of whom twenty-four developed paralysis, with four deaths. From 1906 to 1921, the New York City Health Department treated 6,738 persons affected by rabid animals with a

⁶³ *Indian J. Med. Research*, 1926, 13: 467.

⁶⁴ *Semana méd.*, 1917, 24: 10.

total mortality of 0.47 per cent and a corrected mortality of 0.17 per cent. They had seven cases of paralysis with two deaths.

Simon⁶⁵ gives the following summary showing the incidence of this complication and the relationship between paralysis and the method of treatment.

Method	Number of Cases Treated	Cases of Paralysis	Proportion
Classic Pasteur method.....	32,676	6	1 in 5,446
Modified Pasteur method	8,657	16	1 in 541
Högyes method	51,417	3	1 in 17,139

The figures are not strictly comparable in view of the fact that they are not based upon uniform data.

All students of the subject are convinced that the Pasteur prophylactic treatment saves many lives by preventing rabies in persons bitten by mad dogs, but it should not be administered if unnecessary.

The Immunity.—*Duration.*—The immunity appears about two weeks after the treatment and lasts a varying period of time. In this respect it does not differ from other instances of acquired immunity. In one of Park's cases, it wore off in fourteen months, the patient dying of hydrophobia after having been bitten a second time. The Pasteur prophylactic treatment should therefore be repeated in persons bitten a second time. Marie found that dogs remained protected eighteen months after treatment.

Nature.—The nature of the immunity is not clear. It certainly is not due to an antitoxin. Immune bodies are demonstrable in the blood twenty days after the last injection. The activity of the virus can be neutralized by mixing it in vitro with the blood-serum of an immunized animal. This neutralization is generally considered to be microbicidal or lytic in nature.

Degree.—The degree of immunity varies. The Pasteur prophylactic usually affords definite protection, but that it sometimes fails is evidenced by the fact that a certain small percentage of persons treated die of rabies.

Mortality in Untreated Cases.—Faber found 27 deaths out of 339 persons bitten by mad dogs; Kurrimoto, 17 per cent in Nagasaki; Babes, 15 per cent of 995 in Hungary; Horsley's figures are 15 per cent. Some series of cases give a much higher mortality. Thus, of 855 persons bitten by mad dogs, collected by Tardieu, Thamehayn, and Bouley, 399 ended in death, or 46.6 per cent. In another series of cases given by Bouley, out of 266 persons bitten by mad dogs, 152 died of hydrophobia. But of these 120 were bitten on the face and hands, the greater danger of which has been mentioned. The mortality resulting from bites of wolves is placed by Babes at from 60 to 80 per cent.

Contra-Indications.—There are no known contra-indications to the treatment. All ages and conditions should be treated if exposed. Apparently no harm is done pregnant women. I have injected patients having malaria with-

⁶⁵ *Centralbl. f. Bakteriol.*, Orig., 1913, 68: 72.

out trouble following. The treatment may be continued in patients having colds, fevers, and other ailments without noticeable harm.

The Results of the Treatment.—Statistics giving the results of the treatment are somewhat difficult to analyze, as many factors are unobtainable. Patients should be kept under observation at least a year. Cases that occur within fifteen days after treatment are excluded from the French figures, for the reason that immunity is not expected to appear before that time. The figures on this basis show mortality of less than 0.5 per cent. Better results are being obtained from year to year.

RESULTS OF TREATMENT AT L'INSTITUT PASTEUR, PARIS

Year	Persons Treated	Deaths	Mortality per Cent	Year	Persons Treated	Deaths	Mortality per Cent
1886	2,671	25	0.94	1906	772	1	0.13
1887	1,770	14	0.79	1907	786	3	0.38
1888	1,622	9	0.55	1908	524	1	0.19
1889	1,830	7	0.38	1909	467	1	0.21
1890	1,540	5	0.32	1910	401	0	0.00
1891	1,559	4	0.25	1911	341	1	0.29
1892	1,790	4	0.22	1912	395	0	0.00
1893	1,648	6	0.36	1913	330	0	0.00
1894	1,387	7	0.50	1914	373	0	0.00
1895	1,520	5	0.38	1915	654	1	0.15
1896	1,308	4	0.30	1916	1,388	3	0.21
1897	1,521	6	0.39	1917	1,543	4	0.26
1898	1,465	3	0.20	1918	1,803	3	0.16
1899	1,614	4	0.25	1919	1,813	3	0.16
1900	1,420	4	0.28	1920	1,126	6	0.53
1901	1,321	5	0.38	1921	998	1	0.10
1902	1,005	2	0.18	1922	754	0	0.00
1903	628	2	0.32	1923	727	0	0.00
1904	755	3	0.39	1924	764	1	0.14
1905	727	3	0.41	1925	782	0	0.00

When to Give the Pasteur Prophylactic.—It is sometimes difficult to decide whether the Pasteur prophylactic treatment should be given. Treatment causes sufficient personal inconvenience, not to speak of the danger (however slight) of paralysis, to avoid advising it if unnecessary. In many cases it is impossible to discover whether the dog that inflicted the bite is mad or not. The rule in cases of doubtful exposure is to advise the treatment.

Persons who apply for treatment of dog bites fall into one of the seven following categories with reference to the Pasteur prophylactic:

1. The dog is mad: In this case, begin treatment at once.
2. The dog shows suggestive symptoms: Give the treatment at once; in communities having skilled laboratory facilities wait for diagnosis, provided this does not take more than a few days.
3. The dog is not mad: Observe it carefully for ten days, and if no symptoms develop, there is no danger of rabies in the person bitten. The treatment is then unnecessary. (The dog may develop rabies after ten days and if it has been bitten by another dog should be kept in quarantine for six months).

4. The dog is not identified: This is a common occurrence, especially with children. The rule in such cases is to advise the Pasteur prophylactic treatment, except in regions known to be free of rabies.

5. Exposure to saliva: Persons not infrequently apply for advice giving the following history: They have not been bitten, but they have been licked on the hands and face by a dog that subsequently was discovered to have the disease. Persons are sometimes similarly exposed by washing the mouth of a rabid horse. In these cases the important question is whether there were fissures or abrasions in the skin at the time. There may be little wounds in the skin not evident to the naked eye. It is possible to infect animals by rubbing the virus on the shaved skin. The rule is therefore to advise the protection which the treatment affords in persons thus exposed.

6. In psychoneurotic patients with a distressing phobia of rabies, it may afford comfort to give a mild course of treatment as much for its psychotherapeutic effect as for specific immunity.

7. Fomites: The question is often asked whether the disease may not be contracted from contact with virus in saliva upon floors, on playthings and other objects. This situation arises with a rabid dog in the house, where children may be exposed in this indirect manner. While theoretically possible, the danger is small; in fact, I have never heard of a case contracted in any such way.

CONTROL OF RABIES IN DOGS

Rabies is a disease primarily of dogs, secondarily of man. It is kept alive in nature mainly by the dog and the dog family. The disease may be controlled, even exterminated, by intelligent measures directed towards dogs. Attacking the disease along these lines is logical, practical, effective and permanent. The problem cannot be settled locally, but requires nation-wide action. The chief requirements are federal laws regulating the importation of dogs, the compulsory impounding of all stray animals, and the proper supervision of all licensed dogs. Other prophylactic measures necessary to control the dog question are muzzling and restraint, licensing, legal responsibility of owners, quarantine, immunization, etc.

Muzzling.—Consistent muzzling of all dogs for two years would practically exterminate rabies. There is difficulty in getting consent for this requirement on account of sympathy for the dog. It should be recalled that the measures proposed are for the good of the dogs themselves. In England when the dogs were muzzled rabies diminished, but the law was repealed due to misplaced sympathy for the dog, and the disease reappeared. The law was again enforced, and in about two years rabies disappeared.

Quarantine.—On account of the long period of incubation, the quarantine should be under veterinary supervision for no less than six months. In Australia, where there are few carnivorous animals, rabies does not exist, for it has been kept out owing to early and effective quarantine measures. A strict quarantine of six months is maintained against dogs entering England. This

was broken during the War by the carrying of dogs across the Channel in aeroplanes, and rabies reappeared in 1918.

Licensing and Restraint.—*The Stray Dog.*—All dogs should be licensed and required to wear a tag, and under certain circumstances restrained by chain or leash. Stray dogs should be impounded and ownerless dogs killed. It is mainly the stray dog that keeps rabies alive. A high tax helps limit the number of dogs. Muzzling and other mechanical measures of restraint become unnecessary when rabies is under control.

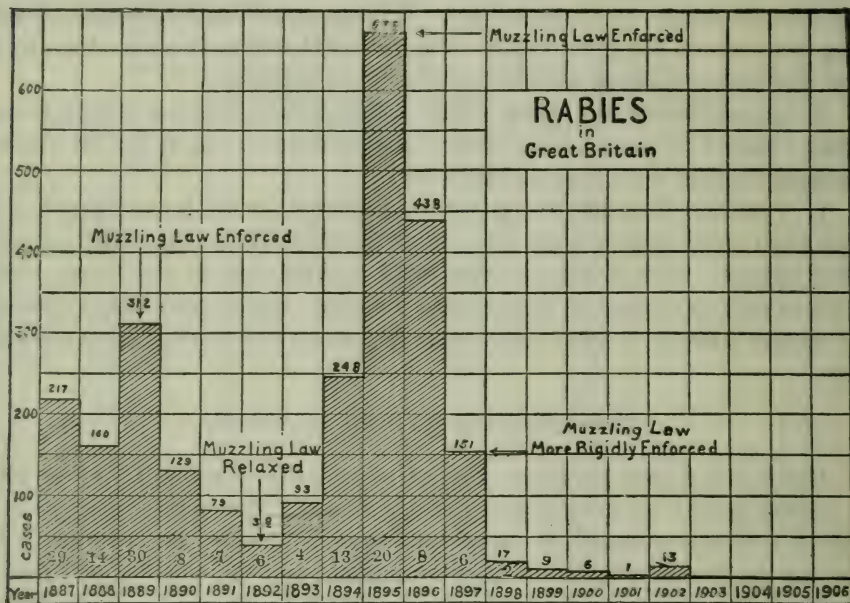


FIG. 6.—CHART SHOWING RELATION OF ENFORCEMENT OF MUZZLING LAW TO PREVALENCE OF RABIES IN GREAT BRITAIN.

The figures in the cross-hatching indicate the number of persons who died of rabies in England and Wales. The ordinates represent cases in dogs. (Straus and Frothingham.)

Education and Responsibility.—Owners should be held legally responsible for damage inflicted by their dogs. Education of the dog-owning public will help toward preventing the spread of communicable diseases, especially rabies. All cases of suspected rabies should be promptly reported.

Immunization.—Prophylactic immunization of dogs against rabies was suggested by Pasteur and has recently been used in Japan with good results. Umeno and Dio⁶⁶ found it possible to confer immunity on dogs with a single injection of a large dose of phenolized fixed virus. Up to 1921, 31,307 dogs were vaccinated in the prefectures of Kanagawa and Tokio with only one case of rabies resulting from natural exposure. On the other hand, the disease con-

⁶⁶ Kitasato Arch. Exper. Med., 1921, 4: 89.

tinued to rage among the unvaccinated animals. Hata⁶⁷ gives the latest Japanese results with Umeno's prophylactic method. Of 104,629 dogs immunized against rabies in Tokio and Yokohama, only forty-one developed rabies, while 1,699 of the uninoculated group contracted the disease, notwithstanding the fact that the latter group represented only one-third of the total number of dogs in the two prefectures. It therefore appears that dogs can readily be immunized against rabies. Just how long the protection lasts is not known, probably on the average at least a year. It may be renewed. The immunization of all dogs when licensed and the impounding or killing of all unlicensed dogs should effectively control rabies.

The immunity following repeated injections lasts longer. Thus, Pasteur kept many dogs under observation and tested them from time to time. In one dog the immunity lasted five years. Högyes kept eight dogs and found that the immunity lasted from four to seven years. The usual limit is probably between three and five years.

Handling of Dogs That Have Bitten Persons or Animals.—Such dogs should not be killed unless it is clear they have symptoms of rabies, and then only if they cannot be apprehended with safety. It is important to know whether the dog is mad. If the dog can be found and kept under observation for ten days and no symptoms appear, the Pasteur treatment is not necessary. The dog should be turned over to the proper authority for observation and study. Animals killed early in the course of rabies may fail to show microscopic evidence of the disease, thus causing a delay in diagnosis. If killed, the head or brain should be sent to a laboratory without delay, and if at a distance on ice. When this is not practicable, the brain should be placed in glycerin.

Diagnosis of Rabies in Dogs.—The diagnosis of rabies in dogs may be made in four ways: (1) from the symptoms; (2) from the presence of Negri bodies in the central nervous system; (3) from the lesions in the peripheral ganglia, and (4) by animal inoculations.

1. The *symptoms* may be very suggestive, but a diagnosis must always rest upon the pathological lesions or the inoculation tests. The course of the disease may be divided into three stages: (*a*) a premonitory stage, (*b*) a stage of excitement, and (*c*) a paralytic stage. The first two stages may be absent or transient. All rabid animals invariably become paralyzed before they die. In dogs the first symptom consists solely in a change in the disposition of the animal. He is easily excited, but does not show a tendency to bite. Soon the restlessness becomes more marked, and the animal may become furious and even show signs of delirium. The dog does not fear water, as is commonly supposed, but rushes about attacking every object in his way. Dogs suffering from furious rabies have a tendency to run long distances (twenty-five miles or more), often biting and inoculating large numbers of other animals and persons en route. Very soon paralysis sets in, commencing in the hind legs, and finally becomes general. The course of the disease is always rapid, averag-

⁶⁷ *J. Immunol.*, 1924, 9: 89.

ing from four to five days, rarely exceeding ten days. When the stage of excitement is brief or absent, the disease is known as dumb rabies.

2. There is a difference of opinion concerning the significance of the Negri bodies (*Neurorrhcytes hydrophobiae*), which, however, are very constant in rabies and peculiar to it. If Negri bodies are found in the dog, the Pasteur treatment should be started at once. The absence of Negri bodies, however, does not necessarily mean the absence of rabies. These bodies are sometimes difficult to find, or may not be present in the parts of the central nervous system which are examined. Negri bodies are found especially in the horn of Ammon and the cerebellum; they are 1 to 23 micra in diameter; usually round or oval; strongly eosinophilic; occur within and without the nerve cells; and sometimes contain a nucleus (?). Owing to their resemblance to red blood cells, the finding of Negri bodies *within* the cells is the safest criterion. The diagnosis may thus be established in about 90 per cent of cases.

Negri bodies for diagnostic purposes are best demonstrated by impression preparations of Ammon's horn and cerebellum, stained according to Van Giesen as recommended by Frothingham or stained by Bond's modification of the Mann stain. Impression preparations are made by gently pressing a microscopic slide upon the cut surface of Ammon's horn and the cerebellum and lifting with a quick movement. Care should be taken to obtain thin uniform impressions because thick impressions do not show differential staining for Negri bodies. Pieces of the Ammon's horn and the cerebellum selected for impressions should be from four to six millimeters thick. Three or four impressions to a single slide should be made from each piece of the Ammon's horn and cerebellum. Four or five pieces of each Ammon's horn and the same number from the cerebellum are sufficient. The impression preparations obtained in this way show the characteristic arrangement of the cells of the hippocampus and of the cerebellum and rarely fail to contain the Negri bodies in infected material. When the brain is badly mutilated or decomposed, impressions taken from any of the available material containing gray matter will frequently show the Negri bodies if infected.

To stain impression preparations as recommended by Frothingham the following slightly modified procedure is given: (1) Fix before the impression dries in methyl alcohol for five minutes; (2) stain at room temperature with Van Giesen, while still moist with alcohol, for eight to ten minutes; (3) wash thirty seconds with running tap water; (4) blot with filter paper. The Van Giesen stain is made as follows: Tap water 20 c.c. to 50 c.c.; saturated alcoholic fuchsin (f. Bac. Grubler) 1 drop; saturated aqueous solution methylene blue (f. Bac. Koch Grubler) one to ten drops. The amount of tap water and the amount of methylene-blue required for good differentiation vary with different stock solutions of the stains. This stain changes little in three to four days.

To stain by Bond's modification of the Mann stain proceed as follows: (1) Fix before the impression dries in methyl alcohol five to six minutes; (2) wash thirty seconds with running tap water; (3) stain for four to five minutes with

a mixture consisting of 1.0 c.c. of 1 per cent aqueous eosin (Eosin W. gelbl. Grubler), 0.7 c.c. to 1.0 c.c. of 1 per cent aqueous methyl blue (Grubler) and 6.0 c.c. of distilled water; (4) wash with running tap water for thirty seconds; (5) blot with filter paper; (6) dehydrate with absolute alcohol; (7) clear with a mixture consisting of one part of xylol and two parts of anilin oil; (8) wash with xylol; (9) mount in balsam. The Mann stain should be freshly prepared each time it is used. In practice it is better to stain by both methods because each has its advantages and disadvantages. The Mann stain gives definite Negri bodies but the differential staining between the red blood-corpuscles and Negri bodies is not always clear. With the Frothingham method Negri bodies can hardly be mistaken for anything else but the stain itself may be capricious in action and show relatively fewer Negri bodies.

3. The lesions of Van Gehuchten and Nélis, described in 1900, are the most characteristic anatomical changes. These lesions are found late in the disease in the peripheral ganglia of the cerebrospinal and sympathetic systems, especially in the plexiform ganglia of the pneumogastric nerve, and the Gasserian ganglia. The normal nerve cells of these ganglia lie in a capsule lined with a single layer of endothelial cells. In rabies these endothelial cells proliferate and the nerve cells may be partly or entirely destroyed and replaced by cells associated with chronic inflammatory processes. In addition, lymphatic infiltration also occurs about the sheaths surrounding the individual nerve cells. Either the proliferative or infiltrative changes may predominate. In order to find these lesions, it is necessary to fix the ganglia in Zenker's fluid and to stain the sections by the eosin-methylene-blue method.

4. The final diagnosis of rabies rests upon animal experimentation. A small quantity of an emulsion of the medulla or pons of the suspected animal is placed under the dura mater of a rabbit or guinea-pig. The diagnosis by this method, however, requires so much time (on account of the long period of incubation of the disease) that it is of no practical value in deciding whether or not the Pasteur prophylactic treatment should be given, but in any critical case the positive evidence furnished by animal experimentation is incontrovertible.

If the inoculated rabbit shows no symptoms in two months and Negri bodies were not seen in the specimen, then a negative diagnosis may be given, although it is customary to observe the animal for six months.

REFERENCES

- PASTEUR, CHAMBERLAND, ROUX and THUILLIER. Note "Sur la rage," *Compt. rend. Acad. de sc.*, 1881, 92:1555.
- PASTEUR, CHAMBERLAND and ROUX. "Nouvelle communication sur la rage," *Compt. rend. Acad. de sc.*, 1884, 98:457; "Sur la rage," *Compt. rend. Acad. de sc.*, 1884, 98:1229.
- PASTEUR. "Méthode pour prévenir la rage après morsure," *Compt. rend. Acad. de sc.*, 1885, 101:765; "Résultats de l'application de la méthode pour prévenir la rage après morsure," *Compt. rend. Acad. de sc.*, 1886, 102:459; "Note complémentaire sur les résultats de l'application de la méthode de prophylaxie de la rage après morsure," *Compt. rend. Acad. de*

sc., 1886, 102:835; "Nouvelle communication sur la rage," *Compt. rend. Acad. de sc.*, 1886, 103:777; "Lettre sur la rage," *Ann. de l'Inst. Pasteur*, 1887, 1:1; "Sur la méthode de prophylaxie de la rage après morsure," *Compt. rend. Acad. de sc.*, 1889, 108:1228.

BABES, VICTOR. *Traité de la rage*, Bailliére et Fils, Paris, 1912. A comprehensive monograph. Contains a good historical account of the story of Pasteur's discovery, with original references.

STIMSON, A. M. "Facts and Problems of Rabies," *Hygienic Laboratory Bulletin*, No. 65, June, 1910. Contains a selected bibliography.

More comprehensive bibliographies will be found in:

HELLER. *Schutzimpfung gegen Lyssa*, 1906.

HÖGYES. "Lyssa" in *Nothnagel's Spez. Path. u. Therap.*, Wien, 1897.

MARIE. *L'étude expérimentale de la rage*. *Encyclop. scient.*, 1909.

MARX, E. *Kolle u. Wassermanns Handb. d. path. Mikroörg.*, 1904, 4, Bd., 2. Tl., 1264.

VENEREAL DISEASES ⁶⁸

As a danger to the public health, as a peril to the family, and as a menace to the vitality, health, and physical progress of the race, the venereal diseases are justly regarded as the greatest of modern plagues, and their prophylaxis the most pressing problem of preventive medicine that confronts us at the present day.

No serious attempt was made by the sanitary authorities of any of our great cities to deal with this problem until New York City in 1912 ⁶⁹ determined to treat the venereal diseases as any other highly communicable and preventable infection, dealing purely with the sanitary features of the problem from a public health standpoint, ignoring the social and moral phases. The opposition to such activity is slowly being broken down. Progress against the venereal diseases is a repetition of the warfare along other lines of sanitation and hygiene. It is the history of a continuous struggle carried on in the name of law, religion, personal rights, or expediency. Although the difficulties in this case are much greater than in any other group of diseases, an intelligent and persistent campaign must end in a long-delayed success.

Biggs states that in 1912 at least 800,000 people, or more than one-fifth of the adult population of New York City, had, or had had, some venereal disease, and that in a large percentage of these persons the disease is still active. The number of new infections occurring each year probably exceeds that of all other notifiable diseases combined. In view of such figures the magnitude and the importance of the problem of administrative control, as applied to these diseases, become clearly apparent.

The venereal diseases are a constant menace to the clean-living public as

⁶⁸ Objection has been made to the stigma implied in the term venereal diseases, for these infections are not always transmitted in venery and are often contracted innocently. Syphilis and gonorrhea, however, are unlike other communicable diseases in that they involve a moral principle. Gonorrhea or syphilis contracted innocently is usually only one remove from promiscuity.

Only one venereal disease among the lower animals is known,—dourine, a syphilis of horses caused by a trypanosome.

⁶⁹ Resolutions adopted by the Board of Health, February 20th.

well as to the licentious. The history of preventive medicine can present no greater tragedy than the home invaded by syphilis or gonorrhea.

There are three venereal diseases: syphilis, gonorrhea, and chancroid. In order to have a clear understanding of the problems of venereal prophylaxis it is necessary to have a knowledge of the essential features of these preventable infections. Two of them, syphilis and gonorrhea, are of great importance, because they are very prevalent and because they are very serious infections with grave consequences. Gonorrhea is the great preventer, syphilis the great destroyer of life.

SYPHILIS

Syphilis is a specific infection caused by the *Treponema pallidum*.⁷⁰ It is acquired by direct contact with infected persons, by inoculation with infected things, and by congenital transmission. Syphilis runs a chronic course with lesions and symptoms of extraordinary diversity. The initial lesion or chancre forms on the skin or mucous membrane at the site of entrance of the spirochetes. The period of incubation is never less than ten days, with a maximum of ninety days. In the majority of cases, the chancre appears between the eighteenth and fortieth days; usually in the third or fourth week.

There are many striking things about syphilis, but nothing so striking as its persistence in spite of knowledge complete enough to stamp it out and in view of the popular dread in which the disease is held. Syphilis causes more mental and physical suffering than any other disease in the whole category. It is preventable, even curable—yet scarcely another disease equals it in the extent and intensity of its ravages. It is the great canker of humanity.

Syphilis is a good illustration of the fact that it is much more difficult to control a disease transmitted directly from man to man than a disease transmitted by an intermediate host, or one in which the virus is transferred through our environment. We have a certain amount of control over our surroundings, and we have dominion over the lower animals, but the control of man requires the consent of the governed.

Civilization and syphilization have been close companions, but syphilis is now less prevalent among civilized than uncivilized peoples—this is promising. Civilization, however, should not be content until it has controlled syphilis as effectively as it has some other preventable infections. The effort to do so, at least, must be persistent and sincere.

From the economic side syphilis is not a serious disease in its primary and secondary stages; that is, persons with syphilis during the early stages are usually not ill enough to cease work.⁷¹ Acutely fatal cases, such as occurred in the sixteenth century, are now rare; in other words, the disease has lost some of its early virulence. It is the late manifestations, or the so-called parasyphilitic lesions, as well as the inherited consequences of the disease, that play

⁷⁰ First described by Schaudinn and Hoffmann in 1905 and called by them *Spirochæta pallida*.

⁷¹ But it is most communicable during this time.

havoc. About one-eighth of all the insane in our asylums are cases of general paresis; 90 per cent of these give a positive Wassermann reaction. Syphilis, alcohol, and heredity fill our insane asylums, jails and almshouses.

The consequences of syphilis are often more severe upon the offspring than upon the syphilitic parent. The infection itself, or various defects, especially of the central nervous system, resulting from the consequences of syphilis, may be transmitted from parent to child, often with fatal results. When death does not ensue the results may be still more tragic. The infection itself may be transmitted to the third generation.

The health officer should regard syphilis just as he does the acute febrile exanthematous diseases. Because syphilis runs a slow and often chronic course with mild constitutional symptoms during its early stages, it is often placed in a class by itself. This is a mistake. Syphilis has its period of incubation, eruption, and decline, just as measles and smallpox have.

Historical Note.—There is an accurate historical record of the startling spread of syphilis over the known world in a few years after 1495, and from that time it has everywhere been endemic. No similar record exists of the sudden establishment of any other great disease among the larger part of the earth's inhabitants. Evidence points to the severe character of the disease during this early epidemic, the cases often running an acute, febrile course, accompanied by symptoms of such severity as are now seen only occasionally. Syphilis was unknown as a clinical entity before the year 1493. It is said to have been brought by the crew of Columbus, on his first voyage from Española, or Hayti. Some of the returning crew accompanied Charles VIII of France in the autumn of 1494 with the army, 32,000 strong, which invaded Italy for the conquest of Naples. The epidemic began in Italy at this time, and the disease spread quickly over Europe with the scattering of the troops. The French called it the Italian disease, and the Italians called it the French pox, or *morbus gallicus*. The name of the disease was taken from a poem written by Fracastor in 1530 entitled, "*Syphilus sive Morbus Gallicus*," in which the symptoms are clearly described in the principal pastoral character—*Syphilus*.

Bearing on the American origin of syphilis, bones from pre-Columbian graves offer evidence that syphilis was present among the aborigines before the Spaniards came to America.⁷² On the other hand, Virchow was unable to find lesions of syphilis in European and Oriental bones of the pre-Columbian age. It is said, however, that the Chinese, more than 2,000 years B.C., had knowledge of this disease and suffered from it. The sexual nature of the infection was not recognized until some time after the epidemic outburst in 1494-1496. The history of the disease before this time is shrouded in difficulties.

In 1903, Metchnikoff and Roux⁷³ transmitted the disease to lower animals and demonstrated the prophylactic value of calomel inunctions, and

⁷² B. P. Thom, *Syphilis*, Lea and Febiger, Philadelphia, 1922.

⁷³ *Ann. de l'Inst. Pasteur*, 1903, 17: 809.

also opened up a rich field of animal experimentation. In 1905, Schaudinn⁷⁴ discovered the cause and thus made diagnosis certain. In 1906, Wassermann, Neisser and Bruck⁷⁵ introduced the indirect method of diagnosis by serum reaction. In 1910, Ehrlich,⁷⁶ after many years of experiment, gave to the world salvarsan, a specific, synthetic spirocheticide. In 1911, Noguchi⁷⁷ cultivated *Treponema pallidum* outside of the body and prepared luetin. In 1913, Noguchi⁷⁸ demonstrated the spirochetes in the brain of paretics and in the cord of a tabetic. This unparalleled group of achievements, all the result of scientific work in laboratories, in ten short years threw light upon the cause, mode of transmission, pathology, treatment and prevention of the disease.

Prevalence.—The percentage of syphilitics in the population at large is difficult to determine. It is commonly estimated at about 8 per cent. The amount of infection in certain groups is given by Vedder as follows: Prostitutes, 50 to 100 per cent; tuberculous in institutions, 20 to 30 per cent;⁷⁹ sick children in hospitals, 2 to 10 per cent; mentally backward and idiots, 20 to 40 per cent;⁸⁰ criminals, 20 to 40 per cent; presumably healthy men of the class that enlist in the regular army, 20 per cent: this group represents unskilled labor and a certain percentage of the tradesmen. Among men of better families the percentage varies from 2 to 10 per cent, depending upon age, marital condition and other factors. Among young women in the community, the percentage of syphilitic infections fluctuates between 3 and 20 per cent, depending upon age, marital condition, education and social status. As among men, the proportion of infections increases as we descend in the social scale. It is estimated that the rates for the colored race are more than double those for the white race (see also page 69).

Stages of the Disease.—Syphilis is divided into four stages which are not always well defined in time or sequence.

1. *The Chancre.*—The *primary stage* consists of the chancre which forms at the site of the initial infection. The regional lymph-nodes become enlarged and hard. The typical Hunterian chancre is an indurated and indolent ulcer in the skin or mucous membrane, and appears about three weeks (not less than ten days) after the receipt of the infection. It is usually single and painless, but frequently is atypical, and may be but a trifling lesion. In the absence of a careful daily inspection, it may exist many days before it is detected, or even escape notice altogether, or may be masked by gonorrhea. The infection becomes fully established even before the ap-

⁷⁴ *Arch. a. d. k. Gsndhtsamte*, 1905, 22: 527.

⁷⁵ *Deutsch. med. Wchnschr.*, 1906, 32: 745.

⁷⁶ *Die experimentelle Chemotherapie der Spirilloesen*, 1910, Julius Springer, Berlin.

⁷⁷ *J. Exper. M.*, 1911, 13: 557.

⁷⁸ *J. Exper. M.*, 1913, 17: 232.

⁷⁹ The prevalence of syphilis among the tuberculous in this country has been variously placed by different investigators on the basis of a clinical examination or a positive Wassermann, or both. Some of these are as follows: Vedder, 23.2 per cent; Snow and Cooper, 20 per cent; Petroff, 21.8 per cent; Lyons, 9.2 per cent; Jones, 29 per cent.

⁸⁰ Recent studies have shown that in the state hospitals of Massachusetts and New York, it is less than 5 per cent.

pearance of the chancre, for by the time the initial lesion becomes evident, treponema are found in the lymph-nodes, spleen and bone marrow.

The chancre contains many spirochetes which may readily be seen with the dark field illumination. This is the best method of early diagnosis. The Wassermann reaction usually does not become positive until from two to six weeks after the appearance of the chancre. It is important to examine every sore on the genitalia for spirochetes, for the initial lesion of syphilis often resembles a chancroid, sometimes only a simple abrasion. Mixed infections occur.

2. The *secondary stage* usually appears about six weeks after the appearance of the chancre. The symptoms are eruptions upon the skin and mucous membranes, fever, anemia and other indications of a generalized infection. The hair falls out, the lymph-nodes are enlarged but painless; headache, pains in the joints and bones, and sore throat are common. This stage lasts from a few months to several years, but in a large per cent of the cases the manifestations are so mild as to escape unnoticed.

3. The *tertiary stage* is subject to all possible variations. It usually begins some three years from the time of the initial lesion and lasts indefinitely. Visceral manifestations are always late, and then the gumma, which is a local granulomatous growth, may appear in almost any tissue or organ of the body.

4. A *fourth stage*, consisting of inflammatory and degenerative lesions of the heart, blood-vessels and central nervous system, is often added to the picture, and occurs long years after the primary sore. This stage, formerly regarded as sequelæ or parasymphilitic phenomena, is now known to be associated with spirochetes, and therefore is part of the disease. This is the most serious and disabling stage of the disease and is a frequent cause of insanity or premature death. Examples of the chief manifestations of the late stage of syphilis are locomotor ataxia and dementia paralytica, also arteriosclerosis, aneurysm, cerebrospinal syphilis, etc. The prevention of these serious sequelæ depends upon early recognition of the chancre, followed by prompt and thorough treatment.

Neurosyphilis may occur independently of any so-called stage, and is seen from the earliest days of the disease to the last. Meningeal involvement has been observed as early as two or three months after the chancre.

The term "latent syphilis," long used, is a misnomer, and the term "concealed syphilis" is being substituted, for microscopic studies demonstrate that syphilitic changes are steadily going on in some deep structure, as the aorta or meninges.

Fatality.—Syphilis is a frequent cause of death in early adult life in persons otherwise hale and hearty.

If it be remembered that syphilis is the real cause of death in all cases of general paresis, locomotor ataxia, and aortic aneurysm, in many cases of apoplexy, and is a contributory cause of death in a host of other conditions, including many cases of pulmonary tuberculosis, the real influence of syphilis

on the mortality rate begins to be suspected. Osler some time ago made the statement that "of the killing diseases, syphilis comes third or fourth." But later from an analysis of the Registrar General's statistics for 1915, he estimated the actual deaths from syphilis in England and Wales at about 60,000, thus moving syphilis to the top of the list. Leredde estimates that syphilis probably kills 25,000 persons each year in France.

According to Lenz, in the large cities, 25 per cent of syphilitics die as the result of endarteritis (angina pectoris, aortic insufficiency, aneurysm), while 3 or 4 per cent of syphilitics die from general paralysis, 1 or 2 per cent from tabes and at least 10 per cent more as the result of syphilitic lesions of the brain, liver and kidneys. Almost half of all syphilitics eventually succumb as the result of their infection. Syphilis is therefore the greatest cause of premature death of men in large cities.

Mattauschek and Pilcz⁸¹ found that of 4134 officers of the Austrian Army who contracted syphilis between the years 1890-1900, on January 1, 1912: 198 had general paralysis; 113 had locomotor ataxia; 132 had cerebrospinal syphilis; 80 suffered from different psychoses; 17 died of aneurysm; 147 died of tuberculosis; 20 died with syphilis designated as the cause; 101 developed myocarditis and arteriosclerosis, 86 of whom died from this condition.

Diagnosis.—Early diagnosis and prompt treatment are the most practical and promising measures to control syphilis (page 72). The clinical symptoms are often atypical and elusive; reliance on laboratory tests is therefore imperative.

Dark field illumination should be applied as a routine to every genital sore, by an expert. A single negative finding is not conclusive, and should be repeated daily for several days. No local antiseptic should be applied until the diagnosis is established. One application of a spirocheticide, such as silver nitrate, mercury, or copper sulphate, is often sufficient to cause the disappearance of the spirochetes from the surface of the sore.

The Wassermann reaction becomes positive only after a general invasion of the spirochetes takes place. At the time of the first appearance of the chancre this reaction is invariably negative. It appears by the tenth day in about 30 per cent of cases, although it has occasionally been noted as early as three days after the initial lesion appeared. It is positive in 70 per cent by the end of the third week, and in 96 per cent of all cases by the fortieth day. The Wassermann reaction therefore cannot be depended upon for early diagnosis, but soon becomes the most reliable laboratory test. It should be remembered, however, that in a small percentage of syphilitics, the Wassermann reaction remains negative, while on the other hand it may become positive in other infections, such as yaws, and sometimes in malaria, pneumonia, scarlet fever, and especially in diseases associated with deranged metabolism of the liver. We must be cautious in drawing conclusions from

⁸¹ *Med. Klin.*, 1913, 9: 1544; also *Berl. klin. Wchnschr.*, 1908, 45: 1213.

positive Wassermanns in other diseases, for it is often difficult to rule out syphilis.

The *luetin test* is an anaphylactic reaction depending upon sensitization of the skin. It does not always appear, and then late in the disease. It is more useful in prognosis and as a guide to treatment than in diagnosis.

Methods of Transmission.—Syphilis is transmitted directly, indirectly, and congenitally.

In a large majority of all cases of syphilis, the infection is transmitted during sexual approach and usually as a consequence of adulterous relations. It is, therefore, usually a venereal disease; many cases, however, are contracted out of venery. These innocent infections are more common than is ordinarily supposed. The spirochete is an animated corkscrew and can probably penetrate the unbroken mucous membrane⁸² and perhaps the skin, although a fissure or slight abrasion is the site of most chancres.

Marital Syphilis.—The subject is of great interest and importance because marital syphilis is so frequent, and because the individual so infected is an innocent victim of the disease, and usually remains ignorant of the infection, and therefore receives little or no treatment. The transmission of syphilis from wife to husband is comparatively rare. On the other hand, the transmission of disease from husband to wife is comparatively common. M. Dechambre says, "Syphilis is divided among husband and wife like the daily bread."

Extragenital Chancres.—Extragenital chancres constitute from 5 to 10 per cent of the total infections with syphilis. Metchnikoff reports that a great number of cases of non-venereal syphilis occur among children in Russia, where peasants live huddled together and in ignorance. Genital syphilis is not necessarily due to immorality, since it frequently results from marital relations, and has occasionally followed the rite of circumcision. Extragenital chancres are usually acquired innocently but may be the result of improper practices. It is particularly desirable to make this distinction from the public health standpoint, as the measures taken to prevent syphilis resulting from immorality and syphilis acquired accidentally are naturally quite different. Innocent syphilis, however, is generally the result by one or two removes of syphilis acquired by promiscuity.

Bulkley,⁸³ Munchheimer, and Fournier⁸⁴ have collected from the literature 20,000 cases of extragenital chancre; Scheuer⁸⁵ has analyzed 14,590 of these in regard to location: 3880 occurred on the lips, contracted mainly by kissing; 2144 on the arm, caused by the old-fashioned arm-to-arm vaccination; 1569 occurred on the breast, mainly of healthy wet nurses from syphilitic infants; 1104 on the tonsils; 897 on fingers and hands, chiefly of physicians,

⁸² Brown and Pearce obtained infection in rabbits through the conjunctiva, but the disease so produced tended to assume a mild or asymptomatic course (*J. Exper. M.*, 1924, 34: 645).

⁸³ *Syphilis in the Innocent*, Bailey and Fairchild, New York, 1894, p. 197.

⁸⁴ *Les Chancres Extragenitaux*, Paris, 1897.

⁸⁵ *Die Syphilis der Unschuldigen*, Berlin, 1910, Urban and Schwarzenberg.

nurses and midwives; 753 were caused by circumcision, 181 by cupping, and 109 by tattooing.

Kissing.—It is difficult to treat this subject without a sense of humor, and yet it is the most important single method by which accidental syphilitic infection is transmitted. When the public understands that not only syphilis, but also pneumonia, influenza, common colds, sore throats, measles, scarlet fever, whooping-cough, diphtheria, and many other infections may be transmitted through kissing, the practice will become automatically reduced to normal and proper limits. The danger from kissing is great when there are mucous patches or other open lesions upon the mucous membrane of the mouth. The kissing party reported by Schamberg⁸⁶ has become classic: Eight girls acquired chancres of the lip from kissing a young man who had a chancre of the lip. The percentage of infection was very high—eight were infected, only five or six escaped.

Indirect Transmission.—The list of articles that have conveyed the contagion by indirect transmission is comprehensive and includes towels, clothing, razors, handkerchiefs, surgical and dental instruments, pipes, etc.; a considerable number of infections have been traced to barber shops, drinking glasses, and minor operations.

For congenital and hereditary transmission of syphilis, see pages 583 and 586.

Infectiousness of Lesions and Tissues.—The *chancre* is highly infectious from its first appearance until it is completely healed. Hence the importance of early diagnosis and prompt treatment.

All *secondary lesions* are potentially infectious because spirochetes have been demonstrated in all of them. The mucous patch is the secondary lesion most commonly responsible for the transmission of the disease. Like the chancre, it fairly teems with spirochetes, it is comparatively painless, and occurring on the mouth or genitalia it occupies the two regions of the body most commonly brought into close and intimate contact with persons of the opposite sex. The vast majority of infections are acquired from syphilitics in the primary or early secondary stages of the disease. Nevertheless, we must regard any uncured syphilitic as a possible source of infection. The secondary stage with its endless lesions may last for twenty-nine years.

Tertiary lesions are infectious, although it was thought for many years that they were not. Practically, cases of infection from tertiary lesions are comparatively rare and only occur from late eruptions of the skin and superficial gummatous ulcerations.

The infectiousness of the *blood* has been demonstrated in all stages of syphilis, but the danger from this source is slight. An exception to this statement is to be noted in the case of surgeons, physicians, nurses, dentists and midwives, among whom syphilis is an occupational disease.

The *milk* of a syphilitic woman must be regarded as infectious, since it is well known that a syphilitic wet nurse without lesions will almost surely

⁸⁶ *J. Am. M. Ass.*, 1911, 57: 783.

infect a healthy child; in such instances, however, the infection is usually not from the milk.

Spermatic Fluid.—Pinard and Hoch⁸⁷ found syphilitic spirochetes in three of eleven specimens of human sperm, and Lackaye⁸⁸ in five of twenty-two specimens examined. They occur either free or attached to the spermatozoön.

Warthin's findings⁸⁹ demonstrate that the seminal fluid from many secondary cases is infectious, and we may assume that the seminal fluid from tertiary cases may be infectious in the presence of a suitable lesion in the testicles, and this is confirmed by clinical experience, which indicates that some cases of syphilis appear to be transmitted in this way.

Spinal fluid contains spirochetes in many cases in which the central nervous system is involved. The *sputum*, *sweat* and *urine* are generally believed not to be infectious, except in the presence of discharging lesions.

For public health purposes, a person suffering from syphilis is considered to be in an infective stage so long as he shows any symptom or lesion of primary or secondary syphilis, or any discharging lesion of the tertiary stage.

Viability.—The spirochete of syphilis is a frail organism, yet it may live long enough on towels, glasses, razors, dental instruments, pipes, and other objects to command hygienic respect. Thus, Zinsser and Hopkins⁹⁰ found that pure cultures lived eleven and a half hours on a moist towel. Dried on covered slips, the spirochete failed to grow after one hour. Bronfenbrenner and Noguchi⁹¹ found that the viability of the spirochete is markedly diminished by lack of nutritive substances, presence of oxygen, effect of light, and the toxic effect of sodium chlorid. Gastou and Comandon⁹² recovered living and active spirochetes from drinking glasses up to half an hour after they were deposited on the glass.

Hertmanni⁹³ found that the spirochete lost its motility as soon as drying occurred. Neisser⁹⁴ also determined that the virus from syphilitics that produced syphilis when inoculated into monkeys lost its power to transmit infection as soon as the fluid which contained the organism was dried. These facts are in accordance with clinical experience, which indicates that infection by indirect contact is through a brief period of time or a short interval of space. Any object recently soiled or still moist with infectious secretions from a syphilitic must be regarded as a possible source of infection.

Treponema pallidum may live and retain its infectious properties for long periods in tissues that are excised from the body and in various bodily fluids when kept in the laboratory under suitable conditions or in cultures.

⁸⁷ Bull. et. mém. Soc. méd. d. Hôp. de Par., May 28, 1920.

⁸⁸ Arch. méd. belges., 1922, 75: 385.

⁸⁹ Am. J. M. Sc., 1916, 142: 508.

⁹⁰ J. Am. M. Ass., 1914, 62: 23.

⁹¹ J. Pharmacol. & Exper. Therap., 1913, 4: 251.

⁹² Bull. Soc. franc. de dermat. et syph., 1908, 19: 292.

⁹³ Ztschr., 1909, 16: 633.

⁹⁴ Arb. a. d. k. Gsndhtsamte, 1911, 37: 203.

Immunity.—There is no natural immunity to syphilis and no true cure without specific treatment. The disease is less acute now than it was in the sixteenth century. This indicates that either the spirochete has lost some of its earlier malignancy, or the human race has become more resistant. A similar phenomenon is seen in malaria and some other infections caused by animal parasites. Our knowledge is summarized by Chesney (*Med.*, 1926, 5: 463).

The return of chancre, glands, eruption and positive Wassermann reaction, a few months after control of the disease by salvarsan in its first few weeks, does not prove reinfection.

Syphilis confers no definite immunity. Second infections may take place after cure. Chancres are not auto-inoculable, but it is now known that a second chancre can be produced if the inoculation from the first chancre is made soon after its appearance. In other words, superinfection cannot take place when the disease is once established. This indicates a kind of "immunity" while the living spirochetes are in the body. The same sort of resistance is found in leishmaniasis and other parasites belonging to the animal kingdom. Superinfections are also rarely seen in active tuberculosis and certain other diseases.

There are indications that there are various strains of the spirochete which have a specific predilection for certain tissues, such as the neuro-pathic strain, vascular strain, etc. This is also explained by difference in tissue resistance by different individuals. The Wassermann reaction is not an index of immunity. See pages 586-7 for heredity and syphilis.

Syphilis and Life Insurance.—Syphilis lowers the standard of health and paves the way for other diseases. Whatever the etiological relationship may be, it is definitely known that syphilitics are prone to die early from affections of the heart and vessels, general paresis, diseases of the central nervous system (locomotor ataxia), chronic nephritis, arteriosclerosis, aneurysm, apoplexy, etc. The actuaries of all life insurance companies know that the morbidity and mortality rates among syphilitics are very much higher than those of any other class of individuals of the community who enjoy apparent good health at the time of examination. It is estimated that the mortality ofluetics is 130 to 100 for normal individuals, and in the thirty-six- to fifty-year period the average mortality in syphilitics is doubled. It is also estimated that the length of life, based on life insurance statistics, is lessened an average of five years by the invasion of syphilis. Of every seven syphilitics one dies as the result of the infection from twenty to thirty years following the chancre.

Most insurance companies refuse to accept syphilitics at all. Some companies require extra premiums to compensate for the extra risks. A few companies will accept exceptionally favorable cases who have had a thorough course of treatment and who have shown no symptoms for from three to five years, but special policies are issued which do not keep the applicant on the companies' books after fifty-five years of age.

Marriage and Syphilis.—Fournier thought that, with few exceptions, syphilis constitutes only a temporary bar to marriage. As a result of his experience, he concluded, in 1880, that marriage should be prohibited to every man having syphilis which was transmissible, and that it ought to be permitted in men in whom the disease was in such a condition as not to be transmissible. It is impossible, however, to insure any guarantee of safety whatever except in the presence of a definitely established cure.

Finger's rules as modified by Vedder⁹⁵ are as follows:

1. A mild normal course of the disease.
2. An efficient course of treatment with both salvarsan and mercury in accordance with the best practice in the treatment of syphilis.
3. An interval of at least four full years between infection and marriage.
4. An interval of three years from the last syphilitic manifestation to marriage with careful observation to determine the existence of symptoms.
5. A negative Wassermann reaction just before marriage, best confirmed by a test at a second laboratory to insure accuracy.

Osler states that the family physician should insist upon the necessity of two full years elapsing between the date of infection and the contracting of marriage. This, it should be borne in mind, is the earliest possible limit, and marriage should be allowed only if the treatment has been thorough and if at least a year has passed without any manifestation of the disease.

It is clear that the great burden of responsibility must be borne by the family physician. It is his duty to warn the patient that marriage may not be absolutely safe, that he must watch for small erosions on the genitalia or in the mouth, that may affect his wife. The family physician should know the facts so that he can watch both wife and children, and afford prompt treatment should it become necessary.

It is not practical to prevent the marriage of many syphilitics by law, although legislation making syphilis a bar to marriage has been enacted by several states. Such laws in practice enable the state to step in after the fact, and while of interest in divorce proceedings, must be regarded as of limited public health usefulness.

The following states prohibit the marriage of venereally infected persons: Delaware, Indiana, Maine, Michigan, Nebraska, New Jersey, Oklahoma, Utah and Vermont. Those states which require a statement from one or both candidates that they are free from venereal disease at the time the license is issued are: Michigan, Pennsylvania, Virginia and Washington. Medical certificates from one or both candidates are required from the following states: Alabama, Louisiana, North Carolina, North Dakota, Oregon, Wisconsin and Wyoming.

In Michigan marriage by a person with syphilis or gonorrhea is deemed a felony; in Indiana, New Jersey, Vermont and Oklahoma, it is a misdemeanor for persons having a venereal disease to marry; in Maine it is a misdemeanor for persons suffering with syphilis to marry; in Utah the law

⁹⁵ *Syphilis and Public Health*, Lea & Febiger, Philadelphia, 1918, p. 209.

provides not only that marriage is forbidden with a person afflicted with syphilis or gonorrhea which is uncured, but that such marriages are void.⁹⁶

The family is the sanitary unit, and it is of fundamental importance to public and private health to protect the sanctity of the home. There is also truth in Hutchinson's⁹⁷ contention that "counsels of perfection are often not trustworthy in practice."

The Wassermann Reaction and Marriage.—Assent to matrimony should be withheld from individuals with a positive Wassermann test. On the other hand, Keyes⁹⁸ concludes that a negative Wassermann is not sufficient evidence of the cure or absence of syphilis. Again, a positive Wassermann, unsupported by clinical evidence, may not be sufficient evidence of the presence of syphilis, and therefore in itself may not prohibit matrimony. A fixed, positive Wassermann in the later years of the disease does not inevitably point to the prospect of grave lesions. A negative Wassermann after salvarsan, in the first year of the disease, does not mean that the patient is cured, or that lesions will not reappear, because the reaction may again become positive.

Syphilitics must remember that efficient treatment requires from one and one-half to three years, and that they should remain under prolonged medical observation to detect recurrences.

Standard of Cure.—A person is not cured until all syphilitic spirochetes disappear from the body. This disinfection may be accomplished easiest during the early stage of the chancre; hence, the importance of early diagnosis by dark field illumination, and the prompt and intensive use of salvarsan, followed by mercury and other remedies, especially bismuth. Many cases are arrested, but true cures are more rare than heretofore supposed. If a patient remains absolutely free from all symptoms for a period of two years following intensive treatment, he may then be relatively certain of a cure. The standard of cure may be set as follows: One year without treatment, without any suspicious clinical signs, with several negative Wassermann reactions and no positive ones, and with a spinal fluid examination negative for syphilis.

Calomel Ointment as a Prophylactic.—Syphilis was regarded as an infection peculiar to man until Metchnikoff and Roux in 1903 transmitted the disease to the higher apes. Later it was found that monkeys and rabbits are susceptible. As a result of these experiments, certain important facts in reference to prophylaxis were discovered. Of all the various germicidal substances tried, Metchnikoff and Roux found that mercurial inunctions are most successful in preventing the development of the chancre. Calomel ointment has proved itself best.

In a communication published in 1906, Metchnikoff and Roux⁹⁹ state

⁹⁶ "American Marriage Laws in Their Social Aspects," and "Medical Certification for Marriage," issued by the Russell Sage Foundation, New York City.

⁹⁷ *Syphilis*, Cassell, 1909, p. 553.

⁹⁸ *J. Am. M. Ass.*, 1915, 64: 804.

⁹⁹ *Bull. Acad. de méd.*, 1906, 55: 554.

that having tried twelve experiments on monkeys with uniformly satisfactory results, they next performed the experiment on a man. A student of medicine offered himself, and they assured themselves that he had never had syphilis, either acquired or hereditary. The man did not develop a chancre, although watched for more than three months. Seventeen days after the inoculation the two control monkeys that were not treated with the ointment developed primary lesions, while the monkey, treated after a twenty-hour interval, developed a chancre after thirty-nine days' incubation. It is interesting to note that the subject of this experiment was Maisonneuve,¹⁰⁰ who published this experiment on himself as part of his thesis for the doctorate.

Calomel ointment to be effective must contain 33 per cent of calomel, should be incorporated in lanolin¹⁰¹ as a base, and great care should be taken in its preparation to ensure thorough mixing. No doubt many failures in actual practice and even in some experiments may be attributed to ignorance or negligence on the part of the pharmacist in not following these directions.

With rare exceptions, those who have watched the use of the prophylactic among troops or other large bodies of men are enthusiastic in regard to the results obtained, but the figures and statements they present would hardly serve to convince the critical. In practice it cannot be expected that the use of the prophylactic will be invariably successful, but it seems reasonable to believe that if properly applied during the first hour after exposure it will prevent the great majority of syphilitic infections. The efficacy of the prophylactic diminishes rapidly as the time between its use and the exposure increases. In addition to this time factor, there will be variations in efficacy in practice, depending upon the care with which the calomel ointment is compounded and upon the intelligence and thoroughness with which the prophylactic is applied.

Colonel Edwin P. Wolfe,¹⁰² who conducted work at Fort McKinley, P. I., found that only two cases of primary syphilis occurred among 19,465 men who were given prophylactic treatment, whereas ninety-nine cases developed among those not treated in a command of 70,910. Acevodo¹⁰³ in 1908 reported 1435 prophylactic treatments in the Chilean Navy following exposures in ports all over the world: only three cases developed syphilis. See also page 73.

Excision, or destruction of the chancre with the actual cautery or with corrosive antiseptics, cannot influence the development of the disease, for the spirochetes strike deep even before the chancre appears.

Further measures for the prevention of syphilis are considered with Venereal Prophylaxis and Hygiene of Sex, pages 68 and 83.

Summary.—Syphilis affects about 8 per cent of the total population, occurs at all ages and in all classes of society, is the cause of from 10 to

¹⁰⁰ *Thèse de Paris*, 1906, Steinheil.

¹⁰¹ *Adeps lanæ hydrosus*, containing not more than 30 per cent water.

¹⁰² Vedder, *Syphilis and Public Health*, Lea & Febiger, Philadelphia, 1918, p. 190.

¹⁰³ *Mal. Cutan. et Syphilitiques*, 1908, 19: 868.

35 per cent of all insanity, and is one of the causes of mentally and physically deficient children. It is the cause of locomotor ataxia, paresis, and the chief cause of apoplectic strokes in early life, and is responsible for a large proportion of diseases of the heart and blood-vessels; is the cause of nearly half of abortions and miscarriages. Syphilis decreases the length of life about one-third; it also lowers the standard of health and paves the way for other diseases; it greatly decreases earning capacity; is the most serious cause of disruption of home and happiness, and causes untold suffering and misery. Withal, it is largely preventable and occasionally curable. The public health control of syphilis depends upon early diagnosis and facilities for prompt treatment.

GONORRHEA

Gonorrhea is much more prevalent than syphilis, and common opinion regards it as a mild and not very shameful disease, that is, "no worse than an ordinary cold." As a matter of fact, gonorrhea is one of the serious infectious diseases, for the gonococcus occupies a position of high rank among the virulent pathogenic microorganisms. From an economic and public health standpoint, gonorrhea does not fall very far short of syphilis in importance.

The gonococcus occurs characteristically in the polymorphonuclear leukocytes as small, biscuit-shaped, Gram-negative diplococci. The organism is aerobic and can be cultivated only on special media and then with difficulty. It is killed in a few minutes at 55° C. It soon dies when dried or when exposed to the air. It is very frail, outside the body, weak germicides being sufficient to kill the coccus. Cultures soon lose their virulence. Infantile types appear to be less pathogenic than adult types.

The complications of gonorrhea are: periurethral abscess, prostatitis and epididymitis in the male; vaginitis, endocervicitis and inflammation of the glands of Bartholini in the female. Perhaps the most serious of all the sequelæ of gonorrhea are those which result from the spread by direct continuity of tissues, such as inflammation of the uterus, often extending into the fallopian tubes to the ovary, and even the peritoneum. The gonococcus has been found in pure culture in cases of acute general peritonitis. Other inflammations caused by the spread of the infection are cystitis, which sometimes extends upward through the ureters to the kidneys.

The gonococcus sometimes invades the blood and produces a general septicemia; death may occur from acute endocarditis. Gonorrheal arthritis is, in many respects, the most damaging, disabling, and serious of all the complications of gonorrhea. It may even follow ophthalmia neonatorum. It is more frequent in males than in females, but a gonorrheal arthritis of great intensity may occur in a newly married woman infected by an old gleet in her husband. The serious nature of gonorrheal complications in the eye is considered separately under Ophthalmia Neonatorum. Gynecologists tell us that the greater part of their practice is made up of the consequences of gonorrhea.

Morrow¹⁰⁴ assures us that 80 per cent of deaths from infections peculiar to women are due to gonorrhea. From 75 to 80 per cent of all operations of the female genital tract are said to be due to gonorrheal infections alone.

Sterility is one of the serious consequences of gonorrhea. This may be caused in the male through epididymitis or orchitis, which is a very common complication, and in the female by salpingitis, which closes or obstructs the fallopian tubes. Gonorrhea is said to be the cause of about one-half of all cases of sterility.

Stricture of the urethra in the male is a frequent sequel.

Gonorrhea is usually transmitted by sexual congress; however, accidental or innocent infections are not infrequent, especially in young girls.

Standards of Cure.—It is difficult to determine when a case is cured. The following tentative rules are laid down by the United States Public Health Service:

Males: (1) Freedom from discharge. (2) Clear urine; no shreds. (3) The fluid expressed from the urethra by prostatic massage must be negative for gonococci on four successive examinations at intervals of one week. (4) After dilatation of the urethra by passage of a full-sized sound, the resulting inflammatory discharge must be negative for gonococci.

Females: (1) No urethral or vaginal discharge. (2) Two successive negative examinations for gonococci of secretions of the urethra, vagina, and the cervix, with an interval of 48 hours and repeated on four successive weeks.

Diagnosis.—The diagnosis of gonorrhea is usually made from stained smears of the secretions. Unless the preparation is well made, properly stained, and examined by a competent microscopist, the results may not be dependable. Positive results are indicated by the presence of Gram-negative diplococci in the leukocytes. Negative findings do not rule out the disease, especially in the female. The specificity of the complement fixation test depends upon the technic and the operator: When positive, it indicates gonorrhea. A negative test does not exclude the disease. This test has not yet been standardized.

Prevention.—Inject, as soon after exposure as practicable, 2 per cent protargol or 10 per cent argyrol (see page 73).

Epidemic Vaginitis, Vulvovaginitis in Children.—Vulvovaginitis is common in young girls and is frequently due to the gonococcus.¹⁰⁵

Outbreaks occur in schools, tenements, playgrounds, asylums, hospitals or wherever children congregate in considerable numbers and where the same lavatories, towels, nurses, etc., are provided in common. Paul Bendig¹⁰⁶ reports the following instance: Of 40 girls sent for convalescence to a brine bath,

¹⁰⁴ *Boston M. & S. J.*, 1911, 165: 520.

¹⁰⁵ Anderson, Schultz and Stein found that in a series of forty-two cases of vulvovaginitis among children reporting to a dispensary clinic for treatment, 37.5 per cent were due to the gonococcus and 64.3 per cent were non-specific. Uncleanliness and local irritation are believed to be important and probably the primary factor in the non-specific cases (*J. Infect. Dis.*, 1923, 32: 44).

¹⁰⁶ *München med. Wchnschr.*, 1909, 56: 1846.

15 showed signs of gonorrhea after the return. The infection came from an eight-year-old girl, who apparently had been suffering from gonorrhea for several years, and was spread through indiscriminate bathing in one bathtub and the use of the same bath towel.

Infants may contract the infection from the hands of the nurse. Syringes, bed pans, catheters, thermometers, towels, diapers, wash cloths, and bed linen may account for the transmission of the gonococcus in hospitals and asylums, although the rapidity with which the gonococcus dies when dried diminishes somewhat the danger from this source. Diapers should always be disinfected by boiling or steaming before they are again used, especially in institutions; this, not only on account of gonorrhea, but of infectious diarrheas. In the public bath, children who use the same towel, tub, soap, etc., run a great risk. Taussig¹⁰⁷ believes the seat of the water-closet favors the infection in little girls. These seats are usually too high and thus readily become smeared with the discharges from the vagina, and thus infect others. In schools and tenements the water-closets are often used by a stream of children one after another. Hence, such seats should be low and U-shaped.

The frequency of gonorrhea in children may be judged from the observations of Pollack, who reports 187 cases treated in the Women's Venereal Departments of Johns Hopkins Hospital during the year 1919.¹⁰⁸ Pollack estimates that 800 to 1000 children are infected each year in Baltimore, and that the same proportion probably holds good for other cities. Seippel estimates that 500 cases occur annually in Chicago. One cause of the infection among children is the horrible superstition that a person infected with gonorrhea may get rid of it by infecting another—especially a virgin. Gonorrhea in children due to rape is rare.

When gonorrhea enters a children's hospital or an infants' home it is prone to become epidemic and is very difficult to eradicate. The story of the infection in the Babies' Hospital, New York, for eleven years, as told by Holt,¹⁰⁹ illustrates the singular obstinacy of the infection. In spite of the greatest care and precaution, there were, in 1903, sixty-five cases of vaginitis with two of ophthalmia and twelve of arthritis. In 1904 there were fifty-two cases of vaginitis, only sixteen of which would have been recognized without the bacteriological examination. In all, in the eleven years, there were 273 cases of vaginitis; six with ophthalmia, and twenty-six with arthritis. Holt urges isolation and prolonged quarantine as the only measures to combat successfully the disease. It is impossible to control such epidemics without bacteriological diagnosis, aided by complement fixation tests.

Hess believes that the greatest obstacle to controlling the disease is the difficulty of recognizing latent cases. It is possible to convert the concealed carrier into an open case by means of provocative inoculations of gonococcus

¹⁰⁷ *Am. J. M. Sc.*, 1914, 148: 480.

¹⁰⁸ *Johns Hopkins Hosp. Bull.*, 1909, 20: 142.

¹⁰⁹ *N. York M. J.*, 1905, 81: 521, 589.

vaccines. The complement fixation test gives a very high percentage of positive results when gonococci are present, except in the very acute stage.

Epidemic vaginitis is intractable in children and tends to a spontaneous cure at puberty. Many cases continue for years despite careful treatment, and disappear spontaneously at puberty.

Complications and sequelæ of vulvovaginitis in children are much less common than gonococcus complications and sequelæ in the adult. Louise Pearce¹¹⁰ has shown that the infant types of gonococci differ from the adult types when tested by agglutination and complement fixation. The infantile types are also less virulent, and cause fewer complications.

The social and public health problems which this disease presents are very great, and our knowledge of it from the standpoint of diagnosis, quarantine, and treatment is not sufficient to enable us to handle this question in a satisfactory manner. A diagnosis resting solely upon the morphological and staining characteristics of the organisms found in smears is not sufficient to brand a child with the diagnosis of gonorrhea. Vulvovaginitis in children is not a venereal disease. If the term gonorrheal or gonococcus vaginitis were dropped from the literature, and the term epidemic vaginitis substituted, it would be much easier to handle these cases from a public health standpoint.

For the prevention of vulvovaginitis in children, the good advice of the American Pediatric Society should be followed:¹¹¹

(a) That cities be required to provide adequate hospital and dispensary facilities for the care and treatment of children having vaginitis.

(b) That matrons be placed in charge of the girls' toilet rooms in public schools.

(c) That toilet seats embodying the principle of the U-shape be used in all schools and that the toilets be of proper height for different ages.

(d) That city and state laboratories be empowered and equipped to make bacteriological examinations for physicians when patients cannot afford to pay a private laboratory fee.

(e) That educational literature on the subject of vaginitis be prepared and distributed to mothers through the medium of physicians, hospitals, dispensaries, health centers, municipal and visiting nurses.

(f) That asylums for children and day nurseries be licensed and that the license be not granted unless: first, the institution has adequate facilities for the recognition of gonococcus vaginitis; and second, the institution excludes children having this disease if they cannot be properly isolated.

That the American Pediatric Society address a special letter to hospitals which care for children, containing the following recommendations:

(a) That separate wards be maintained for the treatment of children with vaginitis who are also suffering from other diseases.

(b) That microscopic examinations of smears be made before admission

¹¹⁰ *J. Exper. M.*, 1915, 21: 289.

¹¹¹ *Arch. Pediat.*, 1916, 32: 361.

to the general wards of the hospital. In securing material for the smears extreme care should be taken to observe rigid aseptic precautions.

(c) That observation wards be provided.

(d) That individual syringes, bed pans, catheters, clinical thermometers, thermometer lubricant, wash basin, soap, powder, wash cloths and towels be provided.

(e) That single service diapers be used (at least for girls); or, that diapers be sterilized in an autoclave at 15 pounds pressure for five minutes.

(f) That nurses be required to make daily inspection of the vulva of each girl at the time of bathing and to report immediately the presence of the slightest suggestion of a vaginal discharge.

(g) That low toilets be provided and equipped with seats embodying the principle of the U-shape.

(h) That for routine purposes, the spray be used in place of tub baths for the bathing of young girls, and that older girls be sponged in beds.

(i) That nurses receive special instruction as to the nature of vaginitis, the ease with which it is transmitted, the methods of preventing its spread and the necessity for rigid aseptic surgical technic in its handling and treatment.

(j) That a dispensary with special facilities for the treatment of gonococcus vaginitis be provided.

(k) That nursing care and supervision be given in the home.

(l) That mothers be instructed as to the dangers of vaginitis, the manner in which it is transmitted, the best method of protecting other children, and the necessity of prolonged observation.

(m) That all cases of vaginitis under observation be voluntarily reported to the local health officer in states or cities where no legal requirements are in force.

Summary.—Gonorrhea is the most constantly prevalent of all serious infectious diseases, except measles; affects all ages and all classes of society; is responsible for from 6,000 to 10,000 cases of blindness in the United States; is the cause of 60 per cent of blindness of the newborn; is the cause of more than 10 per cent of all blindness; is the cause of from 60 to 75 per cent of surgical operations on the female generative organs; of 50 per cent of sterility; of many chronic diseases of the joints, bladder and generative organs; greatly decreases earning capacity; is the underlying cause of untold suffering and misery; and affects practically all prostitutes, public and clandestine. Notwithstanding, gonorrhea is a preventable disease.

Further measures for the prevention of gonorrhea are considered with Venereal Prophylaxis and Hygiene of Sex, pages 68 and 83; Ophthalmia Neonatorum, page 464.

CHANCROID

Chancroid is a specific, local, auto-inoculable, and contagious venereal ulcer, caused by the streptobacillus of Ducrey (1889). The ulcers are often multiple and confer no immunity. Chancroids are local ulcers and cause no sequelæ

or general systemic effects, such as follow chancres. Chancroids, or soft chancres, are peculiarly liable to mixed infections, and are apt to become phagedenic.

Many soft chancres are mixed infections with *Treponema pallidum*. Every venereal lesion should be examined by dark field illumination for spirochetes. This is of great importance from a public health standpoint as well as for individual prophylaxis.

A little soap and water at the time of exposure is almost an absolute preventive against chancroid. If the ulcer has developed it may be aborted by cauterization, provided the chancroid is not more than three days old. Even when seven days old the ulcers may often thus be cured, but when more than a week old, cauterization should not be employed, for, if it fails, it leaves the sore larger than ever. The method of cauterization advised by Keyes consists in washing the ulcers with peroxid of hydrogen, drying, applying pure carbolic acid, then pure nitric acid, washing again with peroxid of hydrogen, and dusting with calomel. Spirocheticides should not be used locally until syphilis has been definitely excluded by dark field illumination.

Chancroids are usually contracted in venery. The disease should not be regarded as a slight or negligible malady, for, on account of the mixed infections to which they are prone, serious consequences, and sometimes death, may result. The complications of the ulcers are various forms of phimosis, resulting from inflammation and swelling; destruction of the frenum; gangrene and phagedena; lymphangitis, with inguinal adenitis. The inguinal buboes are painful and frequently suppurate.

Chancroid is usually given subordinate consideration because syphilis and gonorrhea are much more prevalent and much more serious.

GRANULOMA INGUINALE

Granuloma inguinale is a granulomatous infection of the genital organs contracted usually by sexual intercourse. It is most commonly seen in parts of Africa and India. It is rare in the United States; up to 1926, sixty-six cases were recorded.

The ulcers are painless and extend peripherally, often into the inguinal region and the perineum. The lesions persist for years, are serpiginous, may involve extensive areas and show no tendency to spontaneous healing. Donovan, in 1905, described rod-like bodies in the cells, which are diagnostic and believed to be the cause. Tartar emetic by intravenous injection is specific for most cases.

VENEREAL PROPHYLAXIS

The same principles apply to the prevention of the venereal diseases as apply to the prevention of other communicable diseases. The fight against venereal diseases, however, is especially complicated and difficult because of the close association with prostitution, the problems of sex, morality, and alco-

holism—in fact, the question pervades the woof and warp of society. There are three primitive appetites of man—hunger, thirst and the sexual appetite (libido). The first two persist throughout life; the last blooms at puberty (although certain sexual manifestations are present in childhood), grows stronger during adolescence, and wanes with age. Any program for the hygiene of sex or the control of the venereal diseases must take into account the fact that we are dealing with a primal, impulsive, and natural passion which is the greatest force for social good, when used in accordance with the laws of nature, but may result in dire consequences when these laws are transgressed. The venereal diseases are among the most widespread and universal of all human ills, and enter more largely into the marring of domestic happiness than any other disease known to man. The difficulties of the situation should not deter the health officer and all those who labor for social betterment, for there is no more pressing problem in preventive medicine.

Prevalence.—The prevalence of the venereal diseases among the population at large can only be approximated. Definite figures, however, are at hand for selected groups. The reports from the armies of the various nations give figures which are not comparable.

The venereal disease incidence, expressed as annual rates per 1,000 of mean strength for United States Continental (excluding Alaska) enlisted men, by years 1906 to 1924, follows:

1906	143.62	1913	85.83	1920	66.85
1907	149.21	1914	89.84	1921	62.66
1908	155.17	1915	83.60	1922	63.07
1909	151.35	1916	91.23	1923	56.95
1910	137.98	1917	113.82	1924	57.15
1911	145.29	1918	149.60		
1912	115.74	1919	87.36		

Of the first million (994,206) physical records received by the Surgeon General, United States Army, under the Selective Service Act, a total of 28,754 men, or 2.89 per cent, had venereal disease. Of these, 23,327 had gonorrhea, 4,440 syphilis, and 987 chancroid. But of the so-called second million sent to camp, 967,486 of the 1,961,692 records received for men examined subsequent to about May 1, 1918, a total of 54,843 men had venereal disease—5.67 per cent. Of these, 43,262 had gonorrhea, 10,133 syphilis, and 1,448 chancroid. The latter figures are more accurate and indicate the minimum amount of venereal disease coming from civilian life among the Selective Service men.

During the years 1917-1918, including one and one-half years of the Selective Service Army, approximately 260,000 cases of venereal disease were reported among the troops in the United States to the Surgeon General, United States Army. About 200,000 of these were contracted in civilian life.¹¹² The

¹¹² The venereal disease rate in American troops in France was about forty-five cases per 1,000 men per annum. This rate was never before closely approached in the United States Army. The corresponding incidence rate in the United States averaged above sixty.

complete studies are published in "Defects Found in Drafted Men," War Department, Government Printing Office, 1920.

In civil life accurate figures are not obtainable. Cunningham¹¹³ says that 60 per cent of men acquire venereal infection some time. Twenty per cent of these are incurred before the twentieth year, 50 per cent before the twenty-fifth year, and 80 per cent before the thirtieth year. Gerrish¹¹⁴ estimates that 10 per cent of the population of New York has syphilis. Fischer¹¹⁵ guesses that 18 per cent represents the syphilitic cases in the United States, and further, that there are 250,000 deaths each year due to venereal infections. Biggs¹¹⁶ judges that there were about 200,000 cases of venereal diseases in the city of New York in 1912. Morrow¹¹⁷ states that 75 per cent of adult males acquire gonorrhea at some time, and that from 5 to 10 per cent acquire syphilis; these figures are based, not alone on his own observations, but on the opinion of such men as Neisser and Fournier. Zinsser¹¹⁸ estimates that 10 per cent of the men registered for draft under the Selective Service Act were actively infected. There were 24,234,021 men between eighteen and forty-five registered. It is conservatively estimated that of this number 2,600,000 were diseased, of whom 500,000 were syphilitic.

The pathologists of Melbourne, Australia,¹¹⁹ found syphilitic lesions in 30 per cent of 200 necropsies; furthermore, 5 per cent of the population within a ten-mile radius from the Melbourne post office were positive to the Wassermann test. Banks¹²⁰ states that we have nearly two and one-half million cases of venereal diseases occurring yearly in the United States—about one person in every forty. These findings are ample to indicate the extent of the scourge. See also pages 50 and 53.

Attitude.—Our attitude toward the venereal diseases is very inconsistent. There is a natural aversion toward these afflictions. The sanitarian should make no distinction between the venereal diseases and other epidemic diseases; he should regard the greatpox in the same light that he regards the smallpox. The principles for the control of syphilis and gonorrhea differ in no wise from those used to control smallpox, leprosy, tuberculosis, measles, diphtheria, etc. The health officer must not regard venereal disease as a punishment for sin—the victim or culprit needs help, even sympathy. *The immediate problem is the prevention of further spread of the infection.* A person afflicted with a venereal disease should be treated in the same humane spirit that actuates us in other diseases. Furthermore, the interests of the community require that the patient be accorded the best possible care and treatment. The usual attitude toward the venereal diseases may well startle us when we consider that

¹¹³ *Boston M. & S. J.*, 1913, 68: 77.

¹¹⁴ *Social Diseases*, New York, 1911, Vol. II, p. 1.

¹¹⁵ *Pub. Health*, 1913, 8: 51.

¹¹⁶ *N. York M. J.*, 1913, 98: 1009.

¹¹⁷ *Boston M. & S. J.*, 1911, 165: 520.

¹¹⁸ Letter, June 21, 1919, *Am. Soc. Hyg. Ass.*

¹¹⁹ *West. Canada M. J.*, 1913, 7: 164.

¹²⁰ *U. S. Pub. Health Rep.*, 1915, 30: 618.

with rare exceptions hospitals refuse to take a case of syphilis or gonorrhea during the acute stages, when these diseases are especially communicable. Morrow holds that the notoriously inadequate provision made for the reception and treatment of venereal patients is a disgrace to our civilization. Formerly lepers were segregated in vile lazarettos and cases of smallpox isolated in horrible pesthouses; now we have comfortable and congenial isolation wards or special sanatoria for these diseases. From the standpoint of prevention suitable hospital accommodations must be provided for venereal cases (see page 76).

Notification.—It is not possible to control any communicable disease, especially one that is pandemic, such as syphilis or gonorrhea, without a knowledge of the cases and deaths. It is perhaps even more important to collect morbidity and mortality statistics of the greatpox than it is of the smallpox. But the public registration of private disease at once defeats its own object. Compulsory methods are only partly successful, and little may be expected from voluntary registration. When we consider that in our country we have no means of knowing the amount and distribution of smallpox, except to a limited degree in the registration area, what can we expect from the registration of the closely guarded secrets of the underworld? The public registration of ophthalmia neonatorum is successful because this form of gonorrhea is so apparent and the consequences so immediate and serious.

For the year ending June 30, 1924, 193,844 cases of syphilis, 160,790 cases of gonorrhea and 8,424 cases of chancroid were reported to state boards of health in the United States. Notification by serial number became effective in Massachusetts in February, 1918, and is meeting with partial success which about corresponds to the experience in other states, as illustrated by the figures in the following table.

NUMBER OF CASES OF SYPHILIS AND GONORRHEA REPORTED TO THE MASSACHUSETTS STATE DEPARTMENT OF PUBLIC HEALTH BY YEARS

Year	Syphilis	Gonorrhea
1918.....	3,284	7,681
1919.....	4,127	9,435
1920.....	2,987	7,225
1921.....	2,497	5,563
1922.....	1,933	4,973
1923.....	1,891	4,885
1924.....	2,325	5,241

To be effective, compulsory notification must, in the judgment of the National Council for Combating Venereal Diseases, include as the first and most necessary measures: (1) The provision in every area of adequate facilities for prompt diagnosis and efficient treatment, free of charge. (2) The prohibition of quack treatment. (3) Granting of privilege to any communication made in good faith by a medical man in order to prevent the spread of infection. False reporting by number does not, after all, give the information necessary to deal with the conditions. The Australian law requires notification

by physicians and requires that the patient go to a qualified practitioner for treatment upon penalty of fine and imprisonment. Any system of notification without adequate facilities for treatment will fail of its purpose. The difficulties, however, need not deter us, and registration should be persistent even though the returns are incomplete. A start should be made, and, though the returns will be only partial at first, a gradual improvement may be expected. Every case known and properly cared for is a focus of infection neutralized.

Segregation.—Theoretically, every case of syphilis or gonorrhea should be isolated until the danger of infection is passed. Practically, however, segregation is impracticable except with a limited number of cases. With better and more attractive hospital facilities and free beds a certain amount of segregation may be accomplished voluntarily and humanely. An alert health officer can trace the source of infection in certain cases and induce the women responsible to take the salvarsan treatment in the case of syphilis, or to submit to hospital care in the case of gonorrhea or chancroid.

Sanitary isolation through self-restraint and care necessary to prevent the infection of innocent persons must be taught and impressed upon all infected individuals.

Medical Prophylaxis.—A reasonably efficient prophylaxis against syphilis, gonorrhea, and chancroid is now possible.

Calomel ointment (33⅓ per cent) applied within an hour of intercourse is generally effective in preventing syphilitic infection. There are several more or less efficient irrigations or ointments destructive to the gonococcus if used soon enough—the silver salts being the best (2 per cent protargol or 10 per cent argyrol). Prophylaxis is therefore possible, but it takes a great deal of care and vigilance, and the double method must be promptly and skillfully applied in order to be effective. It has been used with success in armies and navies, but in civil life, where strict routine and control of men are impossible, it is impracticable except in individual instances; even then it requires *time*, *intelligence*, and *sobriety*. For this reason, it has been found inadvisable to furnish soldiers and sailors with prophylactic packages.¹²¹ Better results are obtained by shortening leaves of absence and applying the prophylactic by competent attendants. To be effective it should be applied within six hours of beginning of exposure; the best results are obtained when used within one hour. Calomel prophylaxis is not applicable to women.

Method of Using Prophylactic.—Before intercourse use a liberal amount of vaselin or other lubricant. This aids in preventing abrasions and forms a coating through which infectious organisms penetrate with difficulty. As soon as possible after intercourse:

1. Wash the genitalia thoroughly with soap and water, using plenty of soap. Soap solution is a spirocheticide, and there is good evidence that chancroid infection may also thus be avoided.

¹²¹ Package K of the Army; and Sanitube of the Navy containing 33⅓ per cent calomel and 2 per cent tricesol.

2. When the prophylaxis is performed under medical instruction or by a man of sufficient intelligence this may be followed by a wash of 1:1,000 mercuric chlorid. The efficacy of this solution is undoubted, but it should not be used by ignorant persons, nor should bichlorid of mercury tablets be issued as a routine.

3. Dry, and apply about 1 dram of 33 per cent calomel ointment in lanolin. Anhydrous lanolin¹²² should not be used, and the ointment should be most thoroughly mixed. This should be well rubbed in for at least ten minutes, paying particular attention to the glans, corona and prepuce, but neglecting no part of the penis and the anterior portion of the scrotum. This should be rubbed in for at least ten minutes, and should not be removed but should be allowed to remain for twelve hours, meanwhile protecting the clothes by the application of an impervious paper napkin. This favors absorption and ensures prolonged action of the mercury on any organisms that may remain.

4. For the prevention of gonorrhea, a solution of argyrol 10 per cent or protargol 2 per cent should be injected into the urethra.

In the United States Navy the following method is employed: The entire penis is scrubbed with liquid soap and water for several minutes and then washed well with a solution of mercuric bichlorid, 1:2,000 in strength. If there are any abrasions present, they are sprayed with hydrogen peroxid from a hand atomizer. The man is then placed in a sitting position, well forward in a chair in front of a convenient receptacle, and given two injections of a 10 per cent solution of argyrol. He is required to retain each injection in the urethra for five minutes. After taking the injections, the entire penis is thoroughly anointed with a 33 per cent calomel ointment. He is told not to urinate for at least two hours, and to allow the ointment to remain on the penis for some hours. A temporary dressing is placed on the parts to protect his clothes.

The best figures on this subject are given by Riggs,¹²³ who records 5,103 prophylactic treatments with only eighty-one infections, as shown by the following table:

Hours Subsequent to Exposure	Number of Treatments	Number of Infections (Syphilis, Gonorrhea, Chancroid)	Per Cent of Infections
1	1,180	1	0.08
2	1,172	7	0.59
3	521	4	0.77
4	330	2	0.61
5	199	3	1.57
6	321	5	1.58
7	277	6	2.27
8	390	16	4.22
9	283	10	3.62
10	214	11	5.14
More than 10	216	16	7.40
Total	5,103	81	1.58

¹²² *Adeps lanæ hydrosus*, see page 62.

¹²³ *Soc. Hyg.*, 1917, 3: 299.

There were 1,180 treatments during the first hour which were followed by a single infection. This infection was carefully investigated and there is considerable doubt as to whether it was genuine or not. The disease was diagnosed as chancroid, and was cured in two days. These figures emphasize the importance of time—the efficiency of the prophylactic diminishes as the time increases. Riggs states that out of 3,556 prophylactic treatments there were only sixty-seven infections, and of these only eight were cases of syphilis.

Ledbetter¹²⁴ reports that at Cavite, before medical prophylaxis was instituted, the percentage of venereal diseases of all classes among the men averaged from 25 to 30 per cent annually, and at times even higher. The percentage of gonorrhea was reduced to 8 per cent annually, and this percentage included about thirty patients who did not report for treatment. Chancroid was reduced from 5 to 2 per cent, which included two patients not reporting for treatment. Syphilis has been reduced from about twenty cases annually to one case for the entire year 1910, and this patient did not report for prophylactic treatment. The results speak for themselves and show the efficiency of the prophylactic measures if properly and thoroughly carried out.

Holcomb and Cather¹²⁵ report the following as a result of treatment used by them in 3,268 persons in the United States Navy between May 1, 1910, and August 31, 1911. Treatment taken within eight hours after exposure in 1,385 cases show nineteen infections, or 1.37 per cent; in the interval of from eight to twelve hours after exposure in 741 cases, shows twenty-five infections, or 3.31 per cent; between twelve and twenty-four hours in 920 cases, shows forty-six infections, or 5 per cent. Of the fifty-six cases of gonorrhea occurring in the first twenty-four-hour interval, twenty-six were recurrent cases; the remaining thirty were primary infections.

Riggs states that the normal expectancy for venereal disease resulting from illicit sexual intercourse not followed by prophylaxis is about one in twenty or one in thirty. The expectancy for venereal disease when prophylaxis is used depends almost entirely upon the factor of time. The absence of the time factor in a set of prophylactic statistics invalidates any conclusion that may be drawn concerning probable efficiency of prophylaxis. In actual practice the number of infections appears to be reduced by nearly one-half. The questionnaire method of investigation, in which the identity of the individual is concealed, has proved unreliable and the results obtained cannot be accepted with any confidence as to their accuracy. Furthermore, many of the reported results are difficult to analyze on account of lack of accurate data, and especially lack of control figures.¹²⁶

The prophylactic use of arsphenamin by those exposed to syphilis before the chancre develops is rational and satisfactory. The early and complete treatment of those who develop the disease is one of the best means of preventing further spread.

¹²⁴ *J. Am. M. Ass.*, 1911, 56: 1098.

¹²⁵ *J. Am. M. Ass.*, 1912, 58: 368.

¹²⁶ *U. S. Nav. M. Bull.*, 1921, 15: 1.

Mechanical Methods.—The condom was introduced late in the seventeenth or early in the eighteenth century in England. It was described by Turner in 1717, who attributed its invention to a Dr. Condom, from whom the device was named. Le Pileur, who has made a study of this literature, questions the existence of Dr. Condom, and thinks it more probable that the name is derived from the Latin verb *condere*, meaning to hide or to protect. Most authorities agree that the use of a condom is an almost certain protection against venereal infection, being even more reliable than prophylactic ointments.

Madame de Staël is accused with saying that condoms are a cobweb for protection but a cuirasse against pleasure. In regard to the protection afforded, we may conclude that the condom if properly tested and made of good rubber will afford practically complete protection to anyone who can obtain and will use it, and it may therefore be recommended to those individuals who persist in immorality in spite of advice to the contrary, and notwithstanding the danger of extragenital infection.

Specific Treatment.—From the standpoint of preventing syphilis, the treatment of the infected is perhaps the most practical single method that can be applied. It is obvious that if all infected individuals are rendered incapable of transmitting their infection, the disease will disappear. Notification, early recognition, and prompt treatment, therefore, become our most important prophylactic measures against syphilis.

Salvarsan (arsphenamin and related arsenical compounds) promptly kills the spirochetes in the chancre, mucous patches and other exposed lesions, and thereby destroys the focus of infection. Salvarsan promptly used is a specific and a preventive, even though some of the parasites hidden in the cells of the inner organs escape destruction. Unfortunately, salvarsan has always been a drug of the proprietary class, made in an atmosphere of mystery, under a patent giving an imperfect account of its preparation, produced at small cost and sold at a very high price. Recent efforts in this country to make and distribute this chemical free of charge in the same way that antitoxins and vaccine virus are distributed by boards of health is a commendable public health measure.

The danger of syphilis to the community or individual is increased in proportion to the inadequacy of the treatment received by those suffering with the disease, particularly during the first stages. The possibility of controlling the amount of syphilis acquired, whether by prostitution or general immorality, by means of enforced systematic treatment, has not yet been given the serious consideration that the subject merits. Quarantine may be imposed, but compulsory treatment cannot be legally enforced. When proper facilities are afforded the poor for the treatment of this class of diseases, one of the main props of the quack will have been removed. The Massachusetts State Department of Public Health has made and distributed arsphenamin free of charge since December, 1917.

The inadequate early treatment of syphilis, especially the inefficient administration of arsphenamin, is responsible for grave relapses or incidence of late

neurosyphilis. From the standpoint of prevention, therefore, treatment should not only be early, but intensive and prolonged. Most early syphilis is under-treated.

Hospitals and Clinics.—Pontoppidan, on the basis of his large experience with the Danish system, estimates that 1 bed to 2,000 of the population is insufficient to care for sexual diseases. Stokes,¹²⁷ says that in 1914 it was estimated that the city of London, with 7,000,000 inhabitants, had only 163 beds available for the treatment of venereal disease, and the same condition obtained throughout Great Britain. Of thirty general hospitals in New York City in 1918, only ten received recognized cases of syphilis; thirteen of thirty would not receive medical cases with complications of syphilis or gonorrhea. Chicago has 125 beds set aside for skin and venereal diseases in the Cook County Hospital for 2,000,000 people. The attitude of the general hospital toward syphilis and other venereal diseases is slowly changing. Special hospitals for the treatment of venereal diseases will not serve the purpose on account of the stigma which soon attaches to these places. Every general hospital should have wards and other facilities for the treatment of syphilis and gonorrhea. Beds are necessary in order that salvarsan or similar drugs may be administered under proper safeguards. No patient would occupy a bed long, since salvarsan usually causes a prompt disappearance of external lesions. It will be generally admitted that present facilities are sadly deficient, and that the extension of those facilities should be among the first steps to be taken to control these diseases.

Clinics for outpatient treatment are of equal if not greater importance than hospital beds. An adequate clinic may expect to have at least 5,000 visits per 100,000 population. These clinics should be accessible, open at night and otherwise convenient and attractive. The service must be of good quality. Such clinics for the early treatment of venereal disease diminish the influence of quackery and are the most practical of prophylactic measures.

Prostitution.—Any sanitary measures taken for the prevention of venereal diseases which do not include some method for treating the problem of prostitution are doomed in advance to failure, since they will ignore the main source and root of these diseases. Fournier¹²⁸ states that among the class of men seeking treatment in the hospitals of Paris, 72 per cent of all syphilitic infections were derived from registered prostitutes, 25.1 per cent were derived from clandestine prostitutes and general immorality, and only 2.7 per cent of these infections from adulterous relations. The Chicago Vice Commission states that: "So long as there is lust in the hearts of men it will seek out some method of expression. Until the hearts of men are changed, we can hope for no absolute annihilation of the social evil." While waiting for the slow evolution that aims at bettering the moral fiber of mankind, the following four ways of dealing with prostitution have been attempted: (1) *laissez faire*, (2)

¹²⁷ *J. Am. M. Ass.*, 1916, 67: 1960.

¹²⁸ *Les Chancres Extragénitaux*, Paris, 1897.

suppression, (3) regulation, and (4) the systematic treatment of all infected. A policy of non-interference satisfies no one. Despite the difficulties and complexities of the situation, we must insist that prostitution be met with determined but humane action to lessen its extent and diminish its dangers. Prostitution must at least be made *difficult* and *distant*, for the extent of the patronage is in direct ratio to its accessibility. The total elimination of prostitution is beyond the dream of even the theoretical reformer. Any program must take into account the fact that a great majority of prostitutes are feeble-minded, and often need treatment and guidance rather than punishment.

Suppression does not suppress. Virtue cannot be secured by legislation. Repressive measures drive the traffic into obscurity and reduce it materially. Vice is not flaunted in public, but is driven into corners where the vicious will find it, but where it will not entice the innocent and unwary. Between the flagrant evil of segregation and the imperfections of suppression, the choice is with the latter.

Regulation of prostitution by means of medical inspection and licensure has proven a failure wherever tried. Regulation implies the absence of any expectation of male self-restraint; it is society's tacit assent to laxity. Regulation fails because it makes vice easy, gives a false sense of security, and does not reach clandestine prostitution. The systematic treatment of all infected persons, especially of prostitutes, would go far towards diminishing the prevalence of venereal disease. To accomplish this, we must have adequate and inviting facilities for treatment.

Summary of Venereal Prophylaxis.—Finally, in considering venereal prophylaxis, it should be remembered that these diseases are of great antiquity and seem likely to continue indefinitely; that they already affect a large number of the population, and are spreading; that the existing means for the treatment of them among the poor is insufficient; that the common mode of propagation is irregular and illicit intercourse; that prostitution arose in response to the strongest instincts and passions in the human breast; and that prostitutes themselves need protection and have claims on the humanity of the law. Furthermore, Lecky thinks that "The prostitute is ultimately the efficient guardian of virtue."

To diminish the amount of venereal infection requires education and publicity, notification, laboratory facilities for diagnosis, dispensary and hospital facilities, public health nurses and social service, and good laws actively administered. Medical schools should give more time to the diagnosis and handling of early syphilis, since at this time the best results in treatment and prevention can be offered. A stricter supervision of barber shops, restaurants, hotels, soda water fountains, infant asylums and schools should be maintained; prostitution should be made difficult and distant and early treatment of all cases instituted. Medical prophylaxis should be better understood and the importance of personal hygiene impressed. Insistence should be stressed upon continence and efforts made to improve the moral and physical fiber of mankind. Progress will be slow, but good results are already apparent.

THE PSYCHOANALYTIC APPROACH TO SEX HYGIENE

The New Theories of Sex.—The conceptions of sex have undergone some radical changes within the last twenty years. Up to this time it was almost universally believed that the sexual impulse of man did not begin before the age of puberty, although here and there certain pediatricians (Linder¹²⁹ and later Friedjung¹³⁰) interpreted thumb-sucking as an infantile sexual manifestation. Even infantile masturbation was overlooked or misinterpreted.

In 1905, Freud published his work¹³¹ on sexuality in human beings, tracing its development from early childhood through a period of latency, up to the time when the sexual impulse attains physiological and psychological maturity at puberty and adolescence. The Freudian doctrines provoked violent opposition, but these new theories have a bearing upon sex hygiene and provide a means of study of the etiology of certain psychoneuroses and sexual aberrations which are so widely spread among mankind. Manifold symptoms of the neuroses result from unfulfilled sexual desires, often extending back to the earliest years of childhood.

Psychoanalysis presents difficulties, partly on account of the new terms used and the novel concepts which have been developed, but principally on account of the absurd extremes to which some of its devotees have carried the Freudian theory; also because the Freudian theory inflicts a painful blow to the ego, and therefore is antagonized by our emotional resistances. Consequently, a short description of the meaning of psychoanalysis, with a definition of terms, is necessary to understand the Freudian theory of sex.

Explanation of Terms.—The *unconscious mind* denotes all mental processes of which we are not aware and which cannot be brought to consciousness by an effort of the will or an act of memory. The unconscious mind is popularly spoken of as the subconscious mind. The unconscious mind presumably does not sleep, for it manifests itself at night as dreams; in fact, the unconscious function of the mind has been revealed through the interpretation of dreams.

Dreams are therefore the principal manifestation of the unconscious mind in everyday life. They are the language of the unconscious mental life, although in most instances condensed, distorted and symbolized, and therefore need interpretation. It is believed that the unconscious constantly influences the conscious mind to a greater or less degree, and that this influence is controlled through our resistances and our rationalizations. This often produces mental conflicts that may be only temporary, annoying difficulties or actually disabling psychoneuroses. *Fantasies* are daydreams and also have a significance, which is being studied.

¹²⁹ *Jahrb. f. Kinderh.*, 14: 1879.

¹³⁰ *Ztschr. f. Kinderh.*, 1921, 31: 1.

¹³¹ *Drei Abhandlungen zur Sexualtheorie*, Leipzig and Wien, 1905 (English translation, *Three Contributions to the Theory of Sex*, New York and Washington, 1910).

Psychoanalysis, then, may be defined as the study of the content and working of the unconscious mind, especially its relation to consciousness. As the term indicates, it is an analysis of the mind. It is used for the treatment of psychoneuroses with the object of bringing the repressed material from which these psychoneuroses originate into consciousness. Since both neurotic symptoms and dreams have their origin in the unconscious mind, the psychoanalytic investigation of the latter provides a means for a better understanding of the former and their treatment. Psychoanalysis, however, is a difficult and technical procedure, applicable only under limited conditions, and should never be undertaken except by those skilled in the subject, else harm many result.

Conflicts usually have a sexual trend which may sometimes be resolved through psychoanalysis. In essence this process consists in bringing the unconscious or repressed thoughts into our consciousness, against a resistance which attempts to keep these thoughts repressed or bottled up in the unconscious mind. In this way fixed ideas, conflicts and fears may sometimes be resolved.

A *complex* is a partially or entirely repressed idea with emotions grouped around or attached to it. The Oedipus complex is an excessive attachment of the son to the mother, and is named from Sophocles' fateful tragedy. The Electra complex is an excessive attachment of the daughter to the father. These complexes are assumed to occur in the lives of all children, but if they persist unchanged to maturity they may lead to sexual aberrations and nervous illness.

Repression consists in keeping from our consciousness mental processes which are painful to it. *Resistance* is the instinctive opposition displayed towards attempts to lay bare the unconscious.

Perversions and Inversions.—Abnormal sexuality is common, and manifests itself in many different ways. Such persons are ordinarily distinguished as perverts and invert. *Perversion* is an unfit substitute for sexual gratification. *Inversion* refers principally to the sex of the love object being changed. *Homosexuality* is love for a member of the same sex. *Sadism* is sexual enjoyment at the sight of the infliction of bodily or mental pain on others. *Masochism* is the opposite of sadism: sexual enjoyment on the infliction of bodily or mental pain on one's self. *Fetichism* is sexual enjoyment or gratification with a portion of a loved object, such as a shoe, glove, or lock of hair. *Exhibitionism* is sexual excitement experienced in the act of displaying the genitals.

Sex; Libido.—Sex is exceedingly complicated and possesses both bodily and psychical manifestations, so closely interwoven, that it is more exact to speak of psychosexual trends. The word "sex" as utilized and expanded by psychoanalysis is synonymous with the English word "love" or the Greek word "eros" and therefore possesses a more general and less specific meaning than is conveyed by the usual connotation of limiting it to the reproductive faculty. It denotes the entire sphere of the primary biological pleasure-principle and

therefore according to these conceptions, sensations of a sexual nature may originate during childhood in parts of the body surfaces or orifices (skin, rectum, mouth) which ordinarily do not seem to subserve sexual functions in the ordinary sense of reproduction. These are termed *primary erogenous zones*. The sexual hunger and the mental aspect of the sexual instinct is termed *libido*; it is the force through which the sexual instinct expresses itself.

Freud maintains that sex is born with us: the child brings certain sexual manifestations into the world with him which can be detected in infancy, and from these, he states, "the normal sexuality of adults emerges by a significant development through manifold stages. It is not very difficult to observe the expressions of this childish sexual activity: it needs rather a certain art to overlook them or to fail to interpret them."

Sexual Development.—Sexual development shows three divisions which merge into each other, viz.: (1) the infantile, up to the age of five years (primary erogenous zones); (2) the latency period, from the fifth year to puberty (repression); (3) the period of puberty and adolescence (maturity).

Through an understanding of the infantile and latency period of sex as it has developed before puberty, much misery of adults can be modified and perhaps eradicated, particularly such sexual aberrations as homosexuality and other perversions. The neglect of the facts of prepubertic sexuality is partly due to conventional considerations and partly to the peculiar amnesia which veils from most people the early years of childhood.

It is assumed that the development of the sexual characters begins even during embryonic life. Maturity is consummated at the time known as puberty. "In man and other animals there is from birth to puberty a relatively long period when the development towards sexual maturity proceeds very slowly, or even for a time ceases altogether. There is indeed at the time of puberty no process which is essentially new to the organism. There is in reality only an extraordinary acceleration of processes which were going on up to this time at a slow rate" (Lipschutz).¹³²

Sublimation.—In adults the infantile sexual manifestations are repressed and subjected to the primacy of the genital zone which serves for reproduction. These energies become directed to more socially useful aims, a process termed "sublimation." By sublimation is meant the replacement or deflection of the energy of the sexual impulse to a non-sexual and socially useful goal. If sublimation fails, the adult remains fixed at the infantile sexual level and various perversions and inversions make their appearance. These repressive reactions are formed during the latency period and help make up the character of the adult individual; for instance, the sublimation of infantile exhibitionism gives rise to shame; of the sadistic and masochistic components to pity and disgust; of the homosexual, to morality. It is the Freudian viewpoint, furthermore, that most of the normal sexual activities of childhood, termed "polymorphous perverse" if seen in the adult, unsublimated and unrepressed, are considered perversions.

¹³² *The Internal Secretions of the Sex Glands*, Cambridge, 1924.

Object-Love and Subject-Love.—Subject-love, or *'narcissism'*, is love of one's self. Object-love is normal love directed toward the opposite sex.

The psychosexual life of children is different from that of adults. It is auto-erotic;¹³³ that is, it is largely independent of outside sources, so that the sexual pleasure experienced in childhood is obtained from more manifold sources of the body than the sexual zone alone, such as thumb-sucking, defecation, nursing, etc. In addition, sexual curiosity and infantile ideas of birth are significant and characteristic of this early period. At puberty, under the pressure of educational influences and social repressions and of the physiological activity of the gonads and sex-endocrine glands, the sexual aim becomes object-love instead of subject-love as heretofore, and the sexual excitement becomes more and more localized in the genital region. It is in the latency period, between the fifth year and puberty, that psychic forces develop which act as inhibitions in the adult sexual life. These forces are loathing, shame and moral and æsthetic demands; in fact, everything in the adult which is termed a moral ego ideal. If this change from the subject-love of childhood to the object-love of puberty fails of full development during the course of the latency period, the original auto-erotic interests persist and remain attached to the primary erogenous zones, with consequent failure of the goal of normal reproduction. It is then that we see those pathological variations of the sexual impulse known as perversions and inversions. According to this viewpoint such aberrations are not congenital, but acquired, arising during the course of psychosexual development. Of course another important factor in the genesis of these aberrations must be incriminated, namely, the influence of seduction on the child, but the results of seduction tend to show that the child had already possessed the factors for the aberrations, in its sexual disposition.

There are two kinds of these deviations, one in reference to the sexual object, leading to that form of inversion known as homosexuality, and a second, the perverted deviations of the sexual aim, leading to fetichism, sadism, masochism and exhibitionism. The psychoanalyst interprets the sexual aberrations of adults as persistences of the same tendencies of childhood which have been unsuccessfully repressed and which have failed of ethical and moral sublimation. There may be cited such examples as the Œdipus complex leading to homosexuality, the relation between cruelty and the sexual impulse producing masochism and sadism, the failure of social repression leading to exhibitionism and finally the persistence of childhood auto-eroticism into adult life, encouraging masturbation.

Homosexuality.—The sexual perversions and inversions, particularly homosexuality, are widely spread. In fact, the diffusion of homosexuality among the population has been set as varying from 1.9 per cent to 2.2 per cent, according to various investigators.¹³⁴ These figures refer primarily to the conscious and manifest inverts, but their number is small when compared with the latent homosexuals, the so-called Don Juan type of sexual aggressors of

¹³³ A term first proposed by Havelock Ellis.

¹³⁴ Iwan Bloch, *The Sexual Life of Our Time*, New York, 1920.

woman. The origins of homosexuality in the course of individual libido development have been indicated above. The problem, from the social and psychological viewpoints, is a very important one of contemporary civilization, as homosexual practices date from early Greek and Roman times. Homosexuality may yield to treatment, but not to punishment; psychoanalysis is indicated, as the older methods of hypnosis and suggestion produce only temporary improvement. Prevention here can do much in childhood, the solving of the Oedipus situation which leads to this particular form of inversion, by instructing parents, particularly mothers, against too strong an attachment and too much indulgence in their children. The adult homosexual retains the narcissism and auto-eroticism of his childhood, is in love with his body and therefore is sexually stimulated only by the image of his own body; that is, his own sex.

Other sexual perversions, such as exhibitionism, sadism, masochism and fetishism, are likewise persistences of childhood sexuality into adult life, and they can best be prevented by an understanding of their origins by physicians and educators alike. This understanding is based upon the new psychology of sexual life, which takes into consideration the individual during the entire course of psychosexual development.

The Freudian Theory and Sex Hygiene.—Taking these newer viewpoints of the sexual instinct into consideration, we can now pass conveniently to the light they throw upon the subject of sex hygiene. They are slowly having their influence upon this important aspect of the sexual problem, in contradistinction to the older attitude of fear and superficial explanation. "Although there is danger that a superficial and erroneous interpretation of the Freudian psychology in regard to the repression of the sex instinct may be detrimental to the successful development of the program of the control of venereal disease, a more thoroughgoing, complete and scientific interpretation, however, tends to aid such a program in that it places the emphasis upon the practical means for guiding the sex instinct into socially useful and constructive activities."¹³⁵

Sexual promiscuity and illicit sexual intercourse are claimed to be an inability to properly sublimate the sexual craving; that is, a domination by the pleasure-principle over the principle of reality. The first object of a child's love is coordinated with the Oedipus complex, which succumbs through repression during the latency period. At puberty this becomes a tender emotional tie, not on the surface sexual, but the sexual still exists under repression. For this reason puberty is so difficult a period, the newly acquired physiological and psychological maturity is charged with this repressed energy and unless it is properly directed and sublimated, the young person may succumb to a severe neurosis or even a psychosis.

The aim of the understanding of the sexuality of man is to free him from the resistances built up against an insight into his own sexuality and to view sex in its broadest way and not as something obscene and humiliating. Before

¹³⁵ All-American Congress of Venereal Diseases and Social Hygiene, held in Washington, December, 1920. See also "A Consensus of Medical Opinion upon Questions Relating to Sex Education and Venereal Disease Campaigns," *Ment. Hyg.*, 1920, 4: 769.

we can have a really scientific sexual hygiene free from overmoralizing and conventional preaching, there must be a complete understanding of the social, biological and psychological viewpoints of sex. It must be realized that sexuality is not unclean, but is a deep and primal instinct capable of being guided into useful channels.

Mental disturbances never result from sexual abstinence, yet sexual abstinence, combined with an overmoral and badly directed repression, may lead to a severe neurosis in insufficiently and improperly sublimated individuals. It must be admitted frankly that continence beyond the age of about twenty-one may not be good for the individual, but apparently is necessary for the social structure. This puts man in a dilemma from which it is difficult to extricate him, for all civilization seems to be tending towards city life and late marriages, both of which are contrary to normal sex hygiene. Therefore, the roots of sex hygiene lie deep in social structure and social customs.

Masturbation likewise never leads to insanity, although many erroneous ideas on masturbation have become firmly entrenched since the teachings of Tissot, in the latter part of the eighteenth century. Masturbation is common to young children during the first five years and to a certain extent in the latency period. After puberty, however, masturbation must be looked upon as an infantile or childhood way of obtaining sexual satisfaction through a persistence of auto-eroticism, because at puberty the sexual aim should be object-love and not subject-love. Masturbation itself does not produce neuroses, but the mental and emotional conflicts, particularly the ideas of sin and guilt connected with the act, may lead to neurotic disturbances. It should be controlled, otherwise it may have a deleterious effect on the emotional and psychic characteristics of the individual. The best method of control is sublimation in non-sexual activities. Coitus as a cure for masturbation, as so often advised, does not help, for illicit intercourse is the same as masturbation, as it is not the mere physical gratification but the complete psychosexual life, that needs gratification in coitus and this complete gratification can be obtained only when there is love and attachment.

"Throughout life sublimation acts by transforming some part, at all events, of the creative sexual energy from its elementary animal manifestations into more highly individual and social manifestations, or at all events into finer forms of sexual activity. Purity, we thus come to see, is, in one aspect, the action of sublimation, not abolishing sexual activity, but lifting it into forms of which our best judgment may approve" (Havelock Ellis).¹³⁶

SEX HYGIENE

Education.—Education in sex hygiene and the venereal peril accomplishes a certain amount of good. A knowledge of the consequences will not control passion. Efforts to instill a wholesome fear of venereal diseases are futile. Even one attack of a venereal disease does not act as a deterrent to future

¹³⁶ *Little Essays of Love and Virtue*, London, 1922.

immorality, and medical students who are presumably informed are no more moral than other members of the student body. The only education that will effect a reduction in immorality is the education that forms character, and, as Huxley says, "Molds the desire to live in accordance with the laws of nature."

However, the old-style innocence must be regarded as present-day ignorance. Every boy and girl, before reaching the age of puberty, should have a knowledge of sex, and every man and woman before the marriageable age should be informed on the subject of reproduction and the dangers of venereal diseases. Superficial information is not true education. On the other hand, it is a mistake to dwell unduly upon the subject, for in many instances the imagination and passion of youth are inflamed by simply calling attention to the subject. One of the objects of education is to avoid the dangers of sex impurities, and all agree that this may often best be accomplished by keeping the mind clean; that is, away from the subject. This may be accomplished sometimes by a process termed sublimation; that is, the replacement or deflection of the energy of the sexual impulse to a non-sexual and socially useful goal. The instruction must, therefore, be *clear, pointed, brief and direct*.

The object of education is not alone to help the individual to help himself, but to influence necessary legislation and concerted public action; also to lessen the influence of quacks. A simple knowledge of the facts is a sufficient deterrent for some; others may be influenced through fear of the consequences. Boys, as a rule, cannot be controlled through fear. The spirit of adventure is rife in healthy lads; they love to take a chance. Boys may be reached by an appeal to their better natures and by allusions to sister or mother. Normal boys are heedless of self, but are regardful of others: hence, a knowledge of the peril to future wife and offspring is the most impressive fact to keep boys straight.

Instruction in sex hygiene should emphasize the rewards of strength and virtue, rather than the penalties of weakness and vice. The only foundation for a healthy sex life is an individual and social morality, combined with a knowledge and full understanding of sexual realities. The teaching of sex from a biological standpoint alone is inadequate, for there is little basis for character forming or ethical instruction in the physiological analogies of animal and plant life. Instruction should be individual, not collective; positive rather than negative; constructive rather than destructive. The fear of disease or fear of anything else is not a sufficient motive for goodness. In fact, it is rather important not to frighten youth with their sexual difficulties, for this may lead to repression and morbid anxiety. In contrast to the usual procedures, the emphasis should be placed on the beauty of goodness rather than on the ugliness of vice.

In general, it may be said that the best plan of education in matters sexual is to answer the questions of young children upon the subject of maternity frankly and truthfully, but to offer them no information on the subject. The growing child at or shortly before the age of puberty should be offered a cer-

tain amount of information concerning unnatural habits and should study physiology, biology, especially botany, and the facts of fertilization. At about the age of sixteen or eighteen girls as well as boys should be instructed as to the venereal peril. Emphasis should be placed upon the future hazard to wife and offspring. The person to impart the information may be parent, doctor, minister, friend, or teacher—in any event, two qualifications are essential: (1) knowledge of the facts; (2) an impressive personality. As a rule the school-teacher is not naturally endowed nor is the classroom the best place to attain the reverent attitude essential to teach lessons in sex hygiene.

Unless education in sex hygiene to the young is properly given, which is most difficult, it may do more harm than good. Some of it excites morbid curiosity, and there is a peculiar twist to human nature that drives many to do anything specifically charged not to do. The general reluctance of parents and teachers to discuss sex matters with the young is ascribed by many to prudery, but extremes in either direction must be avoided. The problem of sex should be approached in the spirit of personal reserve that we associate with the better sort of home life rather than in the spirit of eager curiosity and practical experimentation that we associate with the school. The ideal method of instruction in these matters is therefore individual. See also pages 87 and 447.

Admirable pamphlets are distributed by the United States Public Health Service,¹³⁷ Washington, D. C., by the American Social Hygiene Association, 370 Seventh Avenue, New York City, and by some state and city boards of health.

Some of the facts all young men should know are: that the true purpose of the sex function is reproduction and not sensual pleasure; that the testicles have a twofold function, (*a*) reproduction and (*b*) to supply force and energy to other organs of the body; that occasional seminal emissions at night are evidences of normal physiological activity; that sexual intercourse is not essential to the preservation of virility; that chastity is compatible with health; and that the sex instinct in man may be controlled.

The primary function of the testicles is to build the boy into the man. Castration in early life, as in the case of eunuchs, results in a loss of the internal secretion ¹³⁸ of the testicles and a failure in development of the secondary sexual characters which distinguish the male. There are alterations in physical conformation and in the voice, lack of beard, development of the mammae, etc.—in other words, an approach to the feminine type. Healthy sexuality stimulates the imagination, sentiment, the æsthetic sense, and the higher creative functions. Excesses or any influence which weakens the sexual system impair the will power, influence self-respect, produce morbid daydreaming, and diminish mental force. Experience shows that arduous physical and mental labor, even after maturity is attained, is best performed when the sex organs are not exercised; sexual excess distinctly impairs

¹³⁷ Especially "High School and Sex Education," U. S. Pub. Health Service and U. S. Dept. of Education, 1922.

¹³⁸ Both the testes and ovaries produce hormones.

muscular strength and mental efficiency. It is unwise to frighten boys by exaggerating the results of self-abuse, which is rather the effect and not the cause of idiocy, insanity, degeneracy, and other defects of the central nervous organization. Self-abuse is no worse in its effects than natural coitus, except for its influence upon character. Both are alike harmful when indulged in to excess.

Results through education will be slow, for the aggressive conscience of the world in these matters has awakened too recently to have achieved as yet a great deal. Good results are already apparent upon the youth of the growing generation.

Continence.—One of the important facts to teach boys is that continence is compatible with health. Chastity is the best preventive against venereal infection. The testicles are like the tear gland and the sweat glands, in that they do not atrophy with disuse. Benjamin Franklin taught, as many another man of influence believes to-day, that the exercise of the sexual functions is necessary for health. This is a mistake and has done much harm. The physiologic normal for frequency of nocturnal emissions in sexually abstinent males is commonly given as one to four per month.

The sex impulse (libido) is universal in nature. It is the force behind the constructive and progressive processes of all life, from the color adaptations of birds and flowers to its sublimation in the highest leadership in men. Reproduction is only one of its many functions; and the man who assumes that the so-called physical desire that at times thrills him indicates a need of sexual intercourse is in danger of depleting and wasting from his life a chief source of physical and mental power.

The single standard for men and women must be insisted upon, and the parent or guardian is justified in demanding a clean bill of health of the young man who proposes marriage. The young man, in turn, is entitled to the same from his prospective father-in-law. One of the defects of our artificial civilization which leads to harm is the postponement of the marriage age.

To denounce youth as vicious when youth has merely followed the impulse of adolescence is futile, because youth will not believe this; other and juster reasons must be given, if youth is to listen and be controlled. Any young man, properly warned and properly informed, will not be merely willing but anxious to learn from his doctor before marriage if he is fit to be a husband and a father.

Carnal lust may be cooled and quelled by hard work of the body, as well as attention to personal hygiene—hence, one of the great advantages of athletic sports for growing young men.

Personal Hygiene.—Idleness, stimulating food, overeating, impure thoughts, evil associates, and alcohol excite the passions and are the bed-fellows of the venereal diseases. Purity of mind and cleanliness of body are helpful prophylactics. Emotional and mental excitement stimulate sexual craving. Physical exercise and an out-of-door life divert the mind and help

the body; it is a good safety valve for the excess eroticism of youth. The influence of athletics as a means of sublimation has perhaps been over-emphasized, if we agree that libido is a mental attitude little influenced by the body. After all, a normal mental attitude is favored by a normal, healthy outdoor life.

A full diet raises basal metabolism and stimulates sexual desire, whereas a low diet diminishes procreative interest and power.¹³⁹

The public should be taught the necessity for thorough daily cleansing of the external genitals in both sexes, even in children. The large number of secreting glands and the decomposition of their secretions are liable to induce irritation and even minute lesions which open portals to infection of all kinds.

Circumcision is recommended as an aid to genital cleanliness. It is generally believed to be a prophylactic against syphilis and chancroid, and also a deterrent to masturbation. But it does not prevent venereal infection and probably has little effect on masturbation.

In order to prevent innocent infections it is necessary to educate the public to place chief dependence upon personal prophylaxis. The hazard of kissing, the common drinking cup, unsanitary barbers, and the unhygienic practice of mouthing pipes and other things must become better known. Congenital syphilis can be controlled by prenatal treatment of the mother and early treatment of the infant. Infection in childhood occurs especially among families who live huddled together in one room.

Alcohol.—The strongest indictment against alcohol is that it excites the passions and at the same time diminishes the will power. The fact that alcohol blunts moral tone does much more harm than all the cirrhotic livers, hardened arteries, shrunken kidneys, inflamed stomachs, and other lesions believed to be caused by its excessive use. Alcohol is not a stimulant, but depresses the higher functions of the brain from the beginning. See Index for references to Alcohol.

Discussion: A Warning.—Sex education is part of character building; it is not a thing apart. Sex is inextricably woven into the physical and ethical progress of man; it is part of the soul and fiber of mankind. The problems are mental rather than physical, and moral rather than physiological. Religion is a helpful restraint, a positive force.

We must not consider sex as something mortifying and humiliating. Indeed, a judicious balance is here important not to go to either extreme, prudery or boldness. Sex is a normal aspect of healthy life. It should be investigated with the same calm and unemotional attitude as the hygiene of digestion; it should be taught with frankness, but with reverence. Sex instruction should be individual, rather than collective. Sex expression differs greatly at different ages—infancy, the childhood period of latency, puberty, middle life and the climacteric—and also differs somewhat in each sex, and

¹³⁹ Miles, *J. Nerv. & Ment. Dis.*, 1919, 49: 3.

in the various races and climates. Furthermore, individuals are differently constituted in this regard. Heredity plays a dominant rôle in controlling the sexual make-up.

Any program of sex hygiene must consider that we are dealing with a primal, impulsive and natural passion, a constructive force for good when used in accordance with the laws of nature. Self-propagation is a greater force than self-preservation. A successful sex life requires normal and temperate habits. Impulses should be curbed and inhibitions strengthened. We have the authority of Shakespeare, who causes Hamlet to say to his best friend,

"Give me that man
That is not passion's slave, and I will wear him
In my heart's core, ay, in my heart of heart,
As I do thee."

Monogamy as an institution is the best solution yet found for the healthy outworking of sex, both for the individual and for society. The influence of a wholesome family life and the pressure of society's best ideals are useful factors in guiding conduct and in offsetting the present irregularities and unhappiness manifest in plays, prints, moving pictures, comic strips and prevalent attitudes. The glorification of the family is fundamentally important for sex, as well as for other hygienic and social problems. The family is the hygienic unit as well as the social unit.

Finally, we must remember that our knowledge of sex is quite incomplete. The subject has been studied scientifically only in recent years. Our ignorance is abysmal. Therefore, we are quite justified in a conservative attitude, and the irregularities of the individual and the problems of society often need patience and sympathetic understanding, rather than punishment.

COLLATERAL READING

The American Social Hygiene Association, 370 Seventh Avenue, New York City, popular prints. Also the publications of the U. S. Public Health Service.

Social Hygiene, published by Am. Soc. Hyg. Ass., New York City.

Social Pathology, published by the Venereal Disease Division, U. S. Public Health Service.

Venereal Disease Information, published by the Venereal Disease Division, U. S. Public Health Service.

Venereal Disease Bulletin, No. 77, U. S. Public Health Service.

TETANUS

(*Lockjaw*)

Compared with the major plagues of man, lockjaw has always been a rare disease. It is on account of the characteristic and fatal spasms that it early attracted attention. The spasms are tonic in nature, with acute and painful

exacerbations. They usually begin in the muscles of the jaw and neck and in severe cases spread to the voluntary muscles of the body and the extremities.

Tetanus usually occurs sporadically; formerly epidemics in hospitals (especially in lying-in hospitals), and in wars were rather common. The conditions of trench warfare in the World War favored wound complications, and included a frightful amount of tetanus until antitoxin was used as a routine prophylactic in all wounds. Before the days of asepsis the infection was often spread through surgical instruments, fingers, bandages, etc.

Tetanus is one of a small group of infections common to man and many mammals. Horses, mice, guinea-pigs and goats are most susceptible; then dogs, rabbits and cats in the order named. Rats and birds are very resistant.

Historical Note.—Carle and Rattone,¹⁴⁰ in 1884, first clearly demonstrated the infectious nature of tetanus by inoculating rabbits subcutaneously with pus from a human case of the disease. In the same year, Nicolaier¹⁴¹ inoculated mice and rabbits subcutaneously with garden earth and saw the tetanus bacillus at the site of injection. In 1889, Kitasato¹⁴² for the first time grew the organism in pure culture, and by successful inoculation experiments proved that this bacillus was the real cause of tetanus. Kitasato further showed that the tetanus bacillus is not found in the heart's blood of mice dead of tetanus, and therefore concluded that we are dealing with an intoxication, and not a bacteremia. We now regard tetanus as a type of the true toxemias; that is, the bacilli remain localized in the wound, and the soluble toxin does the damage. This pioneer work of Kitasato's was of great importance, and led to the epoch-making discovery of Von Behring and Kitasato¹⁴³ in the following year (1890) upon tetanus and diphtheria toxins and antitoxins, laying the foundation of serum therapy.

Etiology.—Tetanus may be regarded almost solely as a wound complication. All wounds are not equally liable to this complication, even though tetanus spores be present. Punctured, lacerated, and contused wounds are much more susceptible to tetanus than clean-cut or open wounds. The size of the wound is of much less consequence than its character and content. Fatal tetanus may develop from trivial wounds, such as pin scratches, small splinters, insect bites, vaccinations, etc. Necrotic tissue, foreign bodies, pus infections and other irritants favor the development of tetanus.

Symbiosis is an important factor in tetanus. Wounds infected with bacteria, such as the gas bacillus, *vibrio septique*,¹⁴⁴ or pyogenic organisms favor anaërobic conditions, permit the tetanus spores to germinate, and seem to encourage the growth of the bacillus and the development of toxin.¹⁴⁵ Tetanus spores washed free of toxin and placed in healthy tissues may do no harm,

¹⁴⁰ *Gior. d. r. Accad. di med. di Torino*, 1884, 32: 174.

¹⁴¹ *Deutsch. med. Wchnschr.*, 1884, No. 52; *Inaug. Diss.*, Gottingen, 1885.

¹⁴² *Deutsch. med. Wchnschr.*, 1889, No. 31; *Ztschr. f. Hyg.*, 1889, 7: 225.

¹⁴³ *Deutsch. med. Wchnschr.*, 1890, 16: 1113.

¹⁴⁴ Described by Pasteur in 1877; studied by Koch and Gaffky, 1881.

¹⁴⁵ In the laboratory some of the strongest tetanus toxins have been prepared from mixed or contaminated cultures.

but add a trace of gas gangrene toxin, or a chemical irritant as saponin, or a physical irritant such as a foreign body, and tetanus develops.

Weinberg¹⁴⁶ has shown the multiplicity of anaërobic wound infections, and has also shown how they influence each other. The gas bacillus (*B. perfringens*, also known as *B. Welchii*) plays an auxiliary part in the causation of tetanus. The bacillus of malignant edema (*vibrion septique*) is also an ally of tetanus. These are the commonest but not the only infective agents that play a part in stimulating the growth of the tetanus bacillus in wounds.

Tulloch¹⁴⁷ on the basis of agglutination tests has separated tetanus cultures into four types. Fortunately, any one of the four antitoxins will neutralize any or all of the four toxins.

The presence and even the growth and multiplication of tetanus bacilli in a wound do not necessarily mean tetanus. There are several accessory factors which determine the disease. Some individuals have antitoxin naturally in their blood and are, therefore, wholly or partially protected. Some strains are much more toxic than others, and even with the virulent strains the amount of toxin produced depends upon a number of factors.

The important features of the bacillus of tetanus (*Clostridium tetani*) are that it is anaërobic and has a very resistant spore. In common with other saccharolytic anaërobic, it does not digest coagulated serum or milk, but energetically ferments whole carbohydrates with the production of large amounts of gas.

Tetanus is harmless when taken by the mouth. Susceptible animals may be given enormous doses of tetanus toxin by the mouth without producing the disease. The bacillus and its spore may be regarded as a saprophyte in the intestinal tract. There is, however, a suspicion that tetanus spores sometimes invade the organisms through small wounds in the digestive or respiratory tract. Perhaps some of the cases following surgical operations may be accounted for in this way rather than by infection of the catgut used for ligatures.

Tetanus sometimes occurs where no wound can be found. This is the so-called "idiopathic tetanus." One explanation of these cases is to be found in the fact that the spores are numerous in street dust and may enter the respiratory tract. They cannot do harm so long as the mucous membrane is healthy, but may enter through inflamed membranes or through small wounds in the nose.

Tetanus bacilli have been found in the bronchial mucus of idiopathic cases. Tetanus spores have occasionally been found in the lymph glands, liver, and other parts of the body, upsetting our previous view that they are always strictly confined to the site of the wound. The spores may remain latent or dormant in scar tissue or the sequestrum of bone and may be released and start an attack months or years afterwards, thus giving another plausible explanation of some cases of idiopathic tetanus.

¹⁴⁶ *Ann. de l'Inst. Pasteur*, 1917, 31: 442 .

¹⁴⁷ *Proc. Roy. Soc., Ser. B.*, Apr., 1918.

Trismus Neonatorum, or tetanus of the newborn, is still a common and very fatal infection, especially in the tropics. Before the days of asepsis the infection was permitted to enter through the umbilical wound. In certain of the West Indian islands more than one-half of the mortality among the Negro children has been due to this cause. In Venezuela, trismus neonatorum is one of the chief causes of deaths, and goes by the name of mocezuolo.

The following figures are taken from official reports:¹⁴⁸

Year	Population of Venezuela	Total Deaths in Venezuela	Deaths from Infantile Tetanus	Total Deaths from Tetanus, all Forms Except Puerperal Variety
1905	2,598,063	58,100	3,316*
1906	52,949	3,485
1907	52,140	3,713
1908	56,903	4,360
1909	53,364	2,782	3,942
1910	2,685,440	55,436	3,574	4,721
1911	55,428	3,474	4,505
1912	65,729	2,824	3,794
1913	52,847	2,743	3,662
1914	51,697	2,816	3,691
1915	2,818,220	63,133	2,804	3,699
Total	617,726	21,017	42,888

* Infantile tetanus not separated from total of tetanus deaths in years 1905-1908, inclusive.

The placenta is permeable to tetanus antitoxin. Ten Broeck and Bauer¹⁴⁹ found that when tetanus antitoxin is present in the mother's blood, it is also present in the cord blood of the newborn infant. This is a prophylactic factor of importance. The colostrum under similar circumstances contains this protective antibody.

Incubation.—The period of incubation in man is usually from six to fourteen days. The period is directly proportional to the amount of toxin and the severity of the disease. This can readily be demonstrated upon susceptible animals. In a study of 600 serial tests, Rosenau and Anderson found this direct relation between the period of incubation and the severity of symptoms by the subcutaneous injection of varying amounts of toxin into guinea-pigs.¹⁵⁰ Thus, guinea-pigs receiving fairly large doses showed symptoms on the third day and usually died; when the dose is smaller, the period of incubation is longer, the disease milder, and the chances of recovery greater. In man, with a short period of incubation, six days or less, the disease is almost invariably fatal. With longer periods the disease is usually milder and recovery may take place without the use of antitoxin. The incubation period is

¹⁴⁸ Furnished by Surgeon W. J. Stewart, U. S. Public Health Service.

¹⁴⁹ *Proc. Soc. Exper. Biol. & Med.*, 1923, 20: 399.

¹⁵⁰ *U. S. Hyg. Lab. Bull.*, No. 43, 26.

apt to be prolonged when tetanus develops following a prophylactic dose of antitoxin.

Occurrence.—The spores are not affected by gastric digestion, and in the intestines of certain animals find ideal anaërobic conditions, food supply and temperature for growth and development. Here they multiply and pass in the dejecta to pollute the soil. The soil, therefore, in all regions inhabited by man and domestic animals is more or less contaminated with tetanus. The bacilli, however, do not multiply in the soil. While the soil acts only as a vehicle, it is the immediate source of most cases of tetanus in man. The presence of tetanus spores in soil, street dust, fresh vegetables and on clothing and the skin may be traced to fecal contamination.

In Man.—Tetanus spores are present to a variable extent in the intestines of man; claims of their occasional occurrence have long been on record. Those who work in contact with manure are most apt to have them. From 5 to 20 per cent of hostlers and dairymen are "tetanus carriers." Tulloch's studies indicated that they are present in a considerable percentage of men who had overseas service in fields and trenches abounding with tetanus spores. Ten Broeck and Bauer¹⁵¹ demonstrated tetanus bacilli in 34.7 per cent of the stools of seventy-eight persons in Peking. These investigators also proved that tetanus bacilli grow and multiply in the intestinal tract of man, because several millions of tetanus spores were found in a single stool of a man kept on a practically sterile diet for a month. Buzello and Rahmel¹⁵² found tetanus bacilli in the stools of forty of fifty persons from farms near Greifswald; the majority of the positive cases were patients with cancer or ulcers of the digestive tract, but free from any signs of tetanus at the time.

In Animals.—The normal habitat of tetanus is in the intestinal tract of animals. The main reservoir of tetanus is probably the digestive tube of herbivora, although the spores may be found in a great variety of animals. Noble¹⁵³ gives a good review of the incidence in animals. Ninni¹⁵⁴ cultivated tetanus bacilli from various portions of the digestive tract of rabbits and guinea-pigs. Sanchez, Toledo, and Veillon¹⁵⁵ found tetanus in the feces of four out of six horses and in the feces of one of two cows. Park found tetanus bacilli in the intestines of about 15 per cent of horses and calves living in the vicinity of New York City.

In the Soil.—Tetanus spores are frequently found in manure, cultivated or garden soil. Dubovsky and Meyer¹⁵⁶ even found the spores in virgin forest soil. They may be blown or carried great distances.

Tetanus spores are not equally numerous in all localities. The infection is much more prevalent in warm than in cold countries. It is especially severe in the tropics, yet Iceland at one time suffered severely from tetanus neonatorum. In the United States, tetanus occurs especially in the Atlantic States, and in some parts of Long Island, New Jersey, and the Hudson Valley, which

¹⁵¹ *J. Exper. M.*, 1922, 36: 261.

¹⁵³ *J. Infect. Dis.*, 1915, 16: 132.

¹⁵⁵ *Semaine méd.*, 1890, 10: 45.

¹⁵² *Arch. f. klin. Chir.*, 1924, 130: 660.

¹⁵⁴ *Ann. d'ig.*, 1920, 30: 756.

¹⁵⁶ *J. Infect. Dis.*, 1922, 31: 614.

have become noticeable for the number of cases of tetanus complicating small wounds. The soil of the western states is relatively free from this anaërobe. The soil of Flanders and France has through long cultivation become saturated with the spores of tetanus and other anaërobes. One grain of it from the trenches injected into a laboratory animal invariably produced tetanus. Tetanus spores are widely disseminated in India.

Miscellaneous.—On account of the great resistance of the spores, they are blown about in dust and are spread everywhere by dirt and manure. Tetanus has been found in hay dust, on horses' hair, in the dust of houses, barracks, and hospitals, in the mortar of old masonry, in street dust, on food, in gelatin, on the skin, and in the greatest variety of places.

One of the agencies in the distribution of tetanus spores over limited areas is undoubtedly the common house fly. The poisoned arrowheads of certain savages in the New Hebrides contain tetanus spores obtained by smearing the arrowheads with dirt from crab holes in the swamps (Le Dantic).

The wounds produced by blank cartridges are especially liable to develop tetanus. The source of the tetanus spore in these cases is not entirely clear. Wells examined 200 cartridges from five firms without finding the tetanus bacillus. It is probable that the spore is upon the skin and is carried along with the paper and powder from the blank cartridge. The peculiar character of the wound favors the development of tetanus.

The great decrease in the number of cases of tetanus following Fourth of July wounds is due to the vigorous campaign carried on by the American Medical Association. In 1903 there were 406 deaths from tetanus; in 1904, ninety-one; 1905, eighty-seven; 1906, seventy-five; 1907, seventy-three; 1908, seventy-six; in 1911 only eighteen cases and ten deaths and so on until 1916, when no deaths occurred from this cause. Eighty per cent of these followed blank cartridge wounds. The good results are attributed to the more thorough and careful treatment of the wounds and especially the use of tetanus antitoxin as a prophylactic—and more recently to safer and saner methods of celebration.

Tetanus spores or toxin may contaminate bacterial vaccines, antitoxic sera, vaccine virus, and other biologic products used in human therapy. The possible association of tetanus with bacterial vaccines was demonstrated in the unfortunate outbreak at Mulkowal, India, in 1902.¹⁵⁷ One hundred and seven persons were inoculated with Haffkine's plague prophylactic. Of these nineteen were affected with symptoms of tetanus and died. In this case the tetanus probably grew as a contamination in the plague culture, for it is now well known that the anaërobic conditions produced by *B. pestis*, *B. diphtheriae*, *B. subtilis*, and other organisms in liquid culture media favor the growth of tetanus and the development of its toxin.

In St. Louis (1901) diphtheria antitoxin was taken from a horse during the period of incubation of tetanus and used in amounts from 5 to 10 c.c. upon seven children, all of whom died of tetanus. Bolton, Fisch, and Wal-

¹⁵⁷ *J. Trop. M. & Hyg.*, 1907, 10: 33.

den¹⁵⁸ found that the serum was sterile, but contained tetanus toxin in considerable amount. If the serum had first been tested upon animals, its poisonous properties would have been discovered. This test is now required by the United States law of July 1, 1902, for all serums and vaccines sold in interstate traffic. As a further precaution against this complication horses undergoing treatment for the production of immune sera are given prophylactic doses of tetanus antitoxin from time to time. Tetanus sometimes occurs as a complication of vaccination (see page 22).

It is, of course, not the rust on a nail that is dangerous, so far as tetanus is concerned, but the spore-bearing dirt it carries into the deep, contused wound that causes the trouble. Gelatin may contain tetanus spores, and the subcutaneous injection of imperfectly sterilized gelatin as a hemostatic has sometimes resulted in accidents.

Tetanus toxin is a true exotoxin because: (1) It is soluble; (2) it acts only after a period of incubation; (3) it is thermolabile; (4) it is exceedingly poisonous; (5) it reproduces the symptoms and lesions of the disease; and (6) it stimulates antitoxin production. Tetanus toxin is elaborated in the wound and travels up the nerves and is also distributed by the blood stream. It combines loosely with the gray matter of the central nervous system.

The exceedingly poisonous nature of tetanus toxin is shown by the fact that an M.L.D. of the standard toxin was .000006 gram for a 350-gram guinea-pig.¹⁵⁹ Brieger and Cohn had a purified tetanus toxin that killed mice weighing fifteen grams in doses of .00000005 gram.

The toxins of tetanus, diphtheria, and botulism are alike in most respects, except that botulinus toxin is exceedingly poisonous by the mouth, whereas tetanus and diphtheria are not. The toxin of scarlet fever is thermostable.

Resistance.—The tetanus *bacillus* is readily destroyed by the ordinary agencies that kill vegetative bacteria. It is killed almost at once in contact with the free oxygen of the air. On the other hand, few, if any, forms of life have a greater resistance than the tetanus *spore*. Hours of exposure to 60° or 70° C. do not affect them. They usually survive an exposure of one hour to 80° C., but, as a rule, are killed in streaming steam or boiling water in 60 minutes. Tetanus spores, however, vary greatly in the power to resist the boiling temperature. Kitasato¹⁶⁰ found them to resist 80° C. for one hour, but to be killed in streaming steam in five minutes. Vaillard and Vincent¹⁶¹ found that the spores heated in the presence of moisture in a closed vessel would resist destruction at 80° C. for six hours, at 90° C. for two hours, and 100° C. three to four minutes, that they were not always destroyed in five minutes, but never resisted more than eight minutes at 100° C. Levy and Bruns¹⁶² found that destruction begins at eight and one-half minutes at 100° C.; after fifteen minutes few survive, after thirty minutes none. Falcioni¹⁶³

¹⁵⁸ *St. Louis M. Rev.*, 1910, 44: 361.

¹⁶⁰ *U. S. Hyg. Lab. Bull.*, No. 43, 24.

¹⁶¹ *Ztschr. f. Hyg.*, 1888, 7: 225.

¹⁶³ *Ann. de l'Inst. Pasteur*, 1891, 5: 1.

¹⁶² *Grenzgeb. d. Med. u. Chir.*, 1902, 10: 235.

¹⁶³ *Annali d'igiene sperimentale*, 1904, U. S. 14: 319.

studied the subject in view of the dangers of the subcutaneous injection of gelatin. He impregnated gelatin with spores of tetanus bacilli grown in agar or broth for ten to twelve days, and used Koch's steam sterilizer. He found the spores to resist destruction for two and one-half, but not for three, hours in streaming steam.

The experimental results are, therefore, sufficiently varied and conflicting to suggest that races of tetanus bacilli exist, the spores of which vary widely in their resistance to moist heat at 100° C. Theobald Smith¹⁶⁴ found that under certain conditions of cultivation some tetanus spores survive a single boiling or streaming steam regularly for twenty minutes, usually for forty minutes, and occasionally for sixty minutes; in one case seventy minutes' exposure did not destroy the spores. He also showed the possibility of tetanus spores surviving in culture fluids sterilized by discontinuous boiling or steaming in routine laboratory work for fully twenty minutes on three successive days.

In general, dry spores are more resistant than moist spores; and young spores are often harder to kill than old spores.

Tetanus spores resist the action of 5 per cent carbolic acid for ten hours, but are killed in fifteen hours. A 5 per cent solution of carbolic acid, however, to which 0.5 per cent of hydrochloric acid has been added, destroys them in two hours. Bichlorid of mercury, 1:1,000 kills the spores in three hours, and in thirty minutes when 0.5 per cent of hydrochloric acid is added to the solution. According to Park, silver nitrate solution destroys the spores of average resistance in one minute in 1 per cent solution, and in about five minutes in a 1:1,000 solution. *Tetanus spores are destroyed with certainty when exposed to dry heat at or above 160° C. for one hour, or to steam at 120° C. for twenty minutes. Entire confidence may be placed upon either of these two methods, provided there is direct exposure.*

The temperature recorded on the thermometer of the sterilizer may be higher than the actual temperature within the apparatus. Good sterilizing technic is essential and a factor of safety desirable. The time necessary for penetration must be taken into account. Thus, the government regulations¹⁶⁵ require an exposure of 170° C. for two hours for dry sterilization of glassware intended to contain biologic products. These same regulations require 121° C. (fifteen pounds) for thirty minutes for steam sterilization of glassware and rubber tubing. Glassware and rubber tubing must be moistened immediately before steam sterilization and each flask or hollow apparatus should contain one-eighth of its volume of water when put in the autoclave. This is for the purpose of insuring that steam will be in contact with all surfaces. Rubber goods, or other articles injured by dry heat or steam under pressure, may be sterilized by boiling for thirty minutes in 3 to 5 per cent phenol or some similar disinfectant.

Direct sunlight does not kill the spores, but seems to diminish their viru-

¹⁶⁴ *J. Am. M. Ass.*, 1908, 50: 929.

¹⁶⁵ Regulations of the U. S. Pub. Health Service, Oct. 1, 1919.

lence. Under certain circumstances they may live a very long time; Henrijean reports that, by means of a splinter of wood which once caused tetanus, he was able after eleven years again to cause the disease by inoculating an animal with the infective material.

Natural Immunity—A certain number of persons have tetanus antitoxin in their blood sufficient to protect them against this infection. Ten Broeck and Bauer¹⁶⁶ have shown that this protection correlates with the carrier state. Appreciable amounts of tetanus antitoxin were found in the blood-serum of twenty-six persons, all of whom carried tetanus bacilli in their digestive tracts, while the serums of thirty-six persons whose stools were free of tetanus spores contained no antitoxin (with two exceptions). It may be possible to establish the carrier state by the mouth and thus immunize troops and others where tetanus is common. Natural immunity perhaps plays a hitherto unsuspected rôle in this wound infection. Antitoxin may not be the whole story, for Ten Broeck and Bauer¹⁶⁷ have shown that guinea-pigs that carry tetanus bacilli and have antitoxin in their blood may show little resistance to tetanus toxin and therefore conclude that other bodies, specific for type, occur and make for the immunity observed.

Prophylaxis.—*Local Treatment of Wounds.*—Thorough surgical treatment of the wound as soon as possible is the first important measure in the prevention of tetanus. Wounds, however insignificant, should be thoroughly cleansed. Punctured or lacerated wounds, in which there is special danger of tetanus, should be freely opened, and every particle of foreign matter carefully removed. Promptness in cleansing the wound surgically is almost as important as thoroughness. Gunshot wounds and wounds containing garden earth, street dust, or other material liable to contain tetanus spores should receive special consideration. All necrotic tissue, or tissue likely to die, must be removed. The experience of the war demonstrated that thorough excision of the wound (*débridement*) is good practice. Germicides are useless—the surgeon's knife is the best antiseptic. The division of the umbilical cord and the treatment of the navel in the newborn must be done under the strictest asepsis. Wounds in which there is suspicion of tetanus should be kept open and freely drained, and otherwise treated so as to discourage anaërobic conditions.

Tetanus spores gain entrance into wounds not only from manure, garden soil, street dust, and similar sources, but also from the hands, instruments, bandages, suture material, or other objects. It is important to remember that the tetanus spore is exceedingly resistant to heat and chemical agents, and that in surgical and obstetrical practice confidence should not be placed simply upon brief boiling to destroy the spores. Very particular care must be exercised in the disinfection of substances injected into the body, such as gelatin and other organic materials.

Specific Prophylaxis.—Tetanus antitoxin is a specific and trustworthy preventive. The great experience of the World War adds confirmation to the

¹⁶⁶ *J. Exper. M.*, 1923, 37: 479.

¹⁶⁷ *Proc. Soc. Exper. Biol. & Med.*, 1923-24, 21: 267.

protective power of this specific and sovereign serum. Its use, however, must be understood to achieve satisfactory results. The antitoxin should be administered before the advent of symptoms, for after the tetanus toxin has combined with the motor nerve cells in the central nervous system, it is displaced or neutralized with difficulty, if at all. As with other antitoxins, a small amount given early is better than great quantities later. It is, however, practical to give antitoxin to the point of saturation in the disease. This is done with the knowledge that it will fix and neutralize the free toxin and thus prevent further damage, and with the hope that it will dissociate some of the poison combined with the central nervous tissue. Large doses should be given intravenously and subcutaneously as well as into the spinal canal and into the nerves leading from the wound. Nicoll¹⁶⁸ reports favorable results even after symptoms have developed.

The prophylactic dose of tetanus antitoxin is 1500 units. The rule is to give a prophylactic dose in all cases where there is danger of this complication, especially if the wound is lacerated or punctured. If the wound contains necrotic tissue or a foreign body, the injection should be repeated every seven days until the wound is clean or as long as the hazard persists. It is important to remember that tetanus antitoxin is eliminated or destroyed in the body in the course of ten days or two weeks. This makes it necessary to repeat the injection weekly in order to continue the immunity until the danger is past. Occasionally tetanus may develop even after a prophylactic dose of tetanus antitoxin has been given. This is due to the fact that tetanus bacilli persist in the wound and the complication manifests itself after the injected toxin has been exhausted. In these cases, however, the period of incubation is apt to be longer and the symptoms milder than usual. Instances in which 1500 units of tetanus antitoxin, repeated if necessary, have failed to prevent the development of tetanus are rare.

During the latter part of the war, a triple antitoxin, made from the tetanus bacillus, the gas bacillus¹⁶⁹ (*B. perfringens*), and the bacillus of malignant edema (*vibrion septique*), was used as a routine prophylactic. The last two are feeble compared with tetanus antitoxin.

The experience of the British during the World War demonstrated the protective value of antitoxin. Of 1,242,000 wounded sent to England, 1,458 developed tetanus; a little more than one per thousand. In September, 1914 (the second month of the War), the rate was nine per thousand; in October 7, and in December, 1.4. By November, 1918, the rate was only 0.7 per thousand. This remarkable improvement was due to giving antitetanic serum immediately after the wound had been sustained. Furthermore, in the cases that did develop, the period of incubation was prolonged and the disease less severe.¹⁷⁰

¹⁶⁸ *J. Am. M. Ass.*, 1915, 64: 1982.

¹⁶⁹ *J. Exper. M.*, 1917, 26: 119.

¹⁷⁰ Sir David Bruce, *Report of War Office Committee on Tetanus*; also, *J. Hyg.*, 1920, 19: 1.

Our own experience during the World War showed only thirty-six instances of tetanus among 224,089 war wounds, a rate of 1:6,224. During our Civil War ¹⁷¹ there were 505 instances of tetanus among 246,172 wounded, a rate of 1:487.

Operations on the intestines are apt to be complicated by tetanus as a result of infection from bowel contents. In these cases the prophylactic use of antitoxin should be considered.

Gunshot wounds and wounds produced by blank cartridges should always be regarded as suspicious, and should be given careful local treatment, supplemented with a prophylactic injection of antitoxin. Tetanus was a frequent complication of trench foot, and therefore a prophylactic injection of antitoxic serum should be given and repeated at intervals of seven days until the wounds are healed.

The prevention of tetanus complication of vaccine wounds consists in: (1) The use of a reliable vaccine which has been biologically tested in accordance with the federal act. (2) Proper methods of vaccination to avoid unnecessary scabs and anaërobic wound conditions. (3) Surgical asepsis of the operation and after-treatment.

The common experience of mankind teaches that most wounds heal without tetanus, and that tetanus is, in fact, a relatively rare infection. The physician, however, is in no case justified in taking chances, and it is one of the duties of the medical profession to teach the public that it pays to thoroughly cleanse and care for wounds, however trivial, *at once*, and in accordance with modern methods.

¹⁷¹ *Bull. Internat. A. M. Museum*, 1918, 7: 365.

CHAPTER II

DISEASES SPREAD LARGELY THROUGH THE ALVINE DISCHARGES

The control of this group of infections is one of the most notable achievements in preventive medicine. The causes, modes of transmission, and prevention are well understood in theory and readily attainable in practice. Sanitation here finds its first fruitful field, hygiene its useful lessons, and immunology its special applications.

There are four important intestinal infections: typhoid fever, cholera, dysentery and hookworm disease. The amount of these diseases in any country is an index of its sanitary state. It requires sanitary watchfulness and good public health administration to maintain the security now enjoyed from the ravages of the intestinal infections.

TYPHOID FEVER

Typhoid fever is an acute, specific, generalized infection due to the *Bacillus typhosus* and characterized by a continued fever lasting about four weeks, a rose-colored eruption, and diarrhea. The disease varies greatly in severity, from "walking typhoid," so mild that it may be missed, to fatal infections. The symptoms also are quite inconstant. The lesions affect especially the lymphatic system, spleen and bone marrow. The lymph-nodes in the intestines ulcerate and may lead to intestinal hemorrhage, which is one of the complications. The bacilli are found in the blood early in the disease; by the second week a leukopenia develops. Leukocytosis in typhoid fever indicates some inflammatory complication, such as intestinal perforation. One attack gives an immunity.

Typhoid fever has a world-wide distribution. It is endemic everywhere; sporadic cases occur. Epidemics varying from small outbreaks to great calamities are frequent.

The period of incubation is variable, usually ranging from seven to twenty-three days, commonly ten to fourteen days; the extremes are three and forty days. It differs in different epidemics, for the period of incubation depends upon the number and probably the virulence of the organisms ingested. Thus Miner¹ found the average periods of incubation in three epidemics due to infected water to be thirteen, nineteen and nineteen days; whereas, the averages of two milk-borne epidemics, in which the dose was probably more massive, were seven and nine days.

It may truly be regarded as pandemic. Normally, typhoid fever is a warm

¹J. Infect. Dis., 1922, 31: 296.

weather disease. It recurs as an annual crop from July to October.² Epidemics caused by infected water occur especially in cold weather. Milk outbreaks may occur at any time of the year. Autumnal typhoid is due partly to infection contracted at "health" resorts, and has, therefore, been called a vacation disease.

Typhoid fever is now most prevalent in the small towns. In the United States there is more typhoid fever in the southern states than in the northern zone. The only explanation to account for this is the influence of temperature, rural conditions, and association with the Negro. Typhoid fever is no respecter of rich or poor; it attacks those in robust health, all ages, both sexes, and usually during the period of greatest economic value to the community. Typhoid in children is apt to be abortive and atypical. Our Washington studies showed that the disease has its greatest prevalence among children, but its highest fatality in early adolescence.

The case fatality rate averages about 10 per cent, being more constant than most epidemic diseases. It has been found to vary from 5 to 12 per cent in private practice, and from 7 to 20 per cent in hospital cases.

From the standpoint of preventive medicine, it is proper to regard an outbreak of typhoid fever as a reproach to the sanitation and civilization of the community in which it occurs. Now that the matter is better understood, health authorities are held responsible for this and other preventable infections, as in the case of preventable accidents. Examples of court decisions declaring typhoid infection to be an "accident" will be found on pages 1066 and 1228.

Much harm has been done by insisting that typhoid fever is infectious, but not contagious; it is both—that is, communicable.³ Every case of typhoid fever means a short circuit between the alvine discharges of one person and the mouth of another. The physician has a dual duty in the care of a case of typhoid fever: one is to assist the patient, the other is to protect the community. The health authorities are charged with finding the origin in order to prevent further spread from that source. Again, typhoid fever is an infection against which the individual alone cannot protect himself wholly without the aid of the community.

The situation in our large cities is graphically shown in the figures given in the table below. In 1900, typhoid fever was fourth of the communicable diseases on the mortality list; whereas now, it has dropped below twelfth place. In fact there are fewer deaths in the United States from typhoid fever than from automobile accidents.

The great improvement is evident from the tables and curve on the following pages. The causes of this improvement are the general chlorination of water supplies, better milk supplies and more general use of pasteurization, typhoid preventive inoculations, the diffusion of information among the public concerning personal prophylaxis, the elimination of the great typhoid epidemic

² In the southern hemisphere the typhoid season is during our winter.

³ For distinction between these terms see page 480.

foci, and better public health administration, with the raising of the standard of general hygienic and sanitary measures.

DEATH RATES FROM TYPHOID FEVER PER 100,000 POPULATION IN THE REGISTRATION STATES OF 1900, FOR THE YEARS 1900-1923

Year	Rate	Year	Rate
1900	31.3	1912	15.3
1901	27.5	1913	13.2
1902	26.3	1914	10.8
1903	24.6	1915	9.2
1904	23.9	1916	8.8
1905	22.4	1917	8.1
1906	22.0	1918	7.0
1907	20.5	1919	4.8
1908	19.6	1920	5.0
1909	17.2	1921	5.3
1910	18.0	1922	3.9
1911	15.3	1923	3.6

The control of typhoid fever in the United States during the past two decades has been one of the great triumphs of preventive medicine.

Historical Landmarks.—Typhoid fever was confused for centuries with other continued fevers, such as recurrent fever, septic infections, and typhus fever. The first full description of what was probably typhoid fever was written by Thomas Willis, an English physician, who, in 1643, described an epidemic that occurred in Parliamentary troops. Bretonneau in 1826 further described the clinical characteristics and called it “dothienenteritis,” or abscess of the small intestine, a name it frequently bears in French literature. Louis, the distinguished French clinician, in 1829 gave the name typhoid fever to the malady to distinguish it from typhus fever. William Gerhard, of Philadelphia, a pupil of Louis, showed the difference in the lesions between these two fevers, which established typhoid fever as a distinct disease.

William Budd in 1856 pointed out that the disease is transmitted by the patient's excreta. He stated that: “The living human body, therefore, is the soil in which this specific poison breeds and multiplies; and that most specific of processes which constitutes the fever itself is the process by which the multiplication is effected.” The first water-borne outbreak carefully studied and described was at Lausen, Switzerland, in 1872; the first water-borne outbreak to attract attention in the United States occurred in Plymouth, Pennsylvania, in 1885. In 1875, Murchison traced an epidemic to a contaminated milk supply. Eberth in 1880 saw the *Bacillus typhosus* in the tissues, and four years later Gaffky grew it in pure culture. Metchnikoff and Besredka, in 1900, finally established the etiological relation by producing the disease in anthropoid apes with pure cultures. In 1894, Pfeiffer and Kolle first gave small subcutaneous inoculations of dead typhoid bacilli. About the same time, and independently, A. E. Wright began similar inoculations in British soldiers, but it took something over ten years to establish the prophylactic value of typhoid vaccines.

Prevalence.—Typhoid fever is more or less endemic in all countries. The amount of the disease, however, varies greatly. It is clearly a disease of defective civilization, for communities that pay least attention to sanitation as a rule suffer most. The great improvement which has taken place in the United States is clearly shown by the tables and curve.

The gratifying story of these figures is graphically shown in the curve on page 103.

The greatest improvement has occurred in our large cities, where typhoid fever is becoming a vanishing disease. This is well shown in the following table:

GREAT REDUCTION IN TYPHOID DEATH-RATE IN AMERICAN CITIES (1910-1924)

Year	Total Population (57 Cities) * Estimated by the United States Census Bureau Methods	Typhoid Deaths	Typhoid Death-Rate per 100,000
1910	20,996,035	4,114	19.59
1911	21,545,014	3,391	15.74
1912	22,093,993	2,775	12.56
1913	22,642,972	2,892	12.77
1914	23,191,951	2,408	10.38
1915	23,740,930	2,068	8.71
1916	24,205,359	1,842	7.61
1917	24,740,068	1,647	6.65
1918	24,971,278	1,557	6.23
1919	25,526,186	987	3.87
1920	26,154,013	921	3.52
1921	26,561,469	978	3.68
1922	26,936,843	851	3.15
1923	27,365,408	851	3.11
1924	27,868,865	856	3.07

* Twelve cities are omitted from this summary because data for the full period are not available.

This great sanitary reform can be best realized when we recall that in 1900 the toll from typhoid in the United States numbered about 35,000 deaths, which meant about 350,000 cases. In other words, one person out of about three hundred in the United States contracted typhoid fever that year. In recent years there has been scarcely enough typhoid fever in our large metropolitan centers to furnish clinical material to teach medical students.

Residual or "Normal" Typhoid.—When a city, such as Albany, Chicago, Lawrence, Lowell, or Pittsburgh, which had been using grossly polluted water, is furnished with a water supply of good sanitary quality, there at once results a marked reduction in the amount of typhoid fever. The curve is not only lowered, but it is also changed in character. The remaining typhoid after the water-borne infection has been removed is known as residual typhoid, and the curve in such cases is spoken of as the "normal" typhoid curve. The normal curve shows a distinct summer prevalence recurring with marked uniformity each year, and lacks the great irregularities which characterize the curve of a community drinking badly infected water. Residual typhoid is endemic typhoid; Sedgwick proposed the name "prosodemic"

(*proso*, through, and *demos*, the people) as more expressive of this type of the disease. The amount of residual typhoid varies markedly in different localities; thus, it is twice as high in the southern as in the northern part of

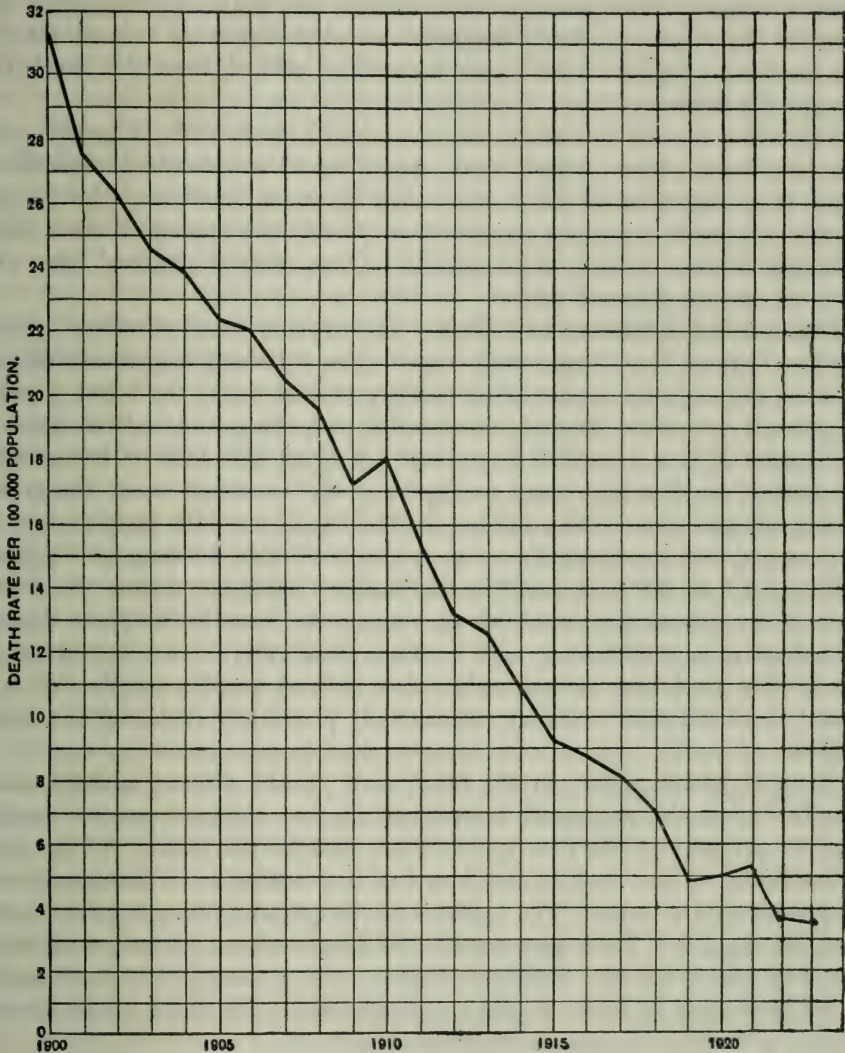


FIG. 7.—DEATH RATE FROM TYPHOID FEVER PER 100,000 POPULATION IN THE UNITED STATES REGISTRATION STATES OF 1900

our country. It is due to carriers, milk, other food, contacts, flies, and imported infection.

The chief factors in its control are pasteurization of milk, control of imported infection and municipal house cleaning. Cities with good health administration and admirable sanitation may have high residual rates, owing

to imported infection from other communities. Thus, when typhoid was epidemic in Pittsburgh, Chicago, and Niagara Falls, it kept the rates high all over the country.

Diagnosis.—An early diagnosis of typhoid fever is essential for the successful treatment of the patient, and is of vital importance in controlling the spread of the infection. Early diagnosis can only be assured through laboratory methods. Typhoid bacilli may be readily isolated from the blood, the feces, or the urine.

Physicians should encourage boards of health to furnish diagnostic aids of a laboratory nature. Such work should be in the hands of specialists rather than entrusted to those who make occasional analyses. Early and accurate diagnosis is just as important to prevent the spread of other communicable diseases as it is with typhoid. These facts emphasized here will not be repeated under each disease.

Channels of Entrance and Exit.—The typhoid bacillus enters by the mouth. Typhoid fever is generally regarded as primarily a gastro-intestinal infection, although the disease itself is not produced unless the blood, glands, and other structures of the body are invaded with the specific microorganism. The disease is now regarded as primarily a blood infection or bacteremia. The typhoid bacillus grows and multiplies in the intestinal tract, penetrates the mucosa, and thus invades the body. The bacilli leave the body mainly in the feces and urine, occasionally in the sputum and other discharges. Typhoid bacilli appear in the feces early in the disease; sometimes before the fever. Later in the disease they diminish in number and usually disappear during convalescence, although they may continue indefinitely (see *Bacillus Carriers*). The feces may contain only a few typhoid bacilli; usually they are present in considerable numbers; occasionally practically replacing the colon bacillus.

Typhoid bacilli appear in the blood early in the disease, perhaps occasionally during the prodromal symptoms. Kayser obtained positive results from 90 per cent in the first week, 65 per cent in the second, 42 per cent in the third, 35 per cent in the fourth. Our results in Washington were approximately the same. The typhoid bacilli probably do not grow in the blood during life. Their presence in the blood stream represents an overflow from the spleen and lymphatic tissues. The presence of typhoid bacilli in the blood may be taken to mean typhoid fever. The same cannot always be said if they are found in the feces or urine.

Typhoid bacilli commonly appear in the urine about the second, third, or fourth week. They grow well in this fluid both within and without the body, and may be present in such enormous numbers that the urine resembles a twenty-four-hour-old bouillon culture. From the standpoint of prevention, it is very important not to neglect the virus in the urine. Hexamethylenamin in 10-grain doses or more, three times a day, diminishes the frequency of typhoid bacilluria. This drug is eliminated as formaldehyd, provided the urine is acid, and is therefore not effective in an alkaline urine.

The sputum ordinarily does not contain the bacilli unless there is a pneumonia or severe bronchitis. Gould and Quales, and also Purjesz and Perl,⁴ have found typhoid bacilli in about 50 per cent of the cases by rubbing the gums, tonsils, and tongue of patients suffering with typhoid fever. The micro-organisms from the mouth were found as late as the fourth to eighth week of convalescence. These findings are important from the standpoint of diagnosis and prevention. The bacilli may be eliminated with the discharges from suppurating middle ears; from abscesses, such as periostitis, months and even years after the disease.

Bacillus Carriers.—About 33 per cent of cases continue to discharge typhoid bacilli for three weeks after the onset of the disease, and about 11 per cent for eight to ten weeks; these are known as *convalescent* carriers. From 2 to 4 per cent of all cases continue to discharge typhoid bacilli indefinitely; these are *chronic* or permanent carriers. Typhoid bacilli are found occasionally in the stools of some persons without a clinical history of having had the disease; these are *passive* carriers, also called healthy or normal carriers, and are very rare. In our Washington studies⁵ three positive results were obtained from the excreta of 1,040 healthy persons. At the Strassburg Station (1903-1905) Klinger reported 11 or 0.64 per cent of 1,700 healthy individuals to be temporary passive carriers, and twelve to be permanent carriers. In my laboratory at Harvard, specimens from over 4,000 healthy persons were examined in 1917, with only one typhoid carrier—a man who had the disease several years before. Leach, Dehler and Havens⁶ found 10.3 per cent of 156 individuals who had recovered from typhoid fever within six months to two years to be typhoid carriers. The per cent of carriers naturally varies with time, place, personnel, and especially with prevalence of typhoid fever.

Women outnumber men as carriers about four to one. About 80 per cent of all chronic carriers and about 60 per cent of temporary carriers are females. Women are more subject to inflammation of the gall-bladder and to gall-stones, and it is now well understood that typhoid bacilli localize and maintain themselves in the gall-bladder and bile-ducts, which are the chief sources of the typhoid bacilli found in fecal carriers. Children are less subject to gall-bladder disease, and therefore seldom become carriers. The significance of these facts is of importance in looking for carriers, special attention being given to women who have or have had symptoms, however slight, in connection with the gall-bladder or liver. The remarkable tendency of women to become carriers is particularly hazardous when we bear in mind their intimate association with the handling and preparation of food.

Typhoid carriers are either *fecal* or *urinary*, or both; the former is more frequent than the latter, and apparently also more dangerous; that is, most

⁴ *Wien. klin. Wchnschr.*, 1912, 25: 1494.

⁵ *U. S. Hyg. Lab. Bull.*, No. 52, pp. 113-150.

⁶ *Am. J. Pub. Health*, 1926, 16: 355.

outbreaks of typhoid fever traced to carriers turn out to be individuals who discharge typhoid bacilli in the feces rather than in the urine.

It seems that some carriers are more dangerous than others. This is due partly to personal habits, partly to opportunity to infect food and drink, and partly to the virulence and number of the organisms. Further, carriers are quite irregular in the elimination of typhoid bacilli.

Typhoid carriers in dairies have been responsible for many outbreaks. A carrier employed as a cook, waiter, nurse, or in a dairy is a special menace. Carriers in dairies have been responsible for many milk-borne outbreaks. A very large but variable percentage (from 5 to 40 per cent) of cases of typhoid fever have been traced to carriers. Carriers have a greater opportunity to spread the infection than bed-ridden cases. The amount of harm which a single individual can cause is amazing.

The story of "Typhoid Mary" was the first of its kind to be reported⁷ in America, and has become a classic. Mary Mallon was a cook in a family for three years, and in 1901 she developed typhoid fever. About the same time a visitor to the family had the disease. One month later the laundress in this family was taken ill.

In 1902, Mary obtained a new place, and two weeks after her arrival the laundress was taken ill with typhoid fever. In a week, a second case developed, and soon seven members of the household were sick.

In 1904, the cook went to a home on Long Island. There were four in the family, besides seven servants. Within three weeks after her arrival, four servants were attacked.

In 1906, Mary went to another family, and six of the eleven members of this family were attacked with typhoid between August 27th and September 3rd. At this time, the cook was first suspected. She entered another family on September 21st, and on October 5th the laundress developed typhoid fever.

In 1907, she entered a home in New York City and two months after her arrival two cases developed, one of which proved fatal. During these five years, "Typhoid Mary" is known to have been the cause of twenty-six cases of typhoid fever.

She was virtually imprisoned by the New York Department of Health in a hospital from March 19, 1907. Cultures taken every few days showed bacilli on and off for three years. Sometimes the stools contained enormous numbers of typhoid bacilli, and again for days none could be found.

"Typhoid Mary" then escaped from observation until 1914. In October of that year, she was engaged as cook in the Sloane Hospital for Women in New York. In January and February of 1915, an outbreak of typhoid occurred, principally among the doctors, nurses and help of the institution, involving twenty-five cases. The cook was suspected, but she left the premises on a few hours' leave, and did not return or leave her address. She

⁷*J. Am. M. Ass.*, 1907, 48: 2019; *Mil. Surgeon*, July, 1919, a review of the facts by George A. Soper.

was, however, located by the Health Department under an assumed name, and an investigation established her identity as the famous "Typhoid Mary."

A subsequent study of her career showed that she had infected still other individuals beyond those already mentioned, and that she may have given rise to the well-known water-borne outbreak of typhoid in Ithaca, New York, in 1903, involving over 1,300 cases (see page 1066). The fact is that a person by the name of Mary Mallon had been employed as a cook in the vicinity of the place where the first case appeared, and from which contamination of the water supply occurred.

Sawyer⁸ reports a very instructive history of a typhoid carrier (H. O.) responsible for several outbreaks. The carrier was carefully studied over a period of several years, during which time he infected thirty persons, five of whom died. Frequent examinations of feces of this carrier gave negative results for four months after he had been treated with autogenous typhoid vaccines; nevertheless, he infected three persons when subsequently released from quarantine on parole. The removal of the gall-bladder failed to cure H. O., for typhoid bacilli were found in the feces several times after the operation. It is particularly noteworthy that forty-one successive examinations of feces during a period of fourteen months all proved negative, yet the typhoid bacillus was finally isolated from the stomach contents containing bile. This carrier, on account of the virulence of the organism, or careless personal habits, is unusually dangerous and represents a class that should be controlled by quarantine or close supervision. He further illustrates the saying "once a carrier, always a carrier," which seems to be true of chronic typhoid carriers.

Another instructive outbreak occurred in Hanford, California, in which ninety-three cases of typhoid fever resulted from a large pan of spaghetti prepared by a carrier and served at a public dinner.⁹ This dish was baked after it had been infected, but this baking was shown by laboratory experiments to have incubated the bacteria instead of disinfecting the food.

The *Widal reaction* is present in the blood of about 60 to 75 per cent of typhoid bacillus carriers. It is, therefore, of value as a preliminary test in the epidemiological search for carriers. In blood testing for this purpose dilutions of 1:50 and 1:25, and even a titer of 1:10, may be used. The test should be made with both *B. typhosus* and *B. paratyphosus* α and β . The bacilli should then be searched for in the urine and feces of those giving a positive reaction. It should be remembered that about 90 per cent of persons immunized with typhoid vaccine will give a positive agglutination reaction, and that on the other hand it may be very weak in a carrier.

The question of preventing the spread of the disease through bacillus carriers is important and difficult. Surgical methods fail to cure carriers, for the typhoid bacillus may continue to grow in the bile-ducts after removal of the gall-bladder, and perhaps also in the small intestine. Hage and

⁸ *J. Am. M. Ass.*, 1915, 64: 2051.

⁹ *J. Am. M. Ass.*, 1914, 63: 1537.

Brinkmann¹⁰ collected forty-eight cases in which the carrierhood was cured by operative measures, and fourteen cases in which they failed; to this they add two more successful cases. Nichols, Simmons and Stimmel¹¹ believe that the so-called urinary typhoid carriers are really kidney carriers, and can be cured by nephrectomy; that intestinal carriers are really bile passage carriers of two kinds: (a) cases in which the gall-bladder alone is infected and which can be cured by cholecystectomy, and (b) cases in which gall-bladder and bile passages are both infected, and which cannot be cured by surgical measures.

Medical measures, such as hexamethylenamin, are efficient for bacilluria, but are of no avail in the fecal carriers. Attempts have been made to relieve the condition by the use of bacterial vaccines. Petruschky¹² and also Meader have reported encouraging results, especially with the use of autogenous cultures. Hektoen suggests the use of kaolin, which acts by adsorption. Most cases resist all attempts to relieve the condition.

It is unnecessary to place bacillus carriers *incommunicado*. It is sufficient to restrict their activities so that they cannot infect food or their surroundings. With proper care and cleanliness typhoid carriers may present little danger to their fellow men. This, however, requires intelligence and conscientiousness. The problem, at present, is to detect the carriers, so as to establish a sanitary isolation, if not an actual quarantine. The quarantine of typhoid carriers has been upheld in the courts.¹³

A chronic carrier should never be allowed to handle or prepare food, even though a number of consecutive examinations prove negative. The intermittent and irregular character of the carrying state should be borne in mind. Gregg reports a carrier who had typhoid fever fifty-two years before, and Bolduan and Noble one of forty-six years standing, who then caused a large milk-borne outbreak. Compulsory control of irresponsible carriers is essential. The prevention and cure of the carrying state is one of the practical and rewardful problems for research.

Resistance of the Bacillus.—The typhoid bacillus has no spore. It is, therefore, comparatively easy to destroy. The only difficulty presenting itself is getting at the bacillus when imbedded in fecal masses. When dry, most typhoid bacilli die in a few hours; occasionally a few survive for months. The fact that typhoid bacilli are killed by drying renders infection through dust unlikely.

When a moist medium, such as water, milk, or urine, is heated to 60° C., practically all the typhoid bacilli such a medium may contain are killed. An exposure at 60° C for twenty minutes will surely kill all of these microorganisms. They are not destroyed by freezing (see Relation to Ice, pages 111 and 1076).

¹⁰ *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1923, 37: 25.

¹¹ *J. Am. M. Ass.*, 1919, 73: 680.

¹² *Deutsch. med. Wchnschr.*, 1912, 38: 28.

¹³ Supreme Court of Illinois: *People ex rel. Barmore v. Robertson et al.*, 134, N. E. 815.

In their resistance to germicides typhoid bacilli behave like the average non-spore-bearing bacilli. Thus, bichlorid of mercury, 1:1,000; phenol 2½ per cent; formaldehyd solution, 10 per cent, are effective upon the naked germs. In order to kill the typhoid bacilli in feces special precautions or stronger solutions are necessary (see page 122).

The viability of typhoid bacilli in feces is very variable, depending on the composition of the feces and the varieties of other bacteria present. Sometimes the typhoid bacilli in feces perish in a few hours, often in a day; under certain circumstances they may live for much longer periods. In the Plymouth epidemic typhoid bacilli probably remained alive and virulent in the feces, exposed to the winter's cold, for several months. Levy and Kayser found that they remained alive in feces for five months in the winter. The life of the organism in privies and in water is usually comparatively short. In nature they seldom, if ever, live in water beyond seven days, and are often dead in forty-eight hours. They probably live longer in clean water than in contaminated water. In the outer world, antibiosis plays an important part, also the presence of deleterious chemicals, temperature, light, desiccation, sunlight, and other factors known to be injurious to spore-free bacteria. As a rule, the typhoid bacillus does not survive long in the soil under the usual conditions.

The typhoid bacillus may live twelve days in crude sewage (Firth); fourteen days in a septic tank (Pickard); four months in butter (Balley and Field); five days in home-made cheese (Heim); twelve days in pot cheese (Lemke); thirty-nine days in ice cream (Mitchell). It is destroyed in twenty-four hours in milk, buttermilk, whey or butter having an acidity of 0.3 to 0.4 per cent. Krumwiede and Noble found that with a moderate contamination, typhoid bacilli are killed in sour cream in about four days.

The typhoid bacillus should be regarded as a pathogen, not as a saprophyte. It lives and grows principally in the human body. It has a tendency to die in water, air, soil, upon fomites, or in nature generally. The grand exception to this statement is in the case of milk, in which the typhoid bacillus grows well.

MODES OF SPREAD

Typhoid fever is spread from cases and carriers both by direct or indirect contact—indirect through water, milk, milk products, oysters, and other foods; also flies, fingers, and fomites. Each of these modes of spread needs separate consideration.

Man is the source of the infection, and the disease must be fought in the light of an infection spread directly and indirectly from man to man.

Water.—Water-borne typhoid is a common occurrence. Not long ago it was regarded as the sole or usual mode of spread; now we know that this was a mistake. Most fecal matter ultimately finds its way to water; most water courses draining inhabited regions are contaminated with human feces. Surface water is, therefore, apt to contain typhoid bacilli. The fact that there may be no clinical case of typhoid fever in the drainage area is no guarantee

that the water may not be infected—in view of the prevalence of missed cases and bacillus carriers.

Fortunately, typhoid bacilli do not grow and multiply in water under natural conditions. They usually die in a few days, and rarely persist longer than seven days. They succumb more quickly in some waters than others, more quickly in summer than winter. Ruediger¹⁴ has shown that typhoid bacilli disappear much more rapidly from polluted water during the summer months than during the winter months when the river is protected with a covering of ice and snow.

Water plays a large but diminishing rôle in the spread of typhoid fever, on account of filtration, chlorination and sanitary control of water supplies. The great water-borne epidemics have overshadowed the other modes of communication. We know that the larger part of the typhoid now prevalent in this country is not water-borne; Whipple in 1908 estimated it at 35 per cent; it is now very much less. Typhoid fever may be excessively prevalent, even epidemic, in a city having a water supply of good sanitary quality.

In the vast majority of cases, water-borne typhoid is contracted from a surface supply; that is, a river, small stream, pond, or lake. Ground water becomes a source of danger only under special conditions, especially in limestone regions (see chapter on Water).

Water-borne epidemics present certain definite characteristics. They almost always occur in the spring, fall, or winter, when the water is cold. Most of the great water-borne epidemics have occurred in northern cities, both in this country and in Europe. They usually have a sharp onset, the curve rises to a peak, and declines rapidly. The pollution is usually nearby; that is, there is a *rather direct transfer of fresh virulent infection*. Granting that the typhoid bacillus does not grow in cold water, there must have been a very considerable dilution in most of the epidemics.

The following examples are given of the fact that water-borne outbreaks of typhoid fever occur during the winter, fall, or early spring, when the water is cold. Thus we have the water-borne epidemic in Plymouth, Pennsylvania, in 1885, which began with the spring thaw and doubtless came from the frozen accumulation of typhoid excrement from a single case. Very similar to the Plymouth outbreak was that at New Haven, Connecticut, in 1901. The outbreak at Ithaca, N. Y., started in epidemic proportions in January. The epidemic in Sherbourne, England, in 1873, likewise started in January. Four acute epidemic exacerbations are recorded in Philadelphia in December of the years 1884, 1890, 1899, and 1903. Several similar epidemics have occurred in the winter time in Chicago—one in January, 1890, another in January, 1896, and one in March, 1891. Another striking instance is the epidemic in Newark, New Jersey, in February, 1899, and one in December, 1891. Abroad, epidemics are recorded in Berlin in February, 1899, in Paris in February, 1894, and in Vienna in November, 1888. In fact, extensive water-borne epidemics of typhoid fever rarely occur in the summer time.

¹⁴J. Am. Pub. Health Ass., 1911, 1: 411.

It was formerly thought that a high typhoid rate necessarily meant badly infected water. We know now that this does not necessarily follow, as has been proved by the experiences in Washington, Winnipeg, army camps, and many southern cities.

Almost all the water-borne epidemics of typhoid fever rest upon circumstantial evidence. It is difficult to isolate the typhoid bacillus from water, and the damage is usually done before suspicion points to the water.

No single measure in reducing typhoid fever on a large scale approaches the effect of substituting a safe for a polluted water supply. Examples of water-borne outbreaks of typhoid fever will be found in the chapter on Water.

Ice.—Ice may, under exceptional circumstances, occasionally be the vehicle by which typhoid bacilli are transferred. Freezing does not kill *B. typhosus*, but there is a great quantitative reduction not only in the act of freezing during storage; hence the danger is greatly lessened. The only suggestive outbreak of typhoid fever attributed to ice was reported by Hutchins and Wheeler in 1903 in the St. Lawrence Hospital, three miles below Ogdensburg. A few other instances in which ice is believed to have conveyed the infection have been reported, but are based upon flimsy evidence. The fact that natural ice is usually stored many weeks or months before it is used is a sanitary safeguard. Manufactured ice made from distilled water and handled with cleanly methods is above reproach. For a discussion of ice in relation to typhoid fever and other infections, see page 1073 *et seq.*

Milk.—Trask collected 317 typhoid epidemics up to 1908 caused by infected milk. Since then many more instances have come to light. Doubt-

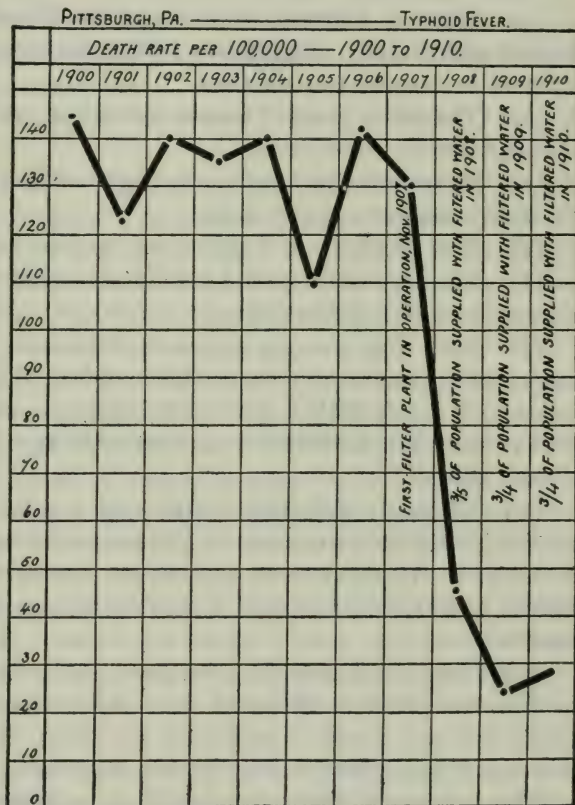


FIG. 8.—IMMEDIATE AND STRIKING EFFECT OF PURIFYING A BADLY INFECTED WATER SUPPLY UPON THE TYPHOID SITUATION.

less many milk outbreaks have escaped attention or have been attributed to water or other sources. The typhoid bacillus grows well in milk, and it is now realized that this medium is a frequent and important mode of communication. Most milk outbreaks are reported from England or America. On account of the almost universal custom of boiling the milk in European and tropical countries, milk outbreaks are rarely reported from these regions. During our four years' study of typhoid fever in Washington, it was found that about 10 per cent of the cases were milk-borne.

The milk usually becomes contaminated on the farm, from a case or a carrier. It may also become infected in transportation, at the city dairy, or in the home. Milk outbreaks come abruptly, rise to a peak like a water epidemic, and subside rather sharply. There are comparatively few secondary cases. Milk-borne epidemics of typhoid fever have certain characteristics which permit ready recognition.

(a) There is a special incidence of the disease on the track of the implicated milk supply. The disease follows the route of the milk wagon. The outbreak is localized to such areas.

(b) The better class of houses is invaded, and persons in better circumstances generally suffer most.

(c) Those who drink milk are chiefly affected and those suffer most who are large consumers of raw milk.

(d) The incidence is high among women and children.

(e) The incubation period may be shortened probably on account of the large amount of infection taken.

(f) More than one case occurs simultaneously in a house. This is a very suspicious circumstance to the epidemiologists. The first indication of a milk outbreak in a city with a good water supply is usually the fact that two or more persons in a household come down with typhoid fever within a few days of each other.

(g) Clinically the disease often runs a mild course, owing to the fact, no doubt, that the virus becomes attenuated in the process of multiplication in the milk. In water-borne typhoid the same germs are ingested that were passed; in milk-borne typhoid it may be the succeeding generations that are ingested.

Milk-borne epidemics are sometimes very extensive. One of the largest on record occurred in Montreal where 5,014 cases with 488 deaths were reported between March 1 and July 16, 1927. In the Boston epidemic in March and April, 1908, 348 of the 410 cases drank the suspected milk.

The number of persons involved in a milk-borne epidemic varies greatly, depending upon the amount of milk infected and other factors. It must not be uncommon for a single bottle of milk or a small quantity to become infected, and thus transmit the disease to one or two persons. Such instances are exceedingly difficult to trace. Ofttimes the milk becomes infected from a carrier. An instance of this occurred in Washington (Georgetown) in 1908.

In this case the milkmaid had typhoid fever eighteen years previously. Examinations showed almost pure culture of *B. typhosus* in her feces. Fifty-five persons who drank the infected milk contracted the disease.

Milk Products.—Fresh milk products, such as cream, ice cream, butter, and buttermilk, and fresh cheese, may contain the typhoid bacillus, and are occasionally media of communication.

Cream contains more bacteria than the milk from which it is taken. The use of infected cream in coffee, on cereals, etc., is sufficient to cause the disease. Several instances in the Washington studies were traced to such use of cream. As a rule, coffee in the cup is not hot enough to kill the typhoid bacillus, if present in the cream added.

In Washington several cases of the disease were traced to ice cream. Mitchell working in my laboratory found that *B. typhosus* survives in ice cream for from twelve to thirty-nine days. Lumsden¹⁵ traced the Birmingham, Alabama, outbreak in 1916, and another in Chattanooga, Tennessee, in 1917, to ice cream. An outbreak at Helm, California, in 1916, was found by Cumming to be due to ice cream.¹⁶

Bruck has shown that the typhoid bacillus will live in butter for twenty-seven days.

Buttermilk may be quite as dangerous as the cream from which it is derived. The acidity and overgrowth of other organisms is said to kill the typhoid bacillus in twenty-four hours. In *cheese* the time of fermentation, antibiosis, etc., lessens the likelihood of survival of the typhoid bacillus. Fresh cream cheese, such as cottage cheese, may be responsible for an occasional case.

Oysters, Mussels, and Shellfish.—The first outbreak of typhoid fever attributed to this source was investigated by Conn at Wesleyan University, Middletown, October, 1894. Twenty-five cases were attributed to eating infected oysters; four died. Not all of those who took sick had clinical typhoid fever. Some had gastro-intestinal disturbances with illness lasting but a few days. About one-quarter of those attending the dinners at which the oysters were served were made ill. Other examples are given on page 765.

L. W. Darra Mair¹⁷ showed that much of the typhoid fever in Belfast, Ireland, from 1897 to 1901, was due to eating cockles and mussels taken from sewage polluted water. The amount of the fever diminished markedly and its seasonal prevalence was changed by betterment of the shellfish situation.

In Brighton, England, J. T. C. Nash¹⁸ proved that much of the typhoid fever in the Borough of Southhead-on-Sea prior to 1899 was due to infected oysters. There was a sharp reduction in the amount of fever when the fore-shore fisheries were stopped, and almost a cessation of all cases when attention was given to all shellfish, including the improved laying and cooking of

¹⁵ *Am. J. Pub. Health*, 1917, 7: 1005.

¹⁶ *J. Am. M. Ass.*, 1917, 68: 16.

¹⁷ *Proc. Roy. Soc., Epidemiology Sec.*, April, 1909, Vol. II, Part 2.

¹⁸ *Idem*.

cockles. The largest outbreak due to oysters occurred in the United States in 1924 (page 765).

In the Washington studies it seems that oysters and shellfish play a minor rôle in the spread of the disease, which occurs mostly in the summer time, while oysters and similar sea food are relished mainly in winter. Comparatively few of the cases studied gave a history of having eaten oysters within thirty days prior to the onset of the disease. Oysters become dangerous when consumed soon after taking them from a polluted bed, or when floated or bloated in infected water. For further discussion of this topic, see page 763.

Fruits and Vegetables.—Vegetables, such as celery, lettuce, water cress, and radishes, partaken raw, and grown on land fertilized with fresh night soil, may be infected, and this probably accounts for an occasional case.¹⁹ Vegetables so contaminated are not made safe by the ordinary methods used in preparation of such food for table use. In large cities it is practically impossible to trace this source of infection. It therefore remains more a suspicion than a conviction. An outbreak which occurred in the summer of 1905, in Springfield, Massachusetts, was attributed to infected fruits and vegetables.

At a wedding breakfast in Philadelphia, June 24th, with forty-three guests in attendance, nineteen persons ate water cress sandwiches.²⁰ Eighteen of these were ill July 22nd with typhoid fever, only two of them being in Philadelphia at the time, while the other sixteen were scattered in suburban territory and in summer resorts along the Atlantic Coast as far north as Maine. The water cress had been secured from a farm on which the sanitary conditions were quite unsatisfactory. A similar outbreak occurred in Hackney, London, in 1903, although the evidence in that outbreak was not so convincing. Morse²¹ reports an outbreak presumably due to celery.

Creel²² found typhoid bacilli upon the tips of leaves of plants cultivated in contaminated soil. Under conditions most unfavorable to *B. typhosus* the infection lasted at least thirty-one days—a period sufficiently long for some varieties of lettuce and radishes to mature.

Vegetable salads are sometimes accused of conveying typhoid and other infections, but in these instances it is more apt to be the salad dressing than the salad itself. See an outbreak reported by Leake due to mayonnaise dressing prepared by a person in the early stage of the disease.²³

Flies.—The evidence is now complete that the common house fly (*Musca domestica*) may convey the infection of typhoid. It is not inappropriately called the typhoid fly. The typhoid bacilli may be smeared upon the feet or other parts of the insect, or may live in the intestinal tract and pass in the dejecta in almost pure culture. Flies live, feed, and breed in fecal matter and decomposing organic substances of all kinds. It is easy to see how they

¹⁹ *J. Infect. Dis.*, 1917, 21: 38.

²⁰ *Eng. News*, Aug. 14, 1923.

²¹ *Rep. Mass. State Bd. Health*, 1899, p. 751.

²² *U. S. Pub. Health Rep.*, 1912, 27: 187.

²³ *U. S. Pub. Health Rep.*, 1920, 35: 2197.

may convey infections from this source to our food, lips, or fingers. Alice Hamilton isolated typhoid bacilli from five out of eighteen house flies captured in Chicago in the privy and on a fence near a sick room during a local water-borne epidemic. It has been shown experimentally that living typhoid bacilli may remain upon the bodies of flies for as long as twenty-three days. Special attention to the rôle played by the fly was given by Reed, Vaughan, and Shakespeare in their studies of the prevalence of typhoid fever in our army camps in 1898. They concluded that flies undoubtedly served as carriers of the infection and attributed about 15 per cent of the cases to this mode of communication. They found that "flies swarm over infected fecal matter in the pits and then deposit it and feed upon the food prepared for the soldiers at the mess tents. In some instances, where lime had recently been sprinkled over the contents of the pits, flies with their feet whitened with lime were seen walking over the food." The danger from fly transmission varies very much, and depends upon circumstances. In a camp it is considerable; in a well sewered city the risk is diminished. In our Washington studies we could find no relation between fly abundance in the summer of 1908 and typhoid prevalence. It is not possible to express mathematically the percentage of cases caused by flies—the figures would vary greatly, depending upon circumstances. The danger of typhoid from flies in cities has doubtless been overstated. However, if only one per cent of the cases were thus transmitted, the suppression of flies would still be quite worth while.

Dust.—Typhoid bacilli soon die when dried, especially when exposed to the sun and air. Dust-borne infection in this disease must be rare. In the South African War there were frequent dust storms in some localities, so that the food was covered with dust and sand. Some of the infection was believed to have been conveyed in this way.

Fomites.—The infection may be conveyed upon soiled linen, blankets, and other objects. It was believed by Reed, Vaughan, and Shakespeare that the clothing, blankets, and tents in the Spanish-American War became infected and were a prime factor in spreading the disease. After the South African War some of the blankets used by the troops were sent back to England and used on a training ship, on which typhoid fever appeared. The blankets were found to be dirty and soiled with fecal matter, from which Klein is reported to have obtained living typhoid bacilli. The danger of fomites contaminated with fresh infection is real, and emphasizes the importance of disinfecting bedding, towels, handkerchiefs, body linen, and other fabrics.

Soil.—The soil, long regarded as the most important factor in the spread of typhoid fever, and by Pettenkofer and others considered an essential element, is now given scant consideration. Pollution of the soil, however, cannot be disregarded. The typhoid bacillus may live for a long time in sewage-soaked earth. A polluted soil may endanger the water, milk, and other foods, or infect indirectly through flies and other means. For more extended information on this subject, see Section VIII.

Contact Infection.—"Contact" is a convenient term to indicate the spread of infection directly or indirectly as a result of close association between the sick and the sound. Actual contact is not necessarily implied. The term is used to indicate the transfer of the infection through a short intervening space in a brief period of time (see page 477). Thus the infection may be passed from one to another through kissing, soiled hands, remnants of food, infected thermometers or tongue depressors, contaminated towels or other fabrics; cups, spoons, glasses, etc. If the nurse infects a cup of milk or glass of water that carries the infection to another member of the household, such cases are included under "contacts." The infection may also be spread in the household by flies, fingers, and various other means, usually difficult to trace, and which are, therefore, all included under this group. Regarded in this light, contacts play a large rôle in the spread of the disease.

Extensive municipal outbreaks have been reported as largely or entirely due to contact infection. Winslow in 1901 studied such an outbreak in Newport. Others have been reported from Knoxville, Winnipeg, Springfield, and from Germany and England. Koch regarded the spread of typhoid in Trier in the light of contact infection. Freeman says that the majority of outbreaks in the smaller towns of Virginia are due to this cause. Extensive outbreaks in institutions are often due to contact with mild cases or carriers. "Flies, fingers, and food" (Sedgwick), and "dirt, diarrhea, and dinner" (Chapin), which too often get sadly confused, explain the occurrence of many a case of contact infection in typhoid fever as well as other diseases.

In army camps with clean water and good milk, contact infection may rise to epidemic proportions. In the Spanish-American War, of 107,361 of our troops in camp, 20,738 contracted typhoid, mostly by "contact"; 1,580 died. Similar conditions prevail in rapidly growing cities, in crowded apartments, and in congested regions with a susceptible population and other favoring conditions. The danger of contact is well shown by the frequency with which nurses, ward attendants, house physicians, and others similarly exposed take typhoid fever. Studies of the incidence of the disease in the Massachusetts General Hospital, Boston, in the Presbyterian Hospital, Philadelphia, and in the Johns Hopkins Hospital, Baltimore, show that typhoid fever was at least twice and may be eight times as prevalent among those who came in close and frequent association with the patient as among the population at large. Further, the disease contracted under such conditions seems to run a course of more than ordinary severity, with a greater number of complications and with a high mortality. This is doubtless due largely to the fact that the contractors receive fresh virulent virus.

In our studies of typhoid fever in Washington we were impressed with the importance and frequency of contact infection in that endemic center. In 1907 we attributed 6 per cent of the cases to contacts; in 1908, 15 per cent, and in 1909, 17 per cent. This included only contact with cases during the febrile stage of the disease. In Strassburg, Kayser attributed 16.8 per cent of the cases occurring during three years in that city to contact infection.

Little groups of cases following a primary case in a suburban focus, in my experience, frequently fall in the category of contacts.

Typhoid fever, in view of all the facts, must now be regarded as a "contagious" disease. We will never have an end of it until it is so regarded and managed accordingly.

TYPHOID VACCINES

Preventive Typhoid Inoculations.—An active immunity to typhoid fever may be induced by injecting dead typhoid bacilli into the subcutaneous tissue. The procedure is *harmless, rational and effective*. It has been put to many severe tests, but has never failed to prove its value.

Our knowledge of inoculations against typhoid fever began with the work of Pfeiffer and Kolle,²⁴ who inoculated two volunteers in 1896. About the same time, Almroth Wright^{25, 26} inoculated several persons, and in 1898 continued the work upon an extensive scale in India upon 4,000 British soldiers. In 1900, during the Boer War, Wright, together with Leishman, prepared a vaccine and supervised the inoculation of 100,000 British troops. The results in India were quite encouraging, but for various reasons the same procedure in South Africa was not as satisfactory as had been anticipated. Prophylactic inoculation on the advice of Koch was used by the Germans in the Herero campaign in southern West Africa in 1904. The prophylactic was voluntary and only about half of the command (7,287 men) availed themselves of it. The results, while good, fell short of expectations. The best results have been obtained in the United States Army where typhoid inoculations were recommended as a voluntary protection in 1909, but were made compulsory in 1911.

Leishman²⁷ in his Harben lecture (1910) explains the lack of success in early years by saying that the vaccine may have been made less efficient by the use of too great heat in killing the bacilli. Further, it should be noted that smaller doses and fewer injections were given then than now. It is well to know that it took about ten years to establish the efficiency of typhoid prophylactic inoculations.

The typhoid vaccines may be prepared in a number of different ways. Usually dead bacilli are used, although live bacilli have been inoculated. The bacilli may be killed either with the aid of heat or germicidal substances; the dead or live bacilli may be sensitized by the addition of anti-typhoid serum; the vaccines may be prepared with pulverized bacilli, from bacillary extracts, or by the use of various chemical methods.

Lipovaccines are made by suspending the bacilli in a fluid fatty substance of suitable consistency. The vegetable oils are better for this purpose than the animal fats. Lipovaccines are absorbed slowly and therefore the entire

²⁴ *Deutsch. med. Wchnschr.*, 1896, 22: 735.

²⁵ *Lancet*, 1896, 1: 807.

²⁶ *Brit. M. J.*, 1897, 1: 256.

²⁷ *J. Roy. Inst. Pub. Health*, 1910, 18: 394.

amount can be inoculated at one time. Experiments indicate that lipovaccines have only about one-half the protective power of saline suspensions.

Usually the vaccine is made from a twenty-four-hour-old culture killed by heating to 53° C. for sixty minutes, depending upon phenol (0.5 per cent) to kill any bacilli that may have survived. Overheating impairs the immunizing power of the vaccine. Most typhoid bacilli die before the temperature reaches 60° C. Some of the strains have a lower thermal death-point. The killed cultures are suspended in saline solution—isotonic salt solution.

Certain strains seem to cause the production of more antibodies than others. In the earlier work it was believed that the more virulent strains produce a greater protection. This is doubtful, for it appears that the protection afforded is not in proportion to the local or febrile reaction, but to the amount and variety of antibodies stimulated. The Rawlins strain is now greatly used in this country for experience has proved its protective power.

The injections are given subcutaneously at intervals of seven days. The first dose is 500,000,000; for each subsequent injection, 1,000,000,000 dead typhoid bacilli are given. Three spaced inoculations is the common practice, although the greater the number of injections, the greater the immunity induced.

The method of giving the typhoid vaccines by the mouth, in accordance with the teachings of Besredka, is in the experimental stage. The results so far reported, though meager, are encouraging, but not promising.

The *reactions* are usually moderate and never serious. They consist of local manifestations; irritation, and inflammation about the site of inoculation, such as pain, redness, swelling, edema; also general symptoms, such as malaise, pains in the back and limbs, and fever. The number and character of the reactions in the experience of the United States Army²⁸ are shown in the following table:

DOSE	NUMBER OF DOSES	REACTION, PER CENT			
		Absent	Mild	Moderate	Severe
First dose	45,680	68.2	28.9	2.4	0.3
Second dose	44,321	71.3	25.7	2.6	0.2
Third dose	38,902	78.0	20.3	1.5	0.1

Children, as a rule, react less than adults. Of 1,101 persons inoculated by Hartsock, 11 per cent showed no reaction, 83 per cent mild reaction, 5 per cent a moderate reaction, and 1 per cent a severe reaction. There is always some local tenderness and redness at the point of inoculation. The systemic symptoms usually pass in twenty-four hours.

The best time to give the treatment is late in the afternoon, for then the severest part of the reaction is over by the morning. The injections are usually given into the subcutaneous tissue of the outer side of the arm or into the abdominal wall; sometimes the interscapular space.

²⁸ *J. Am. M. Ass.*, 1912, 58: 1863.

There is no laboratory index of the degree or duration of the *immunity* produced as a result of the inoculations. The following antibodies appear in the blood: agglutinins, precipitins, opsonins, lysins, stimulins; agglutinins persist for two years and even longer. There are factors involved in the immunity not understood, and therefore, the subsequent freedom of typhoid fever among individuals protected in this manner is the only index of value.

The negative phase advanced by Wright and denied by Leishman and others does not occur. At least there appears to be no increased susceptibility to the disease during the so-called negative phase. There is, therefore, no known objection to giving the prophylactic to those exposed to the disease or during an epidemic. In fact, the typhoid vaccines have been used as a therapeutic agent during the fever.

The immunity varies in degree and also in duration. It comes on gradually and becomes manifest about one week after the third injection. Protection cannot be depended upon to last much more than a year. The evidence is still uncertain, but the consensus of opinion is that the protection should be renewed every two or three years,²⁹ and that two or three inoculations are probably sufficient for practical purposes. In case of special exposure the inoculations should be repeated. Persons who have had typhoid fever need not be inoculated.

In the United States Army typhoid immunization is repeated every three years. In case, however, two or more cases occur in the same command within two weeks, then the entire command is again inoculated. The experience of the American Expeditionary Forces showed that the immunity is not sufficiently strong or durable to protect against mass infections. The immunity may be prolonged or renewed by recourse to reinoculation.

One attack of typhoid fever, however mild, produces, as a rule, a lasting immunity. Second attacks, however, occur. Draschfeld's figures, based on 2,000 persons in the Antwerp Hospital, show that only 0.7 per cent of that number were affected twice. This percentage is doubtless too high, for the figures were collected before our knowledge of paratyphoid infections. The typhoid vaccines do not protect against paratyphoid infections, and contrariwise paratyphoid does not protect against typhoid.

Metchnikoff and Besredka³⁰ failed to protect chimpanzees against typhoid infection by means of killed bacilli, but obtained immunity apparently as definite as that produced by an attack of the disease by the use of living cultures.³¹

Results in the United States Army.—During 1913 the army had only four

²⁹ The experience of the epidemic at Salem, Ohio, showed that our discharged soldiers were protected by their typhoid inoculations for at least two or three years (*J. Am. M. Ass.*, 1921, 76: 1159).

The experience of the A. E. F. demonstrates that under intensive exposure, infection may take place sooner (*J. Am. M. Ass.*, 1919, 78: 1863).

³⁰ *Ann. de l'Inst. Pasteur*, 1911, 25: 865.

³¹ *Ibid.*, 194, 865.

cases of typhoid in the enlisted force of over 80,000 men. Two of these occurred in men who had not been vaccinated; another was among the troops in China, who had been immunized in 1911 and the history of the case is in doubt. In six years, 1909 to 1914, there was only one death from typhoid in the United States Army, while the rate in the country at large averaged over 16.5 per hundred thousand.

During the participation of America in the World War (April, 1917, to November, 1919), four million men served in the United States Army. There were 1,056 cases of typhoid fever, with 156 deaths. This low incidence was clearly due to the results of typhoid inoculation.³²

TYPHOID, 1901-1918, FOR THE WHOLE ARMY, AT HOME AND ABROAD—OFFICERS AND ENLISTED MEN

(Not including native organizations, as Porto Rican Infantry and Philippine Scouts)

YEAR	Mean Strength	CASES		DEATHS			OCCURRING AMONG THOSE WHO WERE VACCINATED	
		Number	Ratios per 1,000 of Mean Strength	Number	Ratios per 1,000 of Mean Strength	Case Fatality	Cases	Deaths
1901	81,885	552	6.74	74	.88	13.0
1902	80,778	565	6.99	69	.85	12.2
1903	67,643	343	5.14	30	.44	8.6
1904	67,311	293	4.35	23	.33	7.8
1905	65,688	206	3.14	20	.30	9.7
1906	65,159	373	5.72	18	.27	4.8
1907	62,523	237	3.79	19	.30	8.0
1908	74,692	239	3.20	24	.31	10.0
1909*	84,077	282	3.35	22	.26	7.8	1	0
1910*	81,434	198	2.43	14	.17	7.1	7	0
1911*	82,802	70	.85	8	.10	11.4	11	1
1912	88,478	27	.31	4	.044	14.8	8	0
1913	80,766	4	.04	2	0
1914	87,228	7	.07	3	.03	42.8	1	0
1915	97,656	8	.08	0
1916	110,454	25	.24	3	.03	12.0
1917	671,156	297	.44	23	.03	7.7
1918	World War	768	.30	133	.05

* Typhoid vaccination was voluntary during 1909 and 1910, and until Sept. 30, 1911, when it was made compulsory for officers and men.

The health record established by the Maneuver Division of the United States Army at San Antonio, Texas, during the summer of 1911, is a triumph in preventive medicine. The division had a mean strength of 12,801 men. All were treated with the typhoid vaccines. The result was that from March 10th to July 10th only two cases of typhoid fever developed; no deaths. One patient was a private of the hospital corps who had not completed his immunization, having taken only two doses. His case was very mild and probably would have been overlooked but for the rule that blood cultures were made in all cases of fever of over forty-eight hours' duration. The other case was a teamster who had not been inoculated. Among the

³² *J. Am. M. Ass.*, 1919, 78: 1863.

12,801 men there were only eleven deaths from all diseases. Typhoid fever prevailed at the time in the neighborhood. Thus, there were forty-nine cases of typhoid fever with nineteen deaths in the city of San Antonio during this period. This contrasts markedly with the typhoid record of the United States Army during the Spanish-American War, when the typhoid record of a division of volunteer troops camped at Jacksonville, Florida, in 1898, under conditions similar to those at San Antonio, was as follows: The division at Jacksonville had 2,693 cases with 248 deaths, which was about the average typhoid incidence of the other camps.

The United States Navy had only seven cases among 50,000 men during the year ending June, 1913. Four of these occurred among men treated at a tropical station, where the vaccine had deteriorated. All the cases were mild. The results in recent years are still better. By contrast, in 1911 the rate was 3.61 per thousand.

Spooner reports that in the Massachusetts General Hospital,³³ among the nurses and others exposed to typhoid fever, 80 per cent of whom had been inoculated during the previous three years, not a case was contracted, and for the first year in the history of the institution (1912) there were no cases among the nurses or attendants. The morbidity rate in training schools for nurses in Massachusetts during three years was nearly nine times greater in the uninoculated than among the inoculated.

Polyvalent Vaccines.—It is now customary to use a vaccine consisting of a mixture of *B. typhosus*, *B. paratyphosus* α , and *B. paratyphosus* β as a routine, because the typhoid vaccines do not protect against paratyphoid infections, and vice versa. During the World War, it was customary to add other cultures such as cholera, dysentery or plague to the mixture for troops serving in localities where these infections prevailed. These various mixtures apparently produce no greater reaction than the single typhoid vaccine and induce a specific active immunity against each virus represented in the mixture. Dose and number of injections remain approximately the same as for single vaccines.

Summary.—The results of typhoid inoculations can no longer be questioned. The morbidity is lowered in those who have been properly "vaccinated"; more striking effect is the lowering of the mortality. Preventive typhoid inoculations involve no risk, and are especially applicable to those unduly exposed to the infection, such as nurses, hospital attendants, physicians, travelers, soldiers in camps, and individuals in the family of a bacillus carrier.

They are especially applicable in endemic centers and during epidemics. They should be taken by persons planning to go to the country for summer vacation and by all persons using unsafe water supplies. Typhoid vaccination should be mandatory in asylums and other custodial institutions. The method is serviceable for general use among the public in endemic foci, but it is a question whether this artificial method of acquiring immunity would serve

³³ *Tr. Ass. Am. Physicians*, 1912.

as good a purpose in the end as fighting the disease along the lines of general sanitation—which has been so successfully done in many countries. It would certainly be a mistake to immunize the population with this artificial method to the neglect of general sanitary improvements, such as good water, safe milk, fly suppression, cleanliness, and personal hygiene. Because a person has received the protection afforded by typhoid inoculations is no reason for reckless disregard of other prophylactic measures. The experience of the American Expeditionary Forces showed that the immunity may be broken down by large amounts of virulent infection, and that *typhoid inoculation is no substitute for sanitary precautions.*

We do not advocate compulsory inoculations with typhoid vaccines for the general public because there are other satisfactory methods of controlling typhoid that are both practical and abiding. On the other hand, we insist on compulsory vaccination with cowpox because this is the only known method of controlling smallpox.

Management of a Case so as to Prevent Spread.³⁴—Success depends upon an early and accurate diagnosis. All cases of typhoid fever and all cases suspected of being typhoid fever should be isolated. This does not mean imprisonment in a lazaretto. The proper place to care for typhoid fever is in a suitable hospital. A private home is a poor makeshift for a hospital, and it is unreasonable to turn a household into a hospital for four to eight weeks or longer. The room in which the patient is treated should be large and well ventilated, and should contain no unnecessary furniture, curtains, carpets, etc. It should be near the bath and must be well screened and kept scrupulously clean, dry sweeping and dusting prohibited. Exposed surfaces should be wiped off daily with a disinfecting solution. Dogs, cats, and pets of all kinds should be kept out of the sick room.

The case should be reported to the health authorities without delay; the house should be placarded so as to warn others. Visiting should be prohibited and no one allowed in the sick room except those who have duty there. The patient should be nursed by one person only, skilled in the technic of preventing the spread of the infection.

The health officer should send an epidemiologist or a public health nurse without delay to the premises to instruct and to see that all necessary measures are being taken. The origin of the infection should be studied so as to prevent further spread from that source.

The disinfection of the stools, urine, sputum, and other discharges is of the first importance, and should be carried out with great care and conscientiousness. For the urine, sufficient bichlorid may be added to make a 1:1,000 solution, or carbolic, 2.5 per cent, or formaldehyd solution, 10 per cent, and allowed to stand one hour before discarding. Stools may be disinfected with bleaching powder, 3 per cent; milk of lime (1:8); cresol, 1 per cent; carbolic acid, 5 per cent; formaldehyd solution, 10 per cent; or unslaked lime and hot water (page 1382). The discharges should be received in a glass or earthen-

³⁴ These general rules, with variations, apply to other communicable infections.

ware vessel containing some of the germicidal solution. Then add more of the solution so that it shall be present in twice the volume of the excreta to be disinfected; disintegrate the masses thoroughly and let stand at least two hours, protected from flies. Masses are so difficult to penetrate that they must be broken up thoroughly with a wooden paddle. It takes a strong carbolic solution twelve hours to penetrate the interior of a small fecal mass; larger masses are impenetrable to most germicides. Urinals, bed pans, etc., should be scalded or washed in a disinfecting solution, and when not in use should contain this solution.

The sputum should be burned. Boiling or strong carbolic acid, cresol or formaldehyd solution are also applicable. As a general rule, more reliance should be placed in heat than in chemical germicides, which may be dangerous in unskilled hands.

The patient should have his own dishes, cups, spoons, glasses, etc., which should be kept and washed apart; they should be scalded after each use. Remnants of lunch, especially meat, milk, gelatin, broths, and other organic food in which the infection may live and even grow, should not be eaten by others. Such remnants may be burned or first boiled and then discarded. Those who nurse the sick should keep out of the kitchen on account of the risk of contaminating the food.

Handkerchiefs, towels, sheets, nightgowns, and all fabrics used about the patient should be disinfected either by boiling, or by immersion for one hour in bichlorid of mercury, 1:1,000, carbolic acid, 2.5 per cent, cresol, 1 per cent, or *liquor cresolis compositus*, 1 to 2 per cent. Germicidal solutions are much more potent when used hot.

The water used to bathe the patient should be disinfected before it is allowed to run into the sewer. This may be done by heat or by adding sufficient carbolic acid or bleaching powder; the latter is cheapest and most practical.

Milk bottles must be kept out of the sick room. In any case, they should be scalded before leaving the house and again disinfected before returning to the dairy.

The thermometer should be kept in formaldehyd solution, alcohol or other suitable germicidal solution. Rectal tubes, especially in hospital practice, must be carefully disinfected each time before using. Individual instruments are preferable. Inexpensive toys and books may be provided during convalescence and then destroyed.

The nurse must protect herself as well as others; a solution of bichlorid should be kept constantly at hand. Every time the patient is bathed, his mouth cleaned, or his buttocks washed, the hands must be washed in soap and water and disinfected. The nurse must exercise especial care if she is to go to the kitchen or to the ice box, etc., as is frequently the case in private houses, where a special diet kitchen cannot be provided.

The nurses, physicians, ward attendants, and others particularly exposed should protect themselves with preventive typhoid inoculations. The physi-

cian should be quite as careful as the nurse, not only that he may not carry the infection to himself or other patients, but also that his practice may serve as a stimulating example.

At the conclusion of the case a general cleansing and disinfection of the room and its contents should be practiced, followed by sunning and airing.

Convalescents should not be given liberty until the danger of bacillus carrying has passed. This may be determined only by bacteriologic examinations of the stools and urine. Four successive negative results of specimens of feces at intervals of several days are required to determine that the carrier state is not established. One examination of the urine is ordinarily sufficient.

The use of hexamethylenamin during the fever diminishes the incidence of bacilluria, and should be a routine practice.

Finally, every case of typhoid fever should be painstakingly traced to its source. In this way, carriers and other foci will be detected and much further harm prevented. The physician has a dual duty in every case of communicable disease: (1) to treat the patient, (2) to protect the community.

Summary—Personal Prophylaxis.—The prevention of typhoid fever may be summed up in the word cleanliness—physical and biological cleanliness. By this is meant not only clean food, especially water and milk, but also cleanliness of person and environment. Typhoid fever has always prevailed where cleanliness is neglected and has diminished where it has been intelligently observed. It is true that typhoid bacilli do not breed in the rubbish and dirt of back yards and alleys, or in unkempt city lots, but these conditions in a community may be taken as an index of the general cleanliness of its inhabitants.

The eradication of typhoid fever is easier in cities than in country districts; clean cities now have less typhoid fever than the surrounding rural regions. Cities can well afford extensive and expensive sanitary works which are beyond the financial possibilities of sparsely settled districts. If a clean water from natural sources is not available, then large volumes of a polluted water may be rendered reasonably safe for municipal use by slow sand filtration and by bleaching powder. Further, cities can afford to inspect their milk supply and to supervise the pasteurization of all that is not safe. These two measures would practically eliminate typhoid infection coming into cities in its food supply—especially if in addition to this a supervision is maintained over oysters and shellfish, and vegetables partaken in their raw state. Further, cities can well afford to employ skilled and experienced health officials and are financially able to engage the services of experts. On the other hand, each farmhouse represents, in miniature, all the problems with which the city deals by wholesale, and is often not financially able to meet its sanitary requirements. The country is the weakest link in our sanitary chain. The good results obtained in the rural region of Yakima, Washington, and in several counties of Maryland by Lumsden, through intelligent and intensive measures, are a great object lesson in rural sanitation.

Cities will find it a paying proposition to suppress flies, rats, and other

vermin, which may be done much more easily than under rural or suburban conditions. This should be done not only on account of the suppression of typhoid fever, but other diseases thus conveyed. The city beautiful must also be the city clean—clean in its cellars, garrets, back yards, empty lots, alleys, stables, wharves, and markets.

To sum up, the main factors in the spread of typhoid fever in our large cities are from cases and carriers through: (1) water; (2) milk; (3) contact; (4) miscellaneous. In a city having a clean water supply the residual typhoid must be attacked along two definite lines, viz., improvement of the milk supply and its pasteurization, and a warfare against the disease in the light of an infection spread from man to man.

The health officer should establish a laboratory for the early diagnosis of cases and for the discovery of carriers. The health officer should at once send a trained agent to every house from which a case of typhoid fever is reported. The visit should be made as early as practicable and with the object of seeing that the stools and urine are properly disinfected, patients isolated, milk bottles scalded, sick rooms screened, house placarded, visiting discouraged, and other necessary measures taken to prevent the spread of the infection. *Each case should be traced so as to prevent further harm from the same source.* Convalescents should not be released until the absence of typhoid bacilli from the urine and stools has been demonstrated by at least four successive examinations. Carriers need not be indefinitely quarantined, but should be prohibited from engaging in any employment having to do with foods, or in which close personal contact, as in nursing, is required. Carriers should be instructed concerning the danger and educated to wash and disinfect their hands thoroughly, especially after a visit to the toilet.

The health officer alone cannot eliminate typhoid fever from a city. He needs the help of the community. Much can be done through education. A stimulating leader may accomplish a world of good through voluntary effort, but in the end it requires comprehensive laws and an energetic enforcement of them, without fear or favor.

The personal prevention of typhoid fever resolves itself into boiling the water, if suspicious; partaking only of milk or fresh milk products that have been pasteurized, and otherwise assuring oneself that all food has been thoroughly cooked. In addition to this, direct and indirect contact with persons who have the disease, or who are known to be carriers, must be avoided. Sanitary habits should be encouraged, especially the one simple precaution of washing the hands after defecation and before eating, and of keeping the fingers and other unnecessary objects away from the mouth and nose. Finally the protection afforded through typhoid inoculations may be used.

PARATYPHOID FEVER

Paratyphoid fever, both clinically and etiologically, is a first cousin of typhoid fever. The two diseases are indistinguishable at the bedside. It

needs the aid of the laboratory to differentiate one from the other. The epidemiology of paratyphoid fever shows some differences from that of typhoid fever.

Paratyphoid is a world-wide infection; outbreaks occur, but, as a rule, are of limited extent. Paratyphoid never occurs as great epidemic catastrophes as does typhoid. Paratyphoid coexists with typhoid both in endemic regions and in epidemic foci. The amount, however, varies greatly. Thus, in Washington somewhat over 1 per cent of all the cases reported as typhoid fever were shown, upon bacteriological examination, to have been paratyphoid. In India the proportion is greater, being as high as 15 per cent (*B. paratyphosus* α predominating). In many localities, about 10 per cent of typhoid fever is in reality paratyphoid fever. During the World War, paratyphoid was more prevalent than typhoid among certain troops vaccinated against typhoid only.

In 1896, Acharde and Bensaude isolated from the urine of a case of apparent enteric fever, and also from a purulent arthritis, following a similar illness, a bacillus which they called the "paratyphoid bacillus." In 1900-1901 Schottmüller obtained from the blood of patients whose symptoms were those of enteric fever two bacilli resembling the paratyphoid bacillus of Acharde and Bensaude. These two organisms were later named by Brion and Kaiser *paratyphosus* α and *paratyphosus* β . Paratyphoid β is much more common and widespread than paratyphoid α . In addition, there are irregular strains of both typhoid and paratyphoid.

The paratyphoid bacillus is a small rod with rounded ends and peritrichal flagellæ resembling the typhoid bacillus. It stains readily with anilin dyes, is decolorized by Gram's method, does not liquefy gelatin, has no spore, and is a facultative aërobe; it clouds bouillon uniformly, and does not produce indol. Upon Endo's medium the paratyphoid colonies are pale, moist, translucent, with a bluish cast, quite similar to typhoid colonies.

The paratyphoid bacillus ferments dextrose, mannite and maltose with the production of gas—whereas the typhoid bacillus produces no gas. They also vary greatly in pathogenicity for the lower animals. Typhoid cultures, as a rule, are not very pathogenic for the lower animals,³⁵ whereas guinea-pigs and mice are susceptible to paratyphoid cultures; most strains will kill guinea-pigs when 1/50 to 1/100 of a loop is injected into the peritoneal cavity. Rabbits are also susceptible; birds are entirely refractory; cattle, dogs, cats, hogs, and sheep show a high degree of resistance to paratyphoid cultures.

A fundamental point of difference between the paratyphoid and the typhoid organisms is that each has specific agglutinating properties. This difference and gas formation in certain sugars are the most important distinguishing features. Care must be taken, in using agglutinins, in differentiating these closely allied species, to guard against confusion through group agglutinins, and also to keep the proagglutinoid zone in mind.

³⁵ Cultures injected into animals do not produce fevers clinically similar to typhoid or paratyphoid fevers.

Paratyphoid bacilli may be found in the blood and internal organs, also in the feces; sometimes in the urine. They produce a continued fever in man closely resembling typhoid fever. As a rule, paratyphoid is milder than typhoid. Lentz³⁶ gives a mortality of 3.3 per cent against typhoid, which averages about 10 per cent.

Paratyphoid fever may be complicated with hemorrhages from the bowels, bronchitis, and pneumonic processes, just as typhoid fever; relapses are rare. It is not definitely known how much of an immunity is conferred by one attack, but it is known that paratyphoid fever does not protect against typhoid fever nor does typhoid protect against paratyphoid. The prophylactic power of each bacterial vaccine is also specific.

Paratyphoid outbreaks have resulted from infected water, milk, and other food. The outbreaks are usually limited in number and extent. The following are characteristic examples: an outbreak in New Rochelle, New York, due to certified milk infected by a carrier, affecting about sixty infants and young children (Williams);³⁷ a milk-borne outbreak among university students in Minnesota, involving 106 cases with two deaths, due to a carrier (Wade and McDaniel);³⁸ an outbreak traced to head cheese infected by an active case, involving forty-four persons in Cascade, Montana (Foard and Walker).³⁹

Paratyphoid Fever and Food Poisoning.—Food poisoning is commonly due to *B. enteritidis* of Gärtner or *B. suispestifer* (see page 642). Some investigators still include *B. paratyphosus* β and these cases were formerly called "meat poisoning."

B. paratyphosus β is closely related to the bacillus of hog-cholera (*B. suispestifer*) and to Gärtner's bacillus (*B. enteritidis*). However, differences can be clearly established by the use of cultural, agglutination and absorption tests. Savage insists that no food poisoning outbreaks have been shown to be due to *B. paratyphosus* β . In most outbreaks of food infection the cases are acute, with the ordinary symptoms of gastro-intestinal irritation—nausea, vomiting, cramps, and diarrhea—but a few protracted cases of febrile infection are met with. Human carriers of the paratyphoid bacillus are always responsible for the infection of milk and other food, while human carriers of the hog-cholera bacillus or Gärtner's bacillus are exceedingly rare, if they occur at all.

CHOLERA

Cholera is an acute specific infection due to the *Spirillum cholerae*, and is characterized by violent purging and rapid collapse with subnormal temperature and cramps. The case fatality ranges from 30 to 80 per cent. Complications and sequelæ are comparatively uncommon.

The incubation period is short, frequently one or two days, rarely over

³⁶ *Centralbl. f. Bakteriöl.*, Referate, Bd. 38.

³⁷ *J. Am. M. Ass.*, 1925, 84: 251.

³⁸ *J. Am. M. Ass.*, 1924, 83: 1416.

³⁹ *U. S. Pub. Health Rep.*, 1921, 36: 2095.

five. One attack produces a mild grade of immunity which is not lasting. The disease is peculiar to man.

The prevention of cholera corresponds to the prevention of typhoid fever. Vigorous measures have been rewarded with signal success. It is quite possible to live in the midst of a raging cholera epidemic without contracting the disease. Within recent years epidemics have been suppressed and the spread of the infection successfully controlled.

Prevalence.—The home of true cholera is the delta of the Ganges, hence it is usually called “Asiatic cholera” to distinguish it from *Cholera nostras* or *Cholera morbus*. That British India is the world’s chief endemic center of cholera is shown by the fact that during the past twenty years there were 7,383,296 deaths, distributed as follows:

DEATHS FROM CHOLERA IN INDIA, 1904-1923 *

Year	Deaths	Year	Deaths
1904	193,457	1914	299,174
1905	442,508	1915	409,498
1906	713,664	1916	300,208
1907	413,180	1917	277,537
1908	607,278	1918	571,643
1909	242,848	1919	602,304
1910	435,077	1920	131,203
1911	356,157	1921	459,843
1912	425,192	1922	118,703
1913	311,127	1923	72,695

* Epidemiological Intelligence No. 8, Health Organization of the League of Nations, Aug., 1924, pp. 14, 15.

In 1923, which was the lowest on record for thirty-four years, the 72,695 deaths in India represented about 97 per cent of the known cholera deaths in the world. Successive waves have swept across Russia during the past twenty years, with a severe epidemic in 1910 (230,232 cases reported) and another in 1921 (207,389 cases reported).

It is only in the nineteenth century that cholera has spread along the routes of trade and travel to Europe (first in 1830); Africa, and America in 1832. There have been four periods of pandemic tendency; one from 1817 to 1823, another 1826 to 1837, a third 1846 to 1862, and a fourth from 1864 to 1875. In 1832 it entered the United States by way of New York and Quebec and reached as far west as the military posts of the upper Mississippi. The disease recurred in 1835 and 1836. In 1848 it entered the country through New Orleans and spread widely up the Mississippi and was dragged across the continent by the searchers for gold all the way to California (1849). It again prevailed widely through this country in 1854, having been introduced by immigrant ships into New York. In 1866 and 1867 there were less extensive epidemics. In 1873 it again appeared in the United States, but did not prevail widely. Since then, only occasional cases at seaports have been reported. In 1892 the great epidemic of Hamburg occurred, and the disease threatened to become pandemic. Cases were brought by transatlantic liners to New York, and a few cases occurred in the city, but its spread was pre-

vented by aggressive measures. Cholera has prevailed for years in the Philippines, but is now under control. While the home of cholera is in the tropics, there is scarcely a country in the world that has not been visited some time or other by the ravages of this fatal disease.

The Cause and Contributing Causes of Cholera.—The *Spirillum cholerae*, discovered by Koch in 1883 and called by him the “comma bacillus” or the cholera vibrio, is the undisputed cause of the disease.⁴⁰ The conditions of infection, as in other diseases, may be complex. Not everyone who takes the specific microörganism by the mouth necessarily gets the disease, but without it there can be no cholera. Many cholera vibrios probably die in the acid juices of the stomach. There is, therefore, perhaps less danger in taking small amounts of infection during active digestion than upon an empty stomach, for it has been shown experimentally that cold drinks do not stay long in an empty stomach, but pass quickly through the pylorus. After the cholera vibrio has passed the pylorus and reaches the alkaline juices of the intestines, it may find ideal conditions for growth or may still have a hard struggle for existence. Here symbiosis or antibiosis must play a dominant rôle. It is well known in all cholera epidemics that a deranged digestion is an important predisposing factor to the disease. An attack may thus be precipitated in healthy carriers. In the Hamburg epidemic a marked access of cases on Monday following the Sunday indiscretions was noted. Raw fruits, crude fibrous vegetables, and fermentable food, difficult of digestion, seem to favor the growth and multiplication of the cholera vibrio in the intestinal tract. In the light of this view such food may often be the predisposing factor rather than the medium which conveys the infection. Freedom from the disease also depends on specific immunity.

Pettenkofer stoutly maintained that the “comma bacillus” was only one of the factors in the etiology of the disease. He placed special importance upon the condition of the host and his environment, and considered at least three fundamental factors in his X, Y, Z theory: X is the germ, Y the host or soil, Z the environment. In his view the disease may aptly be compared to fermentation, in which X represents the yeast, Y the carbohydrate, and Z the temperature, moisture, reaction, and other essential conditions for the growth and activity of the yeast. Pettenkofer maintained that X without Y and Z would not produce cholera, that is, while the cholera vibrio was pathogenic in India or Hamburg (1892), where Y and Z were favorable, it would be harmless in Munich, where Y and Z were unfavorable. To prove this theory, he and his assistant, Emmerich, drank pure cultures of cholera after first rendering the stomach contents alkaline. Pettenkofer, then an old man, soon developed a diarrhea; Emmerich had a choleraic attack. Pettenkofer did not regard his own case as cholera, and insisted that the inconclusive

⁴⁰ Choleraic symptoms may be induced by other organisms, or by certain poisons. These cases, known as *cholera nostras* or *cholera morbus*, are becoming increasingly rare. Winter cholera is the name of a mild diarrheal disease in this country. It is of unknown etiology, but has nothing to do with true cholera. Most outbreaks seem to be water-borne.

results lent confirmation to his theory. Metchnikoff and his pupils drank pure culture of vibrios from four different sources and were able to produce true Asiatic cholera, which although mild had all the classical symptoms of the disease: rice water stools, subnormal temperature, vomiting, cramps, suppression of urine, and vibrios in almost pure culture in the stools. Similar convincing experiments have occurred among laboratory workers, who have accidentally got pure cultures of cholera into their mouths. On the other hand, a number of persons who imitated Pettenkofer's experiment were not affected.

The cholera vibrio is an aerobic, liquefying, actively motile spirillum. It stains best with carbol fuchsin, 1:10. It grows well on ordinary culture media, but is very susceptible to acids and germicides. It is somewhat frailer than the average spore-free bacteria.

Diagnosis.—The diagnosis of cholera depends upon isolation and identification of the cholera vibrio in pure culture. This has become comparatively simple, but great care must be taken not to confuse the true vibrio of cholera with a great host of other microorganisms which closely resemble it.

A presumptive diagnosis of cholera may be made by finding large numbers of comma-shaped bacilli on direct microscopic examination of stained preparations, or in hanging drops of the mucous flakes ordinarily found in cholera stools. This test is only presumptive, the final criteria being the *two reactions which are specific and reliable*—*Pfeiffer's phenomenon and agglutination*.

Dependence should not be placed upon morphological characters, cultural peculiarities, or pathogenicity upon laboratory animals, for these do not furnish the means of certainly defining the cholera vibrio. For isolation, agar is preferable to gelatin, formerly so much used. The suspected material should be planted upon the surface of ordinary alkaline agar or upon Dieudonné's medium, using one of the small rice-like flakes or an equivalent quantity of feces.

When it is suspected that the cholera vibrios are few in number, they may be enriched by first planting in Dunham's solution. Approximately 1 c.c. of fecal matter should be placed in 50 c.c. of the peptone solution. This is incubated at 37° C., and in from six to eight hours a loopful is taken from the surface and transferred to ordinary agar or Dieudonné's medium.⁴¹ Suspicious colonies are fished and studied further.

Kolle and Gotchlich have shown from a large number of observations that with strongly agglutinative serum, the titer of which reaches 1:4,000, the agglutinative power for common vibrios, not cholera, does not, as a general rule, exceed 1:50 and rarely reaches 1:200; agglutination in dilutions of 1:500 has been only very exceptionally observed. On the contrary, the true cholera vibrios agglutinate in dilutions varying from 1:1,000 and 1:20,000 and sometimes even higher. Therefore, with a specific agglutinating serum having a titer of 1:4,000, any organism which is agglutinated in 1:1,000 may

⁴¹ U. S. Hyg. Lab. Bull., No. 91, 1913.

be considered true cholera. Organisms agglutinating in dilutions of 1:500 should be regarded as doubtful.

Modes of Transmission.—Cholera is spread by man from place to place. It follows the lines of trade and travel. Seaports are invariably first attacked. The epidemic at Hamburg in 1892 was brought to that port by immigrants on board vessels from Russia. There are many similar instances. In 1849 many a gold hunter found another Eldorado than the one he was searching for, as cholera was dragged across the continent by the caravans seeking fortunes in California. The same thing takes place in the Indian pilgrimages to Mecca.

The cholera vibrio enters the digestive tract through the mouth. It is taken in food and drink. Infected water is a frequent medium of transference, and probably the chief vector of the great epidemic outbursts. Cholera, however, may be transferred from man to man directly, also indirectly by flies, fingers, food, and all the innumerable channels from the anus of one man to the mouth of another that have been described in the case of typhoid.

In endemic or residual cholera, water-borne infection plays a minor rôle. This was well proved in the sanitary campaign against the disease in the Philippine Islands, in which the disease was conquered in the light of a contact infection communicated rather directly from man to man. Convalescent carriers and healthy passive carriers are common means of spreading the infection.

The cholera vibrio leaves the body in enormous numbers in the dejecta, also sometimes in the matter vomited. The cholera vibrio is seldom found in the blood, but has been located in the gall-bladder and other tissues. Disinfection in this disease must, therefore, be concentrated upon the discharge from the bowels and mouth, and also the urine at the bedside.

Water.—The cholera vibrio may live and even multiply in water. Koch in his original investigations found the organism in the foul water of a tank in India which was used by the natives for drinking purposes. It has been shown by experiment that the cholera vibrio may multiply to some extent in sterilized river water or well water; and that it preserves its vitality in such water for several weeks or even months. In recent times cholera organisms have been found not infrequently in the water of wells, water mains, rivers, harbors, canals, and even sea water (the North Sea near the mouth of the Elbe), which have become contaminated with the discharges of cholera patients. It is plain from the nature of the case that infected water must play a very large rôle in spreading this infection.

The earliest and now classic instance in favor of the water-borne theory we owe to John Snow. This is the Broad Street pump outbreak in London in 1854, an account of which will be found on page 1054.

The best example of water-borne cholera is the Hamburg epidemic of 1892, which I was fortunate enough to see in part. In this case no link in the chain of evidence is missing. Cholera was brought to Hamburg by immigrants from Russia. The water of the Elbe was infected with their discharges. The *Spirillum cholerae* was readily isolated from the river water which was dis-

tributed throughout the city for drinking purposes without purification. The sewers of Hamburg emptied into the river Elbe near the water intake, which produced a vicious circle. An account of the epidemic will be found on page 1058.

Other Modes of Transference.—The fact that water-borne infection is practically the only cause of the large cholera epidemics must not overshadow the importance of other modes of transmission. In addition to the violent outbreaks, cholera occurs in nests or smolders like endemic typhoid. It is difficult to trace the connection between cases in endemic areas. Thus, a careful study of the cholera situation in Manila disclosed the fact that isolated cases would crop up at widely different points without any evident connection between them. Cholera carriers were suspected and later demonstrated. At irregular intervals of several years the disease would gather force, and cases multiply, until it assumed epidemic proportions, entirely independent, it is believed, of the water supply. The way cholera was dragged across our continent by the "forty-niners," and its occurrence among the Mecca pilgrims, are instances of its spread largely independent of infected water. The outbreaks in Russia which I investigated in 1922 were in no case water-borne.

Contact Infection.—Contact infection in cholera must not be underestimated. Persons frequently become infected through handling the dejecta or through freshly infected fomites, such as soiled linen. Direct transmission from person to person was formerly seen among physicians and nurses. In congested quarters, where many persons live under uncleanly conditions, contact infection plays an important part. The same thing may be seen on board vessels, in which case the disease may be confined to the firemen, stewards, or some other limited group who are required to live in close contact with each other. Epidemic outbreaks due to contact infection have been recorded, such as the thirty cases which occurred in the fall of 1892 in Boizenburg.

Cholera is not highly "contagious," for physicians, nurses, and others in close contact with patients need not become infected provided intelligent measures are adopted. On the other hand, there is great danger of the spread of the disease through devious and hidden routes, as is the case with typhoid and dysentery.

Milk may be contaminated, but is probably not a frequent medium of infection, for the reason that its acid reaction is inimical to the cholera vibrio. Green vegetables and fruit that have been washed in an infected water may convey the disease. The bacilli may live on fresh bread, butter, and meat for from six to eight days, if not too acid.

Flies, etc.—It has been shown that the cholera vibrios may live in the intestines of flies for at least three days, and these and other insects may also spread the infection mechanically. The cholera vibrio is a frail organism and dies rapidly when dried or exposed to light and other injurious influences. Infection through the air is, therefore, not to be dreaded. Fomites, such as bed and body linen or other objects, including spoons, dishes, toys, etc., contaminated with the discharges, can be regarded as possible sources of infection.

There is, however, a special limitation in this case, owing to the fact that this organism is so readily destroyed by desiccation and crowded out by saprophytic microorganisms. Thus, as a rule, only fresh dejecta and freshly contaminated objects are possible hazards.

Bacillus Carriers.⁴²—The cholera vibrios are passed in enormous numbers in the feces during the early part of the disease. They usually disappear after the fourth to the fourteenth day, but may remain one or two months. These are convalescent carriers. Chronic carriers are rare. Healthy or passive carriers occur and are an important and insidious means of spreading the infection; they may also develop the disease.

McLaughlin (1908) found bacillus carriers numerous in epidemic centers in the Philippine Islands—6 to 7 per cent among healthy individuals living in infected neighborhoods in Manila—but rare in neighborhoods having few cases. Carriers are the principal factor in the spread of endemic cholera and in keeping the infection alive.

Detection of Carriers.—Specimens are best obtained by administering a saline cathartic or by using a rectal tube with “eyes” cut into it.

Several different methods for the detection of cholera carriers are applicable. All of them are based upon the facility with which the vibrio grows upon Dunham’s alkaline peptone solution. Particles of feces are planted in this medium and subsequently examined for comma-shaped microorganisms. If found, the diagnosis is presumptive. Pure cultures should then be made and studied for agglutination.

Immunity and Prophylactic Inoculations.—The immunity produced by an attack of the disease is of short duration. An active immunity may readily be induced in susceptible animals against cholera vibrios. Attempts to immunize man with cholera vaccines were made by Ferran in Spain in 1884, and Haffkine in India in 1895. These early attempts did not produce a high degree of immunity, and the results were not conclusive. It has since been shown by Kolle, Powell, Simpson, Wright and others that prophylactic vaccination against cholera is a practical and useful method of protection. Convincing figures are not readily obtainable, but all observers are assured of its usefulness. Cholera vaccines, either alone or mixed with typhoid vaccines, were much used in the war to protect the troops serving in districts where the disease prevailed. Cholera vaccines have been used much in Japan, where good results are reported. Most of the inconclusive results were due to using too small a dose or not giving a sufficient number of inoculations.

The dose of the Haffkine prophylactic for an adult is 0.5 c.c. of an emulsion of an agar slant in 5 c.c. of sterile water, injected subcutaneously. It is not possible to produce cholera by introducing the spirilla parenterally.

Kolle uses a more practical method, standardized so that one c.c. of the

⁴² The recognition of the carrier principle in connection with the dissemination of various communicable infections was first established by Robert Koch in the course of his well-known investigations into the cholera outbreak which occurred in Germany during the winter of 1892-1893 (*Ztschr. f. Hyg.*, 1893, 15: 83).

suspension contains 2 milligrams of culture. The vibrios are grown on agar slants, suspended in salt solution and then heated to 58° C. for one hour; 0.5 c.c. of phenol is added as a preservative. One c.c. (2 mg. of culture) is given subcutaneously, three or four times, at intervals of five to seven days.

The immunity produced by these protective inoculations lasts about a year, when the specific antibodies begin to diminish in the blood-serum.

There seems to be little doubt in Japan concerning the value of the protection afforded by the inoculation of dead cultures, for in the district of Hiogo, during the epidemic of 1902, 77,907 persons were inoculated. Of these 47, or 0.06 per cent, took cholera, and 20, or 0.2 per cent, died, whereas, among 825,287 persons not inoculated, 1,152, or 0.13 per cent, took the disease, and 863, or 0.1 per cent, died. It is especially noteworthy that all the cases among the inoculated group were in those who received an injection of 2 mg. of the dead culture. Later 4 mg. were used, and in this group no cases occurred. Good results were also obtained in the Russian epidemic of 1908-1909, and in the World War.

Protective inoculations as a prophylactic measure against cholera will never be popular or necessary in communities with sufficient sanitation. It is, however, of value in camps, armies on the march, for physicians, nurses, ward tenders, travelers, and others especially exposed. The method of oral administration, according to Besredka, is still experimental.

Quarantine.—Cholera is an infection which fully justifies maritime quarantine practice. The disease may be blocked by a careful system of inspection, detention, and disinfection at the seaport. In order for maritime quarantine to be effective for cholera, it must have the assistance of a bacteriological laboratory to diagnose cases and recognize carriers. A bacteriological examination of the feces of all persons coming from cholera-infected places has entirely supplanted the old-fashioned period of indiscriminate detention for five days.

In the summer of 1912 the quarantine authorities at the large seaports on our Atlantic littoral examined about 34,000 specimens of bowel discharges from passengers and crews from cholera-infected ports. At the New York quarantine the cholera vibrio was isolated from twenty-eight persons sick with the disease, and twenty-seven healthy persons were found to be discharging vibrios in their feces. These carriers could not have been discovered except by laboratory examination. Seven cases of cholera were detected at other ports by the same methods. There can be no doubt that the adoption of this measure kept cholera out of the country.

Similar measures used at the port of departure can be successfully applied. Owing to the fact that most cholera carriers soon purge themselves of the vibrios, the detention in quarantine is ordinarily not prolonged. Three or more negative bacteriological findings at intervals of two days should be required before releasing a carrier (see page 133).

Personal prophylaxis requires, first of all, scrupulous cleanliness on the part of the person and his surroundings. Those who handle cholera patients,

their dejecta, or infected articles must carefully disinfect their hands each time, and should under no circumstances eat or drink anything in the sick room. During cholera times all water and food of every description should be boiled or thoroughly cooked just before it is partaken. Great care must be exercised that the water or food does not become infected after it has been boiled or cooked. The usual measures should be taken to guard against flies and other vermin. With strict attention to these measures, it is possible to avoid the infection. In addition, however, attention to general hygiene and especially to the character of the food and regularity of meals should be given. Slight attacks of indigestion and diarrhea should receive prompt medical attention. Those exposed should protect themselves with cholera inoculations. The disinfection of fecal discharges and other special measures are the same as for typhoid fever.

Summary.—A summary of the preventive measures necessary to control an epidemic of cholera are: centralization of authority in one person; establishment of a system of securing and reporting information; organization of the personnel for the sanitary work; enactment of necessary ordinances; house to house inspection; safe disposal of feces of entire population; provision for a safe water supply; supervisory control of food and drink; a search for, and control of carriers; isolation and care of patients in special hospitals; separate hospitals or wards for suspects; a laboratory; detention camps or barracks for those desiring to leave the infected area; disinfection, etc. For further discussion concerning the control of epidemics, see page 523.

DYSENTERY

(*Flux*)

Dysentery is a form of intestinal flux, characterized by frequent passages of blood and mucus, and straining at stool. Diarrheas of this type may be due to several causes, and for the purpose of prevention we may consider all dysenteries under four heads: (1) bacillary, due to *Bacillus dysenteriae*; (2) amebic, due to *Endamæba histolytica*; (3) balantidial, due to a ciliate, *Balan-tidium coli*; and (4) symptomatic, due to a variety of irritating poisons.

Contrast between Bacillary and Amebic Dysentery.—There are fundamental distinctions between these two diseases which are summarized in the following parallel columns:

BACILLARY DYSENTERY	AMEBIC DYSENTERY
Cause: <i>Bacillus dysenteriae</i>	Cause: <i>Endamæba histolytica</i>
An acute, febrile, self-limiting disease with severe toxemia (sometimes very mild).	An insidious and chronic infection, often afebrile, and little or no signs of toxemia (sometimes acute with fever).
Lesions are diffuse in the large intestine and sometimes occur in the small intestine; varies greatly in severity.	Lesions localized in large bowel, seldom in the small intestine; ulcers undermined.

BACILLARY DYSENTERY	AMEBIC DYSENTERY
Cause: <i>Bacillus dysenteriae</i>	Cause: <i>Endamæba histolytica</i>
Stools: abundant; cellular exudate chiefly polymorphonuclears; toxic necrosis produces "ghost cells"; evidence of intense inflammation.	Stools: scanty exudate, mononuclears predominate, only a few polymorphonuclears. Proteolytic action in cells produces pyknotic nuclear remains. Absence of evidence of inflammatory reaction and toxic necrosis.
Few sequelæ.	Liver abscess common sequela.
Incubation: 2 to 7 days.	Prolonged and variable.
Definite immunity.	No immunity.
Chief cause of infantile "diarrheas."	Uncommon in children in the temperate zone.
Temporary active carriers play a rôle but probably are not the chief agent in spread; chronic carriers rare.	Chronic carriers are frequent and play chief rôle in spreading infection.
Epidemics frequent and often widespread; outbreaks are so common in institutions that the disease is called jail, asylum, camp and ship dysentery.	An endemic disease; does not occur in epidemic outbreaks of any magnitude.
Most prevalent and most severe in tropical and subtropical countries; widespread in temperate climes.	Same geographic distribution, but less common in temperate and cold climes.
Predominant form of dysentery among troops during war campaigns.	Forms a small portion of the fatal cases of dysentery among troops in service.
Mode of spread like typhoid: water, milk, food, flies, fingers, contacts, carriers, etc.	Spread by intimate personal contact; probably by flies and rats; water also a probable factor.
Vaccines are protective.	No specific prophylactic is known.
Antidysenteric serums are remedial.	Emetin and stovarsol cure the symptoms and often eradicate infection.

BACILLARY DYSENTERY

Bacillary dysentery is a specific infection caused by *Bacillus dysenteriae*, of which there are a number of strains. It is usually a self-limiting disease, running an acute course without complications or sequelæ. Bacillary dysentery occurs in all countries and climes, with a tendency to summer prevalence. All grades of severity are met with, and the case fatality rate varies greatly, often running as high as 30 per cent. Bacillary dysentery increases in amount and severity as we approach the equator, but severe outbreaks occur in temperate and even in Arctic regions. It affects all ages and is one of the chief causes of summer diarrheas in infants. The disease occurs as great epidemics, in local outbreaks and sporadically. In all armies up to recent times it has caused great ravages. Dysentery bacilli have killed many more soldiers in the world's history than bullets. In the federal service during the Civil War, there were 259,071 cases of acute dysentery. Practically all epidemics of dysentery in camps and institutions are bacillary. Over-

crowding, lack of cleanliness, and other unhygienic conditions favor its spread so that it is called famine, asylum, jail, ship, or camp dysentery.

B. dysenteriae is a Gram-negative, spore-free, non-liquefying bacillus belonging to the typhoid-colon group. There are many strains, which group themselves in four types: Shiga, Flexner, Strong, and Y. The Flexner type is most frequently found in the United States. The symptoms and lesions produced by the different strains are identical, although they vary greatly in severity. The Shiga strain is the most virulent. A strong toxin may be obtained from the Shiga strain which when injected intravenously into rabbits produces a fatal toxemia with a faithful reproduction of the symptoms and lesions of bacillary dysentery. Two poisonous substances have been demonstrated by Olitsky and Kligler⁴³ in this toxin: (1) an exotoxin, which acts especially upon the nerves and is therefore called a neurotoxin; (2) another, which is probably an endotoxin, acts upon the intestinal tract and is therefore called an enterotoxin.

The exotoxin is an early product of the growth of the bacillus, is relatively heat-labile and yields an anti-exotoxic immune serum. The endotoxin is a product of autolysis or disintegration of the bacillus with the resultant liberation of intracellular components, is heat-labile and is not neutralized by anti-exotoxic serum.⁴⁴

Resistance.—The dysentery bacillus has about the same resistance to germicides and other unfavorable conditions as the general class of spore-free bacteria. It dies in about eight to ten days when dried. It may live for months when moist. It is sensitive to acids. Phenol, 0.5 per cent, kills the dysentery bacillus in six hours, 1 per cent in thirty minutes, 3 per cent in one to two minutes. Bichlorid of mercury, 1:1,000, kills it at once, and direct sunlight in about one-half an hour. I have found certain strains of the dysentery bacilli somewhat more resistant to heat than the typhoid bacillus. They are killed with certainty at 58° C. for one hour, or at 60° C. for twenty minutes. The dysentery bacillus resists cold and may live for months when frozen.

Bacillus Carrying.—Bacillus carrying in dysentery probably plays a more important part in spreading the disease than has been suspected. As a rule, the bacilli soon disappear from the stools in the light cases, but they may continue from two to six weeks after clinical recovery. Shiga has found them more persistent in some instances.

It is doubtful whether permanent healthy carriers have even been discovered, although Fletcher and MacKinnon reported carriers among non-dysenteric convalescent English soldiers who gave no history of the disease. The large surveys made upon soldiers invalided to England during the World War not infrequently disclosed convalescent carriers among both dysenteric and non-dysenteric men. In 1917-1918, the feces of 4,154 healthy persons in and around Boston were examined in my laboratory without finding

⁴³ *J. Exper. Med.*, 1920, 31: 19.

⁴⁴ *J. Exper. Med.*, 1923, 37: 767.

a single carrier. It is now believed by many that the convalescent carriers are essentially cases of relapsing dysentery in whom the disease is more or less chronic or latent with acute exacerbations. Investigations indicate that the convalescent carrier constitutes the chief class of carriers instrumental in the spread of the infection.

Immunity.—The susceptibility to dysentery varies greatly. This is doubtless due in part to the bacterial flora of the intestinal tract as well as the conditions of the intestinal mucosa. Symbiosis or antibiosis must play a very important rôle either in permitting or hindering the growth of the dysentery bacillus in the intestinal tract. Further, the specific resistance of the intestinal mucosa, which is the first line of defense, must play a critical rôle in determining whether or not dysentery bacilli can penetrate and produce the disease. There is still a question whether a true immunity is acquired by one attack of bacillary dysentery. This seems probable, although it is not unusual for a person to have two or more attacks of dysentery in one season. Kolle looks upon this as an exacerbation of a chronic type brought on by errors of diet, exposure, etc. The experiments on animals indicate that dysentery probably belongs to that group of diseases which leave a certain amount of protection after one attack. A definite and high grade of immunity can be produced experimentally in several of the lower animals. Upon this question, however, we need light. Horses may be immunized to a high degree, and their sera contain a certain amount of antitoxin and other antibodies. This serum has been used in treatment, but has no particular value as a preventive.

Methods of Spread and Prevention.—The methods of spread and prevention of dysentery are an exact counterpart of those for typhoid fever, varying only in degree. Food, fingers, flies, contacts and carriers play a similar part. Milk- and water-borne outbreaks have been traced, although less frequently than in the case of typhoid fever. An interesting outbreak spread by a baker was reported by Louste and Godlewski⁴⁵ in a village of six hundred souls. There were thirty-one cases of bacillary dysentery and six deaths in less than a month.

Bacillary dysentery is a common disease in infants, and it would be a wise precaution to consider all cases of infantile diarrhea as infectious and to take precautions accordingly. The protection of babies against dysentery requires biologic cleanliness, sanitary isolation, and a ceaseless and intelligent technic. Dysentery is one of the greatest single factors in high infant mortality (see page 508).

Good results were claimed by the use of dysentery vaccines among certain troops during the World War, but convincing figures are difficult to obtain. The use of dysentery vaccines by the mouth is in the experimental stage, but deserves further trial, especially in view of the discomfort and even danger attending the subcutaneous method of vaccination with the Shiga type.

⁴⁵ *Bull. Soc. méd. d. hôp.*, 1919, 43: 1022.

AMEBIC DYSENTERY*(Amebiasis, Tropical Dysentery)*

Amebic dysentery is caused by the *Endamoeba histolytica*, and is characterized by an ulcerative colitis and frequent, blood-stained mucoid stools. There is a tendency to chronicity and a special liability to the formation of abscesses of the liver.⁴⁶ Amebic dysentery often starts insidiously and shows a tendency to become chronic with exacerbations and relapses. The attacks vary from comparatively mild intermittent diarrhea to the acute fulminating type of bloody flux.

Amebic dysentery has always been considered a tropical disease, but while it is more prevalent there, we now know that it may occur wherever there are carriers of *Endamoeba histolytica*. During and just after the World War, numerous protozoan surveys, made in England,⁴⁷ on the Continent and in the United States,⁴⁸ demonstrated a surprisingly large number of carriers of the ameba of dysentery. Amebic dysentery occurs in persons who have never been in the tropics; in fact, the disease is widespread throughout the temperate zone and has been reported as far north as Alaska.⁴⁹

Amebic dysentery occurs mainly in endemic form and does not cause explosive outbreaks such as we see in typhoid fever and bacillary dysentery. The reason for this lies in the fact that the amebæ are transferred almost always by rather direct and intimate contact. There is evidence, however, to show that flies⁵⁰ and wild rats⁵¹ may be important agents in the dissemination of the cysts (the infective stage) of the organism. Milk-borne outbreaks do not occur as in the case of bacillary dysentery, but undoubtedly water is instrumental in the spread of the disease by carrying the feces containing the cysts and contaminating the water and food supplies. Evidence of water-borne infection is seen in the comparatively small number of cases of amebic dysentery during the dry months in India and the increase immediately following the onset of the Monsoon season of heavy rains. In Panama, the disease decreased markedly with a good water supply and other sanitary improvements. In water the life of the cyst is prolonged and they remain apparently normal in appearance for a considerable number of days.⁵² Drying destroys them in a few minutes. Lettuce and other fresh vegetables, especially from soil fertilized with human feces, may be contaminated with amebic cysts. Family infections are common; that is, with one case of dysentery in a family, most or all the members may show infection, even though they do not have symptoms. Cooks and waiters, if chronic carriers, may also

⁴⁶ Sir Leonard Rogers, *Bowel Diseases in the Tropics*, London, 1921.

⁴⁷ *Medical Research Council*, Spec. Rep., Series, No. 59, 1921.

⁴⁸ *U. S. Hyg. Lab. Bull.*, No. 133, 1923.

⁴⁹ *Northwest Med.*, n. s., 1911, 3: 51.

⁵⁰ C. M. Wenyon and F. W. O'Connor, *Human Intestinal Protozoa in the Near East*, London, 1917.

⁵¹ *J. Am. M. Ass.*, 1915, 65: 2232.

⁵² *Am. J. Hyg.*, 1921, 1: 526.

contaminate foods. The cysts are not constantly in the stools, but appear and disappear irregularly.

Endamaba histolytica is a parasite living in the large bowel. Its life cycle is not completely known. Two stages have been studied: (1) a motile stage which may invade the tissues and produce ulcers and (2) an encysted stage which is more resistant and passes out of the body with the feces. The cyst is the infective stage and when ingested it hatches out in the bowel to establish itself in the colon. It is probably not specific for man, as previously believed, for rats harbor an ameba⁵³ which is indistinguishable from *E. histolytica*. Kittens may be infected by feeding them cysts, by inoculating cysts directly into the cecum,⁵⁴ or by injecting the motile and encysted forms into the rectum. In the kittens an acute amebic dysentery is produced and occasionally a liver abscess. Recently *E. histolytica* has been cultivated in artificial media by Boeck and Drbohlav⁵⁵ and this work has been confirmed by others. Dobell⁵⁶ has obtained encystment and excystment in his cultures so that the complete life history should be known shortly.

Endamaba histolytica is seen in the discharges from the bowels and in the pus from liver abscesses. In the human feces there is one other but harmless ameba (*Endamaba coli*) with which it is most often confused. The cytoplasm of *E. coli* is invariably filled with a large number of ingested bacterial and food remains. *E. histolytica* cysts are smaller (10 to 12 μ in diameter usually) than those of *E. coli* (15 to 22 μ) and when mature contain four nuclei, while the latter has eight. This difference is an absolute criterion in diagnosis. The motile forms of *E. histolytica* are distinguished from those of *E. coli* by their (1) slightly greenish color, (2) distinct hyaline ectoplasm and clear pseudopodia, (3) faint nucleus with a central granule, (4) rapid slug-like motility, and (5) the presence of red blood cells whenever they are seen in the cytoplasm.

It is also claimed that *E. histolytica* is the cause of Hodgkin's disease⁵⁷ and arthritis deformans Type II of Ely.⁵⁸ Thus far, there is no experimental proof to substantiate this claim, and furthermore, in the tropics these two affections do not prevail to the same extent as amebic dysentery.

Emetin, one of the alkaloids of ipecac, is a specific for amebic dysentery and amebic hepatitis. Rogers, in 1907, first demonstrated its use in the cure of amebic abscesses in the liver. Experience during the World War has shown that there are two generally accepted methods of treatment⁵⁹; (1) the daily administration by mouth of three grains of emetin-bismuthiodid in hard gelatin capsules for twelve consecutive days, and (2) the daily administration of one grain of emetin hydrochlorid hypodermically with one-half

⁵³ *Proc. Nat. Acad. Sc.*, 1925, 9: 239.

⁵⁴ *Johns Hopkins Hosp. Bull.*, No. 25, 1914, p. 323.

⁵⁵ *Am. J. Hyg.*, 1925, 5: 371.

⁵⁶ Letter to Boeck.

⁵⁷ *J. Am. M. Ass.*, 1924, 78: 1604.

⁵⁸ *Calif. State J. M.*, 1922, 20: 59.

⁵⁹ C. Dobell and F. W. O'Connor, *The Intestinal Protozoa of Man*, London, 1921.

grain in keratin capsules by mouth for twelve consecutive days. Both of these methods give satisfactory results, although a certain number of relapses occur and then it is well to repeat the course of treatment until a cure is obtained. Cases that do not clear up with emetin should be followed by large doses of bismuth, in accordance with the method advocated by Deeks. The bismuth reaches the amebæ in the lumen of the intestines, while emetin acts mainly upon those in the tissues.

Recently, another drug, stovarsol,⁶⁰ has been advocated for the cure of amebic dysentery. The results thus far are promising and the drug is said to be less irritating than emetin when taken by mouth. A dose of 0.5 to 1 gram is given daily by mouth for five or six consecutive days.

Dysentery should be included in the notifiable diseases and laboratory aid furnished by boards of health to assist diagnosis. Cases should be isolated in the same sense that cases of typhoid are isolated, and disinfection practiced at the bedside. Outbreaks in institutions should always be investigated and vigorous measures taken to check further spread and to prevent recurrences. In all respects, the prevention of bacillary dysentery is a close parallel to that of typhoid.

BALANTIDIAL DYSENTERY

This form of dysentery is caused by *Balantidium coli*, an intestinal ciliated protozoan, with two nuclei, two contractile vacuoles and a funnel-shaped mouth; it forms large cysts. Man in some way contracts this parasite from pigs, in which it is a harmless inhabitant. *Balantidium coli* may also live in man as harmless commensals, but frequently they invade the tissues, producing an ulcerative colitis with marked symptoms of dysentery, when it is a serious and often fatal disease.

Balantidial dysentery has practically a world-wide distribution, affecting especially the Baltic countries; also South and Central Americas, the Philippines and our southern states. It occurs sporadically and never in epidemic form.

The disease selects those who are in close contact with hogs, as slaughterers, gut-strippers, skinnners and swine tenders. The exact method of spread has not been demonstrated, but clearly involves the direct or indirect transfer and ingestion of the infectious material from pigs. This gives the keynote for prevention.

There is no specific prophylaxis or treatment. Success has been reported with enemata containing 0.75 gram of quinin hydrochlorid in 300 c.c. of water.

⁶⁰ *J. Am. M. Ass.*, 1925, 84: 1913.

HOOKWORM DISEASE

(*Uncinariasis or Ancylostomiasis*)

Hookworms affect the health and vitality of millions of people living in tropical and warm climates. In badly infested countries hookworm disease stunts the growth of children, cripples agriculture, handicaps the progress of the race and is a great economic disadvantage. It offers a good opportunity to demonstrate the uses of public health administration, for sure and striking results may be obtained quickly through simple methods. A hookworm campaign has been the beginning of a permanent public health organization in many rural and backward communities.

Theoretically, the prevention of hookworm disease is comparatively simple, for we know the parasite and its life history, its mode of entrance into and exit from the body, and a satisfactory cure for the disease within reach of all. Furthermore, prevention consists in a simple and primitive hygienic requisite: the proper disposal of human feces so as to prevent soil pollution. Practically, however, we have ignorance, apathy, poverty, and uncleanness to deal with before satisfactory prevention, much less eradication, can be achieved. It is now plain that hookworm disease presents a sanitary problem of first magnitude, not alone in our southland, but in practically all tropical and subtropical countries. Further, there is a large economic and sociologic aspect to this question in medical biology.

Hookworm infestation⁶¹ differs from bacterial infections in that the worms do not multiply within the body; they are contracted from time to time, usually over a long period. Each worm in the intestinal tract represents a separate infestation through the skin.

We are indebted to Looss for the discovery of the method of transmission, which revolutionized prevention and control. Interest in this country was aroused through the work and enthusiasm of Stiles.

Prevalence and Distribution.—Hookworm disease encircles the globe in the tropical and subtropical climes; it diminishes toward the temperate regions. It is not endemic in the colder latitudes, except in mines. The infestation belts the earth in a zone about 66° wide, extending from parallel 36 north to parallel 30 south latitude. The incidence is high, ranging from 50 to 95 per cent in all tropical and subtropical countries where conditions, such as rainfall, type of soil and habits of the people, are favorable for hookworm infestation. In 1904 the Porto Rican Anemia Commission found that 90 per cent of the rural population of the island was infested. It is also very prevalent in Samoa, China, India, Egypt, Africa and tropical America.

In 1879 an outbreak of hookworm disease occurred among the laborers in St. Gothard's tunnel and was called miner's anemia. This aroused the

⁶¹ Infestation refers to the larger and more highly organized animal parasites, and does not involve the idea of large numbers as the word is ordinarily used in its non-technical sense. Thus, animals are infested with tapeworms, roundworms, fleas, lice, etc.

interest of the scientific world. The polluted soil of the tunnel was found to be impregnated with the eggs and larvæ. Gunn⁶² has shown that from 50 to 80 per cent of the underground laborers in the California mines harbored hookworms in 1911. It is probable that all the older mines employing foreign laborers sooner or later become endemic foci if the temperature of the mine and the chemical composition of the water are favorable. Many of the mines in Europe were formerly infested, but this problem has been solved to a great degree in Wales, Germany, Belgium and the Netherlands.

Species of Hookworm.—Many mammalian animals have hookworms, but in general each host species has a different kind of hookworm; that is, the hookworms of the dog, sheep, horse, seal, etc., differ from each other, and are specific. The common hookworm of the dog in this country will not infest man; the hookworms of man do not develop to maturity in the lower animals.

Several different hookworms infest man, but only two are of public health importance—the Old World form, *Ancylostoma duodenale*, and the New World form, *Necator americanus*. The distinction between these worms has a zoölogical rather than a practical bearing, for they produce the same symptoms, require the same treatment, have the same life history, and call for the same preventive measures. It seems, however, that *Ancylostoma duodenale* is about five to ten times more pathogenic than *Necator americanus*; that is, infestation with some 100 worms of the former produces symptoms equal to about 500 to 1,000 of the latter.

Ancylostoma duodenale is distributed in all countries lying in Eurasia, south of 35° N. latitude and north of 20° N. latitude, and was introduced into the American continents and the Antilles in historic times from India, the Mediterranean Basin and from China and Japan. Its original endemic focus was probably the Orient. It has one pair of ventral hooks, two conical dorsal teeth, and the dorsal ray of the caudal bursa divides two-thirds its way from the base, and each division has three tips (tripartite). The head of the adult worm is a prolongation of the general curve of the body, whereas the head of *Necator* is bent backward. *Ancylostoma* is also thicker and a little longer than *Necator*.

Necator americanus is found in largest proportions in Eurasia, Africa, Indonesia and Polynesia south of 0° N. latitude. This species was also introduced into America within historic times by Kaffir slaves from Africa. It is the species most commonly found in America and has spread from Virginia to Argentina. It has ventral lips, a dorsal median tooth, one pair of dorsal and one pair of ventral lancets deep in the buccal capsule, and the dorsal ray of the caudal bursa divides at its base and each division has two tips (bipartite). It is less pathogenic than *Ancylostoma*.

Ancylostoma braziliense (*ceylonicum*) is found in the Philippine Islands and the Malay State. It was early described from Ceylon. In Brazil, it

⁶² *J. Am. M. Ass.*, 1911, 56: 259.

infests cats and dogs. Compared to the other two forms, this hookworm has a limited distribution and minor public health importance.

The geographic distribution of the intestinal worms of man is definite enough so that the habitat of an individual may sometimes be determined by the parasites he harbors.

Hookworm Disease.—The first stage of the disease is an irritation of the skin where the larvæ penetrate. This is called “ground-itch” or “maza-morra.” The common site of the primary lesion is the soft skin between the toes.

It takes about two weeks for the larvæ to migrate from the skin to the small intestine. During their travel in the body, symptoms are not produced, except occasionally bronchopneumonia.

The symptoms are proportional to the number and kind of hookworms; also to the nutrition and resistance of the host. The damage done by hookworms depends upon the ability of the host to compensate. This is largely a question of food, occupation and hygienic habits. In this regard, it is like heart disease, which may be well compensated, but there is a small factor of safety.

When the symptoms are well developed, hookworm disease is characterized by anemia, mental and physical weakness. Early and heavy infestations cause a stunting of growth. In severe cases, edema comes on and the anemia is extreme—hemoglobin may be as low as 10 to 15 per cent. There is an eosinophilia, which disappears in the final stages; otherwise, the blood-picture is that of an essential anemia.

Many moderate or even relatively light infestations are associated with constipation, indigestion, headache, dizziness, amenorrhea, or tendency to fatigue, which may or may not be due to the worms.

The worms do not multiply in the body and, therefore, the degree of hookworm infestation is practically proportional to the number of hours of contact with infested soil. Infestations in the United States with *Necator americanus* may be divided as follows:

1. Negative.
2. Very light—1 to 25 worms.
3. Light—25 to 100 worms.
4. Moderate—100 to 500 worms.
5. Heavy—500 to 1,000 worms.
6. Very heavy—1,000 to 3,000 worms.

It is assumed that it takes about one hundred to five hundred worms, *Necator americanus* (or fifty to one hundred *Ancylostoma duodenale*) to check the growth and cause noticeable anemia in growing children. Any less than twenty-five or fifty *Necators* do not produce measurable symptoms and these persons may be regarded as carriers. Symptoms of hookworm disease are rare before the fourth to fifth year, because little children are not often exposed and seldom acquire twenty-five or more worms before the fifth year. In this country, hookworm disease is mainly one of school age. The infestation is

cumulative. It is progressive up to the fourteenth or fifteenth year, and then there is a natural reduction due to the wearing of shoes. In Brazil, the heaviest infestations occur in adult life, since agricultural workers seldom wear shoes.

Modes of Transmission.—The usual mode of transmission is through the skin. The larvæ may also be taken by the mouth in drinking water or on solid food, or may contaminate objects, such as dirty fingers. It has been shown by experiment that animals can be infected by drinking water containing the larvæ. Sources of infection other than infested soil may occasionally occur, but play a minor public health rôle.

The mode of exit is exclusively in the feces, which contains the eggs of the parasite.

The Parasite.—For a correct understanding of the prevention of hookworm disease it is necessary to have a knowledge of the essential features of the life history of the parasite. Hookworms live a double life, one in man and the other in the soil.

Hookworms are round worms (nematodes) belonging to the super-family *Strongyloidea*. The adult worm is about one-half to three-quarters of an inch long, and about the diameter of a wire hairpin.

The adult hookworm lives in the intestinal tract, usually in the small intestine. It attaches itself to the intestinal wall, wounds the mucosa, sucks blood, eats the epithelium, and probably produces a toxic substance which injures the host.

The female worm lays a prodigious number of eggs in a never-ending stream, which pass from the host in the feces. The embryo does not mature within the egg except in the presence of oxygen. Hookworm embryos, therefore, do not undergo full development until the eggs are discharged into the outer world. On the other hand, *Strongyloides stercoralis*, the parasite of Cochin-China diarrhea, develops embryos which may escape from the shell while in the intestinal tract and are passed in the stools. The hookworm embryos may become mature outside the body in six to eight hours in the presence of favorable conditions of moisture, warmth, and oxygen. It is, therefore, necessary to examine the fresh stools in order that this difference between the two infections may be of value in differential diagnosis. In field work, where this is not practical, the eggs of these two worms may be differentiated by simple cultural methods.

Under favorable conditions the hookworm embryo escapes from the egg and becomes a larva in about twenty-four hours. This free-living larva exists in moist soil and feeds upon the organic matter found there. In the course of two days or more it sheds its skin (*ecdysis*) and thus passes to the first molt. The resulting larva continues as a free-living parasite, and in about a week approaches the second molt, after which the sheath may be lost. This is the second ecdysis and this encystated larva no longer takes food. This stage in the life history of the parasite is of special importance for the reason that it is capable of piercing the skin; that is, it is the infecting stage. In this

condition the parasite may continue its free living existence for five to twelve months, perhaps longer.

Hookworms are sensitive to their environment, especially to temperature, moisture and character of the soil. The worm spends part of its life cycle on the surface, and if the soil is clay or silt, as in parts of Alabama, the hookworm problem is reduced. Most of Texas is too dry and much of Virginia too cold. The larvæ do not develop if the temperature reaches 40° F. or less. They are checked at 60° F.; the optimum temperature is about 85° F.

The infective larvæ live on or near the surface of the soil, where they await a host. They do not move laterally to any appreciable extent and, therefore, the area of soil infestation does not spread.



FIG. 9.—HOOKWORM LARVA
EXTENDING ITSELF IN
SEARCHING POSITION FROM
SOIL PARTICLE.

(From Payne, *Am. J. Hyg.*,
1923, 3: 547.)

The hookworm larva passes in all through four ecdyses or molts. Two of them occur during its free-living stage and two of them during its residence in the host. With each ecdysis the larva approaches more nearly the appearance and structure of the adult worm.

Mode of Entrance.—The larva pierces the unbroken skin and passes by a circuitous route to the intestinal tract. The parasite may enter the skin at any place, but it usually goes through the soft and thin skin between the toes. In its passage through the skin the larva produces an inflammatory reaction (ground-itch) which results partly from the irritating action of the presence of the foreign body, but mainly from

the bacteria carried along with the larva. These primary lesions may consist of a few itching papules or pustules to a severe dermatitis. Of 4,741 patients questioned by Ashford, King, and Guitierrez in Porto Rico, 4,664, or about 98 per cent, gave a history of ground-itch, which is now recognized as the first stage of the disease.

The fact that the infestation with hookworm disease is usually contracted through the skin was discovered by Looss in Cairo, Egypt. He also unraveled the course of the parasite from the skin to the intestine. This brilliant discovery, which is one of the romances of medical biology, is the foundation upon which prevention against the infection depends. In 1895, Looss accidentally spilled a drop of water containing many encysted larvæ upon his hand, and noted that they disappeared, leaving their delicate sheaths behind them. Seventy-one days subsequently he developed intestinal uncinariasis. The experiment was then repeated upon a volunteer, and hookworm eggs appeared in his stools in seventy-four days. Claude Smith found eggs in the feces six and one-half weeks and seven weeks after experimental skin infection on two persons with the American parasite (*Necator americanus*).

Route of Travel from the Skin to the Intestines.—The wanderings of the parasite from the skin to the intestine were worked out by Looss partly

by placing larvæ upon an amputated leg and also by studying the question upon puppies. The hookworm larva pierces the skin, usually between the toes, enters the subcutaneous tissue, and then finds its way through the lymphatics to the neighboring lymph-nodes. The larvæ are able to squirm through the lymph-nodes, pass to the thoracic duct, and thence to the vena cava and the right heart. From the right heart they are carried in the blood stream to the lungs. The larvæ are too large to pass the capillaries of the lungs. They pierce the capillary walls and appear in the alveoli and are now, to all intents and purposes, again in the outer world. They pass up the bronchi and trachea to the throat, whence they are swallowed, and finally lodge in the small intestines. During their travel through the body they pass through two ecdyses. Perhaps half of those that penetrate the skin finally reach the intestines; many get lost or die.

The adult worm attaches itself to the mucous membrane by means of the powerful buccal lancet. The epithelium is drawn into the buccal cavity as though by a powerful suction. The worms are usually found in the small intestine, especially in the jejunum, less often in the duodenum, and rarely in the ileum and lower reaches of the intestinal tract; they are very rarely met with in the stomach.

The parasites imbibe large amounts of blood, some of which passes through the worm unaltered. The wound continues to bleed after the worm releases its hold, owing perhaps to a hemolytic substance in the mouth parts of the parasite. The worm does not remain fastened to one place indefinitely, but releases its hold and attaches itself anew. This produces numerous minute wounds, favoring secondary infections. The hookworm probably produces a poison which is absorbed and which accounts, in part, for the anemia and other symptoms of the disease. The severity of the symptoms bears a definite relation to the number of worms and the resistance of the host. A few hookworms rarely produce clinical evidence of their presence. Smillie believes that an average infestation of twenty-five to fifty *Necators* has no measurable effect on the normal growth in height and weight or the vital capacity or hemoglobin of rural school children in the southern states. He regards these light infestations as carriers rather than true cases of hookworm disease. These so-called carriers, however, need treatment for themselves as well as to prevent the spread of the infestation.

The number varies greatly in individual cases; from one or two to thousands. Sandwith counted 250 worms and 575 bites in one case; 2,000 worms are not an uncommon number. The Porto Rican Anemia Commission counted as many as 4,600 passed by one individual.

Diagnosis.—Diagnosis depends upon finding the eggs in the feces. The plain smear method is the most direct and simple method of examination, but unfortunately light infestations are missed in half the cases. The salt flotation method is used extensively in hookworm campaigns. With this method a sample of the feces to be examined is thoroughly mixed in saturated sodium chlorid solution and filled up to the brim of the container ($\frac{1}{4}$ ounce tin

ointment jar). A two by three inch glass slide is then placed over the container in contact with the surface of the salt solution. This is left to stand for about fifteen minutes to give the eggs time to rise to the top. The slide is then carefully removed, turned over, and examined under the microscope. This method is accurate in diagnosing light infestations, but does not give a measure of its severity. The severity of hookworm infestation in the individual or groups may be closely approximated by an egg counting method devised by Stoll. The steps in this technic are as follows: (1) Weigh three grams of feces into a large test-tube, add N/10 NaOH up to 45 c.c. level and a few glass beads. Close tube with rubber stopper and shake until a homogeneous suspension has been obtained. Immediately transfer 0.15 c.c. to a two by three inch slide, cover with twenty-two by forty millimeter cover-slip and count total number of eggs in the preparation, using a mechanical stage. The number of eggs times one hundred equals the number of eggs per gram of feces. The severity of the infestation is then expressed either in the number of ova per gram feces or reduced by calculation to the approximate number of worms harbored.

Immunity.—There is no acquired or transmitted immunity to hookworm disease. Apparently all races are alike susceptible. Payne believes the Negro has a natural immunity to *Necator*. It is commonly supposed that the dark-skinned tropical races through long association with the parasites have acquired a resistance. It was formerly assumed that the Negro and the Filipino have an immunity, because these races, while often infested, have comparatively slight symptoms. In this country, the Negro suffers less than the white living under similar conditions, and probably with more exposure, not because he has an immunity, but because he has fewer worms. The reason for this lighter infestation is assumed to be due to the thickness of the skin, which may make it more difficult for the worms to penetrate. The relative insusceptibility of certain races also finds an explanation in the fact that they are infested with different hookworms which vary in pathogenicity.

Hookworm disease lowers resistance and greatly increases the effects of other infections, especially tuberculosis. The secondary results are often more disastrous than the primary effects.

Resistance of the Parasite.—The adult worm in the intestinal tract may be benumbed or killed with oil of chenopodium, thymol, betanaphthol, carbon tetrachlorid and other vermifuges. From the standpoint of prevention, it is of practical importance to know the resistance of the free-living stages of the worm. Fortunately, the larvæ are readily killed by drying and freezing. Unsheathed larvæ are less resistant than sheathed larvæ and are the first to succumb to dryness. Unsheathing is a continual process among a given number of larvæ, which accounts for their early death under natural conditions. The usual span of life of mature larvæ under tropical conditions is limited to a few weeks. This holds true for the southern United States. Under favorable conditions, that is, deep shade, constant moisture and favorable soil, a very few may persist for from two to four months. Rapid drying of the upper

layers of the soil is very disastrous to the larvæ. They have been kept alive in water in laboratory experiments in temperate climates for eighteen months. Infective larvæ of *Ancylostoma duodenale* are much more resistant than those of *Necator americanus*.

The fact that freezing kills the eggs and larvæ largely explains why the disease is not endemic in this country north of the Potomac.

It has been shown that chlorinated lime fails to kill hookworm eggs in twenty-two to forty hours. Schüffner kept the larvæ alive almost four months in water with two or three drops of a one per cent quinin solution to 10 c.c. Oliver found that sea water killed the larvæ in thirty-seven minutes.

The adult worms live about five years in the intestines, so that when new infestations are prevented there is a rather rapid decline in the intensity of infections, for most of the worms are quite old. Thus, in our south the habit is to wear shoes about the sixteenth year, and there is a sudden falling off in intensity after this age.

Factors in Dissemination.—There is no active migration of the larvæ; they are definitely limited to the spots where the feces are deposited. The larvæ may, however, be scattered about by outside agencies. The means of mechanical transportation are: (1) domestic animals, (2) feet of man passing through infested centers, and (3) washings from heavy rains.

1. The majority of hookworm eggs passing through the alimentary canal of the chicken fail to produce mature larvæ, while they apparently pass unharmed through the digestive tract of the pig. Chickens may be considered a factor in hookworm control.

2. Passive transfer on shoes of individuals passing through infested centers and deposited on paths frequented by others. In one instance, 144 hookworm larvæ were isolated from soil taken from the sole of the shoe of a man passing through an area of gross soil pollution. Mechanical transfer on food and other objects may occasionally take place.

3. When washed by rains, the larvæ are usually carried away into streams, etc. While this is a possible source of danger, it plays a minor rôle and may be disregarded in a hookworm campaign, except perhaps in some mines.

Prevention.—The prevention of hookworm disease is based on three principles: (1) sanitation: the prevention of soil pollution; (2) treatment of cases so as to diminish the amount of infestation; (3) mechanical protection as the wearing of shoes. Sanitation is fundamental and enduring, but slow; treatment is quick, but temporary; shoes are effective, but not always economically practicable.

The principles of prevention are easy in theory, but their application is difficult in practice on account of the widespread and enormous amount of the disease. The suppression of hookworm disease means the social and economic uplift of nations, the education of millions of people, and an entire change in their daily hygienic habits. Education of the masses is an important factor, calling for coöperation between the health authorities, civic

forces, the medical profession, schools, and philanthropic agencies; it is something for the preacher and teacher.

Soil Pollution.—The prevention of soil pollution is the essential factor; it is the key to the situation. This one line of prevention could blot hookworm disease out of existence. This requires not only the building of proper privies, but insisting upon their being used in country districts. Payne showed that burial of feces is not effective. Larvæ will migrate up through four feet or more of soil, but do not migrate laterally. Hookworm disease is not a city disease. In warm countries direct pollution of the soil is much more common and also much more dangerous than in cold countries. Add to this the custom of going barefooted, and we have all the factors necessary for the dissemination of hookworm infection.

Stiles estimated that ten years ago about 50 per cent of the rural homes in the south were without privies and that 2,000,000 persons were infested in our southland. Even many schools and churches did not have these accommodations and were, therefore, hotbeds of infestation. The conditions are now rapidly improving. For the care and disposal of night soil, see chapter on Sewage.

Types of Soil Favoring Hookworms.—Stiles first noted that persons living in sandy soils had much heavier infestations than those living on rock, clay or other soils with hard, dry surfaces. This is due to physical conditions, namely, the size of the soil particles. Clay and dense compact soils are very unfavorable for larvæ; coarse soils, as sandy or sandy loam soils, are very favorable. Augustine made a soil and hookworm survey of Alabama, and showed that where there was a heavy compact clay soil, there was no hookworm disease. It is possible for a sanitarian to study a soil map of an area and determine the zones of potential danger.

Method for Determining Degree of Soil Pollution.—It is of value in hookworm control campaigns to determine the sources and degree of soil infestation. This can easily be accomplished by examining samples of soil from polluted places with an apparatus originally devised by Baermann. This apparatus successfully isolates hookworm larvæ from infested soil. It consists of a brass sieve 6 inches in diameter, 3 inches high with 1 mm. mesh, lined with coarse muslin cloth and a glass funnel 9 inches in diameter. A tightly-fitting rubber tube, closed with a Hoffman clamp, is placed on the stem of the funnel. The sample of soil is put in the lined sieve and placed in the funnel, which is supported upright in a padded rack. Hot water is poured between the outer edge of the sieve and the funnel up to one-half an inch of the bottom of the sieve. When the water becomes luke warm, more luke warm water is added until the soil in the sieve is covered by it. Fifteen hours later the Hoffman clamp is slightly opened and the water run out until a 50 c.c. centrifuge tube is filled. After centrifugation the supernatant water is drawn off with a long pipette and the residue examined microscopically for infective hookworm larvæ.

Hookworm larvæ must not be confused with larvæ of free living nema-

todes, which are normal inhabitants of the soil. This difficulty can easily be overcome by a study of the structure, movements, etc., of hookworm larvæ before taking up work requiring identification.

Treatment and Eradication of the Infestation in Man.—Hookworms may be expelled from the intestinal tract by the use of thymol, oil of chenopodium, betanaphthol, or certain other anthelmintics. The control of the infection through the treatment of infested persons (carriers and cases) is an essential factor in prevention. It is not feasible to eradicate hookworm by treatment alone. A hookworm campaign based on treatment without sanitation is disappointing, for in a few years the plight will return. In a hookworm campaign in an endemic area, medical attention is given to the heavy infestations; the less severe infestations belong to private practice rather than to public health work. Complete eradication among the light cases is obtained only through sanitation. One or two treatments will usually bring away about 95 per cent of the hookworms. Complete elimination can be obtained only after repeated treatments. The few remaining worms may not appreciably harm the host, and the danger to the community is neutralized by the prevention of soil pollution. As a public health measure, repeated treatments to expel the last hookworm is not economically a wise administrative policy, and the course to be followed in private practice depends upon circumstances.

The ideal vermifuge is one which would remove all intestinal parasites without toxicity to the host, but this vermifuge has not yet been discovered. All vermifuges have some toxic action and when administered on a large scale, some few cases of idiosyncrasy to the drug are sure to be encountered.

Thymol.—Thymol was the first drug employed on a large scale in the treatment of hookworm disease. It has now been almost entirely abandoned, because it is less effective and much more toxic than some of the other vermifuges. The adult dose is 2 grams repeated in two hours, and preceded and followed by a saline purge.

Betanaphthol is a very efficient drug in the treatment of hookworm disease, but cannot be employed where malaria is endemic, for it may produce paroxysmal hemoglobinuria in individuals infected with malaria. It is given in 1 to 1½ gram doses each day for three days without dietary régime and without purgation, or a single dose of 3 grams mixed with 25 grams magnesium carbonate in water.

Oil of Chenopodium.—The active principle of oil of chenopodium is ascari-dol. Good preparations contain at least 70 per cent of the active principle, and the pure drug itself is now obtainable. It is a powerful anthelmintic,⁶³ but is sometimes quite toxic for small children. The oil will not be absorbed unless emulsified by the bile, and thus the chief aim in treatment is rapid elimination. Its toxic action is limited almost exclusively to the central nervous system, and first signs of poisoning are dizziness, headache, tingling of the fingers and ringing in the ears.

⁶³ It acts especially on the male hookworms and also brings away ascaris.

The drug should be given in one dose on an empty stomach in the early morning. No preliminary purge is necessary. For adults the dose of oil is 1.5 c.c. If pure ascaridol is used, 1 c.c. is sufficient. One hour after the drug is given, one ounce of magnesium sulphate should be administered in a full glass of water. The treatment should not be repeated until two weeks have elapsed. One treatment is sufficient to reduce a case of moderate infection to the carrier state. Two treatments may be necessary in severe cases.

Carbon Tetrachlorid.—Carbon tetrachlorid is by far the best single vermifuge for the treatment of hookworm disease. It is less toxic in effective doses than the other vermifuges. Its toxic action resembles chloroform and the chief lesion is the production of a central necrosis of the liver. It is extremely toxic for alcoholics, even in small doses. (One c.c. has produced death when taken with alcohol.) Lambert also believes that cases with heavy ascaris infection are subject to intoxication by the drug.

The effective adult dose is 3 c.c. on an empty stomach in the early morning. It may be divided into two doses—1.5 c.c. at 6 A.M., 1.5 c.c. at 7 A.M. and a purge of magnesium sulphate at 8 A.M. One treatment will remove 95 per cent of all hookworms harbored. It acts especially on the female hookworms and also oxyuris.

Mixed Treatment: Carbon Tetrachlorid and Oil of Chenopodium.—The most effective and least toxic method of hookworm treatment is the method devised by Smillie and Pessoa of administering carbon tetrachlorid with oil of chenopodium. Neither drug increases the toxicity of the other, while their specific actions when given in small combined doses are complementary. Thus, greatly diminished doses of the two drugs may be administered without decreasing the efficiency of the treatment.

Subjects to be treated are advised to take a mild purgative the afternoon previous to treatment, eat but a light supper the night before and on the morning of the designated day to take no food. The treatment is begun early, usually about 7 A.M. Carbon tetrachlorid and ascaridol are administered in the proportion of 2 to 1, the total dosage in tenths of a c.c. being equal to the age of the child in years; thus, a child of ten years would get 1 c.c. A maximum total dose of 1.5 c.c. (1 c.c. carbon tetrachlorid and 0.5 c.c. ascaridol) is given at fifteen years or over. The carbon tetrachlorid is given in a hard gelatin capsule at 7 A.M.; the ascaridol in the same manner at 8 A.M.; and a large dose of magnesium sulphate at 9 A.M. If the salts do not give desired results within an hour, a second dose is given. Ninety to 95 per cent of all hookworms harbored will be removed by one treatment with a minimum of danger to the host.

Plan of a Hookworm Campaign.—The principles of a successful hookworm campaign depend upon the recognition of the fact that there are cases and carriers; and that both need treatment as well as sanitation and education. The first rule is to employ treatment to control hookworm disease, then education and sanitation to control and eliminate the carrier hazard.

The first step in a hookworm campaign should be to study the general

conditions of the area. An experienced sanitarian may often determine by a brief survey whether the district is heavily or lightly infested. Much additional information concerning regional distribution within the area may be learned from monthly record of maximum and minimum temperatures, the rainfall, the type of soil, the sanitary habits of the people, and their economic status. A preliminary survey may be made of 5 to 10 per cent of the population to determine the intensity of infection of various age, sex, racial and occupational groups. Hookworm is not a city disease; it is to be looked for in the country, especially among rural school children.

If the intensity of infection of the area is not severe enough to produce evident economic injury to the people (as in Australia), the slow but sure methods of education and sanitary installations are the essential means to be employed. If a certain age or occupational group is chiefly affected, as, for example, in certain parts of the southern United States, where mainly the rural children of school age have true hookworm disease, the hookworm campaign may start by treating the affected group only to the point of economic cure. This procedure relieves the burden of the disease upon the community until education and sanitation will have had an opportunity to become effective and permanent.

Based on the above principles, the following plans of a hookworm campaign have been tried and need consideration:

1. *The traveling dispensary method*, patterned after the Porto Rican Anemia Commission, was first tried. The dispensary moves from place to place, treating all who apply. The results have been disappointing, because many people who need the treatment do not come to the dispensary, the system is expensive and, unless followed up by sanitation, the results are temporary, reinfestation occurring within a few years. The method is still an admirable one for the purposes of demonstration and education.

2. *Privy construction* is radical and permanent, but deliberate and expensive. Even where sanitary privies are built, they will not serve their purpose unless they are used. It is not easy to change the habits of people in this regard. Much better results are obtained if the privies are built by the people than for the people.

3. *Intensive Campaign: House-to-House Survey*.—A nurse goes from house to house throughout the area obtaining specimens of feces. All positives are treated until relieved. After this comes education and the building of privies. This method requires a doctor, a nurse, a microscopist and assistants. It is expensive and time consuming, but radical and enduring, and suitable for intelligent communities lightly infested and economically competent.

4. The method of *mass treatment* was designed by Darling and carried out by Hackett on a large scale in Brazil. It is based upon the difference between hookworm infestation and hookworm disease. It requires a preliminary survey to determine the percentage of infestation and a knowledge of the population. In certain areas in the tropics, a preliminary survey will

show that the whole rural population (85 to 90 per cent) is infested with hookworms, most of them heavily. Here, mass treatment is employed. Microscopic diagnosis of each case is omitted; but every person in the area over four years and under sixty years of age is given two complete hookworm treatments. This brings away about 95 per cent of the worms.

Mass treatment is repeated every two to three years not with the expectation of reducing the incidence of infection, but with the intention of relieving the people of the severe health drain and economic burden of the disease. In so far as possible, installation of sanitary toilets should follow, or better still, immediately precede the treatment campaign. Where treatment only is employed and no change occurs in the sanitary habits of the people, it has been shown that the intensity of infection will return to its original level in five to ten years, irrespective of how thoroughly treatment has been employed. Mass treatment was used with success in Brazil by Hackett without diagnosis, because 98 per cent of the rural population there is infested. The plan is useful to accomplish immediate relief at a minimum cost in heavily infested and backward communities.

Education.—Education is one of the most important factors in eradicating hookworm disease, for the reason that its final control depends upon improvements in the sanitary habits of the people, especially in the rural districts. To change the daily habits of half a nation is an uplift that requires time and patience. It is perhaps best to begin with the school children; even then it will take a generation for results. Very little can be accomplished by force, and, if the customs and prejudices of the people are ignored, the reformer and benefactor meet with rebuff and failure. It is a good idea to have a public health day or a public health week in the schools, during which time lectures and educational work upon hookworm, typhoid, tuberculosis, and other prevalent infections are considered. The children carry the lesson into the home. Portable posters, lectures, exhibits, and popular articles in the magazines and newspapers all contribute their share. The moving picture is one of the best mediums. Pamphlets have been almost entirely abandoned, for many of the people affected cannot read. The medical profession in the infected areas should instruct and lead to awaken interest in the problem.

Personal Prophylaxis.—Personal prophylaxis consists in wearing shoes and otherwise avoiding contact with the infested soil. Miners, brick makers and others compelled to work in infested soil should wear shoes and may wear gloves, although the infecting larvæ will pass through ordinary gloves. Other measures, such as drinking only clean water, eating only clean food, washing the hands and avoiding infested areas, are obvious. After all, the prevention of hookworm disease is a question of decency and cleanliness. With cleanly habits there would be no soil pollution, and the disease would be checked.

Immigration.—Hookworms are carried from country to country by immigrants. This is not an important factor in the spread of the disease in the United States. Every country that brings laborers from hookworm regions is bringing in a constant stream of infection. California was the first to

establish measures with respect to Indian coolies, 90 per cent of whom are infested; fecal examinations and restrictions were later extended against all suspected immigrants at all ports.

Collateral Benefits.—The best part of a hookworm campaign is the collateral good it does. This applies as well to a sanitary campaign directed against almost any disease. The suppression of hookworm disease will diminish the amount of tuberculosis, typhoid fever, dysentery, and other infections. Thus, in Bilibid prison, Manila, the death rate was formerly excessive—234 per thousand when the Americans took charge. This was reduced to seventy-five per thousand by sanitary measures, such as boiled water, screens, disinfection, improved food, less crowding, better air, more sunlight, etc., but despite these sanitary improvements the death rate could not be hammered down below seventy-five per thousand. Then it was found that many of the prisoners were infested with hookworms. Thymol was administered and the death rate fell to 13.5 per thousand. Schapiro⁶⁴ found that treating hookworm disease on plantations in Costa Rica caused an increase in earning capacity and in acreage cultivated. On one farm, the laborers earned 27 per cent more, and on another 14.6 per cent more. Another farm cultivated 33 per cent more coffee with the same number of laborers, at a lower unit cost. He also noted a reduction in morbidity and infant mortality.

Another instance of the collateral benefits resulting from sanitary work is the plague campaign in San Francisco, which cut typhoid fever in half, although no special attention whatever was paid to the latter disease. The purification of the water supply in Hamburg by filtration cut down the general death rate and diminished the morbidity of diseases not water-borne. One of the most encouraging phases of sanitary work directed against tuberculosis, typhoid fever, and hookworm disease is the assurance that a successful campaign will result in fundamental and permanent control or eradication of other communicable diseases. The prevention of tuberculosis deals especially with personal hygiene, and the prevention of typhoid fever and hookworm with the sanitation of the environment. The combination of the two, therefore, embraces almost the entire range of preventive medicine.

A hookworm campaign is an entering wedge for permanent public health work in backward countries and especially in rural districts. Many full time county health officers and satisfactory administrative units in this country had their origin with a hookworm campaign.

COLLATERAL READING

Bibliography of Hookworm Disease, Publication No. 11, International Health Board of the Rockefeller Foundation, 1922, contains an exhaustive bibliography of practically everything published on the subject up to 1922 and also an admirable history.

"Investigations on the Control of Hookworm Disease," Studies 1-24, Cort *et al.*, *Am. J. Hyg.*, 1921, 1922, 1923, contain important contributions.

⁶⁴*J. Am. M. Ass.*, 1919, 78: 1507.

For discussion of sanitary privies, see special bulletins on this subject by the United States Public Health Service, and by many, especially the southern state boards of health, particularly *Health Bulletin, N. C. State Board of Health*, 1919, 5:34.

CHAPTER III

DISEASES SPREAD LARGELY THROUGH DISCHARGES FROM THE MOUTH AND NOSE

This group of diseases is the most prevalent and damaging of the infections to which flesh is heir. The respiratory diseases prevail more especially in temperate, cool and variable climates, but occur also in warm latitudes and even in the tropics. They are endemic everywhere, epidemics are frequent, and pandemics sweep the world like a devastating plague about once a generation. As a group, the respiratory infections are less well understood and hence less controllable than the intestinal diseases. These diseases are spread chiefly through the discharges from the mouth and nose, and many times by direct personal contact with cases or carriers; sometimes by droplet infection or indirectly. They may in fact be transmitted in many other ways; thus, infection may be contracted in food and drink, by hand to mouth infection, or by fomites, such as cups, spoons and other things that are mouthed. Infection taken into the mouth and nose does not necessarily cause a respiratory disease, as, for example, cerebrospinal fever, also measles, small-pox, etc.

TUBERCULOSIS

Tuberculosis is the most frequent and widespread of all the major infections. It is a disease of cattle in barns, not on the range; chickens in coops, not birds in nature; monkeys in zoos, not in the jungle; man in houses, not primitive races. In this country 9 per cent of all deaths, and in Germany 12 per cent, are caused by tuberculosis. The toll falls heaviest during the period of life of greatest usefulness—thus 30 per cent of all deaths between the years of fifteen and sixty are due to pulmonary tuberculosis alone. Naegeli, from a careful examination of a large number of bodies in Zurich, found evidence of tuberculosis in over 90 per cent. Todd¹ found tuberculous lesions in 69 per cent of 386 persons who died from disease other than tuberculosis. The lowest figures based on the evidence of pathologic anatomy are those of Beitzke, who examined 1,100 bodies in Berlin. In children under fifteen he found evidence in 27.3 per cent, and in persons over fifteen, 58.2 per cent. The difference between Naegeli's figures and Beitzke's is due to a difference in the interpretation of the pulmonary scars and adhesions at the apices, and the small fibrous nodules in the lungs. Beitzke does not consider such lesions as of tuberculous origin, and leaves them out of his figures. If these were included, his per-

¹ *Edinb. M. J.*, 1926, 33: 303.

centage would also be very much higher. The frequency with which we become tuberculized is indicated by the fact that 70 per cent of persons over fifteen years old give the Pirquet tuberculin cutaneous reaction. "Everyone has a little tuberculosis."

In the United States over 100,000 persons die each year of tuberculosis.² Of the 110,000,000 people now living in this country, it is estimated that 9,000,000 are doomed to die of tuberculosis, unless the disease is checked. The loss in life and treasure is appalling. It costs the United States alone about \$500,000,000 annually. It is, therefore, most encouraging that preventive measures based upon modern conceptions of the disease as a communicable infection are beginning to give promising results.

The number of cases of clinical tuberculosis in a community may be estimated, according to Philip of Edinburgh, by multiplying the number of deaths from tuberculosis at a given time by twenty. More conservative estimates in this country use ten as a factor. Thus in 1922 there were 3,732 deaths from tuberculosis in Massachusetts, which would mean nearly 40,000 cases in the state during that year. About one-fifth of these are extrapulmonary, and about one-fifth, or 8,000, need hospital or sanitarium care. Therefore, the number of deaths from tuberculosis multiplied by two gives an approximate estimate of the number of beds necessary to provide for the open cases. In the Framingham demonstration, Armstrong found 1 per cent of the population had active tuberculosis, and 2.15 per cent had active or arrested tuberculosis. From this it is estimated that in the United States at large the ratio of the known cases to deaths would be about seven to one for active tuberculosis and fifteen to one for active and arrested cases. In Cleveland, Ohio, 4.3 active cases were registered with the health department per death from tuberculosis.

Tuberculosis began to decline before the nature of the infection was known.³ The decline has been gradual. It was interrupted by the World War and the malnutrition following the war in Europe. Modern methods have so far made little apparent impression upon the gross amount of the infection. The social and economic conditions of the mass of the population must be improved before any great decline in the mortality rate can be expected, as will presently be pointed out.

Tuberculosis is fast becoming, in fact already is, a class disease; it is more prevalent among the poor than the well-to-do. Hence the prevention of tuberculosis has become a sociologic problem. Poverty with its attendant hardships—poor food, bad housing, crowding, overwork and worry—diminishes resistance to the disease; while prosperity, which buys good food, rest, change of air and scene, choice of occupation, and diversion, increases our resistance

²The death-rate from all forms of tuberculosis in the registration area of the United States in 1917 was 146.4 per 100,000, and from tuberculosis of the lungs 128.9. In 1922, the rate for all forms of tuberculosis fell to 97.

³Villamin in 1865 demonstrated by inoculation of rabbits that tuberculosis is an infectious disease; proven by Koch in 1882.

to the disease, and avoids contact with the infection. An increase of wage or decrease in the cost of living; improvement in food and diet; shortening the hours of work; improving the conditions of industrial hygiene; adding to the number of holidays; playgrounds, parks, and wholesome recreation, all help to increase our resistance against and diminish the prevalence of tuberculosis. Science has shown the way; it remains for society to apply the knowledge. "Social justice" is part of the program of preventive medicine.

A distinction should be drawn between *tuberculous*, which means a process due to the tubercle bacillus, and *tubercular*, which means tuberculous-like. The student should also keep in mind the difference between tuberculous *infection* and tuberculous *disease*, for only a small proportion of those infected develop clinical tuberculosis. If tuberculosis were an acute disease with a short period of incubation, its contagious nature would be as apparent as diphtheria or scarlet fever.

The Difference between the Human and the Bovine Tubercle Bacilli.—

There are at least three kinds of tubercle bacilli: human, bovine, and avian. The human and bovine varieties resemble each other closely; the essential difference lies in the fact that the human type is very pathogenic for man, but has little pathogenicity for cattle, rabbits, monkeys, and other animals. On the other hand, the bovine type is very pathogenic for almost all mammalian animals⁴ except man; it is pathogenic for man, but less so than the human bacillus. Even when large numbers of the human bacilli are injected into a calf, a general disease does not usually result; at most only a local lesion is produced. The critical test used in almost all laboratories is upon rabbits. When 0.01 milligram of a bovine culture is injected intravenously, or 10 milligrams subcutaneously, into a full-grown rabbit, generalized tuberculosis results in about six weeks; whereas ten to one hundred times these amounts of a human strain produce at most a slight localized tuberculosis. The culture must be young, that is, about three weeks old; it should be taken from solid media and weighed while moist.

The human bacillus grows luxuriantly upon culture media, covering the entire surface of the medium with a rich, dry, crinkled, mold-like vegetation. The growth of the bovine bacillus upon artificial culture media is more sparse, thinner, less extensive, and somewhat slower. According to Theobald Smith, who in 1898 pointed out the differences between these two types, the human bacillus produces in artificial culture media a different reaction curve from that produced by the bovine bacillus.

Morphologically the bovine bacillus is often shorter, plumper, and stains more uniformly than the human bacillus, which is ordinarily club-shaped, irregular, and stains with interrupted markings. The morphological and tinctorial characters are not sufficiently distinctive to distinguish one type from the other. It has been found that considerable variability exists in strains of both the human and bovine types and some cultures are quite indeterminate.

⁴ Goats, horses, dogs and cats are quite resistant and seldom have tuberculosis.

There are no specific differences between the tuberculins of bovine and human origin.

The **avian tubercle bacillus** is found most frequently in chickens and also in pigeons, pheasants, and guinea-fowl. Geese and ducks appear immune. The avian bacillus is quite pleomorphic and stains somewhat more readily than either the human or bovine types. The avian bacillus grows luxuriantly upon artificial culture media at 45° C., which corresponds to the high temperature of birds, and even multiplies at temperatures as high as 50° C., which is in marked contrast to the mammalian types, which do not vegetate above 40° C. The avian bacillus grows rapidly, so that upon glycerin-agar or upon blood serum there is an abundant growth in ten days, which consists of a white, moist, and greasy mass quite different in young cultures from the dried and crinkled appearance of the human type. Guinea-pigs show a decided resistance to the avian cultures, but rabbits are susceptible, although characteristic lesions do not develop. Chickens and pigeons may be infected with certainty by feeding, and it is probable that in nature avian tuberculosis is generally transmitted in this way.

Fish Tuberculosis.—Acid-fast bacilli are found in cold-blooded animals. They grow best at room temperature (20° to 30° C.), and are inhibited at blood heat. They are not pathogenic for warm-blooded animals. Some of these cultures are called *fish tubercle bacilli*, but their relationship to tuberculosis is doubtful. Friedman's vaccine, exploited as a cure for tuberculosis, was made from one of these organisms obtained from a turtle.

Many acid-fast bacilli are widely distributed in nature and are frequently found in milk, on grass, in dung, and upon the skin. These strains have little or no pathogenicity.

Bovine Tuberculosis in Man.—Concerning bovine tuberculosis in man, we now possess definite knowledge which permits of precise statements. At one time the danger of bovine tuberculosis to man was greatly exaggerated. Koch went too far on the other side when he announced at London before the International Congress on Tuberculosis in 1901 that there was practically no danger of man contracting tuberculosis from cattle. Later Koch modified this dictum, for it was soon proved that the bovine bacillus has a certain amount of pathogenic power for man and that some of the tuberculosis in man is contracted from bovine sources. About 25 per cent of all tuberculosis in children under five years is associated with the bovine bacillus. Fatal bovine tuberculosis is rare after the fifth year. The bovine bacillus is responsible for about 0.5 per cent of all deaths due to tuberculosis.

Pulmonary tuberculosis in man is practically never associated with the bovine bacillus. Bovine tuberculosis in man is usually a disease of the lymph glands or bones—the lymph-nodes of the cervical region and the lymph-nodes in the abdomen being especially attacked. This is due to the fact that the portal of entry of the bovine bacillus is usually through the tonsils or the small intestines. Bovine tuberculosis may become a fatal infection in man when it is generalized through the blood in the form of acute miliary tubercu-

losis or when it localizes in the meninges or other vital parts. Tuberculous meningitis and tabes mesenterica, or the abdominal form, are particularly fatal. About one-quarter to one-third of all cases of tuberculosis in children under five years of age is associated with the bovine type but only about four per cent of tuberculosis deaths under five years are due to bovine tuberculosis. It is probable that all these cases derive their infection through the ingestion of tubercle bacilli in cow's milk. There is little danger from meat, as it is usually cooked and tuberculosis of the muscles is very rare. Tabes mesenterica is now seldom seen since the more general pasteurization of milk and improvement in dairy methods. The following table ⁵ shows the relation between bovine and human tuberculosis in 2,527 cases:

These cases include all that were published up to 1917, namely:

1511 cases collected by Park and Krumwiede (1912), which also include those of the British Royal Commission (1907-11), and those of Bulloch (1910)

356 cases of Eastwood and Fred Griffith (1914), (1916)

430 cases of Stanley Griffith (1914), (1915-17), (1916)

70 cases of Fraser (1912)

72 cases of Mitchell (1914)

88 cases of Wang (1917)

2527 total cases

COMBINED TABLE OF ALL CASES

CLASSIFICATION	ADULTS 16 YEARS AND OVER		CHILDREN 5 TO 16 YEARS		CHILDREN UNDER 5 YEARS	
	Human	Bovine	Human	Bovine	Human	Bovine
Pulmonary tuberculosis, including sputum	1000 *	5	28	...	45	1
Abdominal tuberculosis	24	7 †	13	17	29	34
Generalized tuberculosis	39	2	32	3	169	22 ‡
Tuberculous meningitis	6	..	13	5	55	10 ‡
Tuberculosis, genito-urinary	35	4	4
Tuberculous skin	12	3	4	6	2	...
Tuberculous cervical adenitis	62 §	10	61 §	76	18	75
Tuberculous axillary adenitis	6	..	8	...	4	...
Tuberculous bones and joints	82	4	255 ¶	61	89 **	54 ††
Latent tuberculosis	2	1	2	2	4	1
Miscellaneous (other forms)	5	2	...	1	...	2
	1273	38	420	171	415	199
Percentage of bovine infection at each age period	2.9		28.9		32.4	
Mixed strains reported by Park and Krumwiede	Total2,516 11					
	Grand total...2,527					

* Including 4 atypical strains.

† Including 1 intermediate strain.

‡ Including 1 mixed strain.

§ Including 1 atypical strain.

|| Including 3 atypical strains.

¶ Including 10 atypical strains.

** Including 2 atypical strains.

†† Including 3 mixed strains.

⁵ *J. Path. & Bacteriol.*, 1917, 21: 131.

From a study of 1,038 of these cases we find:

16 years and over.....686 cases, 9 with bovine bacilli= 1.3 per cent

Between 5 and 16 years..132 cases, 33 with bovine bacilli=25.0 per cent

Under 5 years.....220 cases, 59 with bovine bacilli=26.8 per cent

Many of the cases included in the above total were selected cases. The 436 cases studied in the Research Laboratory in New York, however, were not selected; of these cases the following were found associated with the bovine bacillus:

Diagnosis	Adults, per Cent	5 to 16 Years, per Cent	Under 5 Years, per Cent
Pulmonary tuberculosis	none	none	none
Tuberculous adenitis, cervical	4	37	57
Abdominal tuberculosis	16	50	68
Generalized tuberculosis	3	40	26
Tubercular meningitis with or without general- ized lesions	15
Tuberculosis of bones and joints	5	3	..

Kossel's studies also bring out the significant fact that in organs directly related to the digestive tract, like the lymph-nodes of the neck and the abdominal organs, bovine bacilli are as frequent as human bacilli.

Eastwood and also Griffith⁶ studied a series of 195 deaths from all causes, between the ages of two and ten years. The results of these interesting studies are combined in the following table:

AGE PERIODS	NUMBER OF CASES			CLASSIFICATION OF CULTURE ISOLATED		
	Free from Tubercle Bacilli	Tubercle Bacilli Dead	Tubercle Bacilli Living	Bovine	Human	Mixed Bo- vine and Human
2- 3 years	27	1	19	6	13	..
3- 4 years	12	2	21	4	17	..
4- 5 years	14	4	14	1	13	..
5- 6 years	12	3	15	..	14	1
6- 7 years	1	3	16	3	13	..
7- 8 years	3	2	2	..	2	..
8- 9 years	4	2	4	1	3	..
9-10 years	4	3	7	2	5	..
TOTAL	77	20	98	17	80	1

It will be noted that of the total of 195 children, 118, or 60.5 per cent, showed evidence of tuberculous infection. The condition found in the 118, was as follows: In 92 (47.2 per cent of 195 or 7.0 per cent of 118) tuberculous lesions, verified by subsequent cultures, were found; in six (3.1 per cent of

⁶ Reports to the Local Government Board on Public Health and Medical Subjects. London, 1914, n. s., 88, 1914.

195 or 5.1 per cent of 118) living bacilli were obtained in culture, but there were no tuberculous lesions; and in 20 (10.3 per cent of 195 or 16.9 per cent of 118) tuberculous lesions were present, but the tubercle bacilli apparently were dead or too feeble or too few in number to infect guinea-pigs. One of the interesting features of this investigation is that living tubercle bacilli may be present in children in the absence of lesions, and on the other hand, tuberculous lesions may be present while the bacilli responsible for them may be dead.

The more recent figures are well summarized by Griffith,^{6a} who analyzed 1,068 cases studied by the British Commission on Tuberculosis. Of this number, 803 showed human bacillus infection, 194 bovine bacillus infection, and five a mixed infection. Of various regions involved, the examination showed that bovine infections occurred as follows: bones and joints, 19.7 per cent; genito-urinary organs, 17.65 per cent; cervical glands, 46.3 per cent; meninges, 20 per cent; scrofuloderma, 34.65 per cent; lupus, 48.9 per cent. As to the age periods, bovine infection occurred as follows: during the first five years of life, 37.55 per cent; from five to ten years, 29.45 per cent; from ten to sixteen years, 14.66 per cent; after sixteen, 6.25 per cent.

Woodward voices the prevailing opinion when he maintains that the more deeply we go into the subject, the bovine side of the question comes to take a larger and larger place, especially in connection with surgical and abdominal tuberculosis, not only in the child but even in the adult.

From the standpoint of our present knowledge we must consider that practically every case of bovine tuberculosis in man is ingestion tuberculosis, contracted from milk or fresh milk products. Experimental evidence indicates that tubercle bacilli may penetrate a healthy digestive tract probably either through mouth and throat or the small intestines. Their entrance may be facilitated by diseases of the teeth, tonsils, or of the mucous membranes of the digestive tubes. Ofttimes the bovine bacillus lodges in the glands but fails to set up disease on account of small numbers, low virulence, or resistance of the host. How the tubercle bacilli get into milk and the frequency with which it is infected are discussed on page 719.

Occasionally butchers and also pathologists at autopsies become infected with the bovine bacillus through wounds. These accidents furnish further experimental proof that the bovine type of the tubercle bacillus possesses a certain degree of pathogenicity for man, though in the adult it appears to be comparatively slight. Skin tuberculosis is usually benign.

MODES OF INFECTION

There are two great sources of human tuberculosis: the principal source is man himself; the secondary source is cattle.

From man tubercle bacilli leave the body mainly in the sputum, where they are found in great numbers in all open cases of pulmonary tuberculosis.

^{6a} *J. Path. & Bacteriol.*, Feb., 1920.

Tubercle bacilli may also leave the body in the discharges from any open tuberculous lesion wherever situated, especially in discharges from the lymphatic glands, bones, intestinal or genito-urinary tracts, or the skin. In open cases of pulmonary tuberculosis some of the sputum is swallowed and live bacilli pass in the feces, therefore any or all of the discharges from the body may be infective. But, from the practical standpoint of prevention, the bacilli in the matter brought up from the lungs are the source of the danger in the overwhelming majority of cases.

Practically all observers agree with Koch that *human sputum is the main source of human tuberculosis*. Whether the tubercle bacillus is usually transferred directly or indirectly, in moist or in dry state, by inhalation or ingestion, are questions still undetermined. There is no general agreement among students of the subject concerning the portal of entry of tubercle bacilli which cause pulmonary tuberculosis. The question at issue is a quantitative one; that is, how often are we infected by the direct aërogenic route, how often through the tonsils and upper respiratory passages, how often through the digestive tube, etc.?

Aërogenic Infection—The Cornet-Koch Theory.—The belief that tuberculosis is air-borne, that is, that pulmonary tuberculosis is a primary inhalation infection, has long been the natural and favorite theory, from the fact that the lungs are most frequently affected. This opinion was strongly expressed by Koch in 1884, and repeated by him in 1901, at the British Congress on Tuberculosis. For many years it found practically universal acceptance. Cornet taught that the tubercle bacilli entered the lungs in the dust of dried and pulverized sputum.

The evidence of pathologic anatomy strengthens the belief in the importance of aërogenic infections as the chief portal of entry. Thus, the studies by Ghon,⁷ at the St. Anne's Children's Hospital in Vienna, indicate that the actual path of infection is by the air passages. Approximately 95 per cent of 184 autopsies studied by him represent a primary localization of the bacilli in the lungs. On the other hand, it seems that direct aërogenic infection has been greatly overestimated, while some students of the subject go so far as to state it is of little or no practical importance. It is believed that very few bacteria suspended in the air actually reach the lungs, being caught on the moist mucous membranes of the upper air passages. Further, tuberculosis of the lungs is usually at the apex, which is not in the direct line that floating particles in the air would usually be mechanically carried. It is true that dust under certain conditions may contain tubercle bacilli, but it is now known that this organism soon dies when exposed directly to the sun and air, and that the dust out of doors is not apt to contain many live tubercle bacilli, and when it does so the dilution must be enormous. It is different with house dust. Tubercle bacilli may live a long time in dark, moist places, but even here the danger cannot be as great as might be supposed when we study the nature

⁷ *Der primäre Lungenherd bei der Tuberkulose der Kinder*, Berlin, 1912.

of tuberculous sputum. This substance is usually tenacious and gummy, and dries into tough, glue-like masses, which are pulverized with difficulty. It therefore seems unlikely that dust under ordinary circumstances would contain dangerous numbers of live tubercle bacilli. The danger from this source is further diminished when we consider that a large number of tubercle bacilli die in sputum even when protected from sunlight and other injurious influences. The danger of house dust containing live tubercle bacilli from a quantitative standpoint is, therefore, reduced; on the other hand, street dust raised by a March wind, or house dust raised by beating carpets or dry sweeping may be a real peril.

A dusty atmosphere, even though it contains no tubercle bacilli, is, however, dangerous, in that it irritates the delicate mucous membranes and thus opens the door for infection.

One point of importance in this controversy is the experimental evidence that it requires very few tubercle bacilli by inhalation to produce the disease, whereas it may require hundreds and even thousands to cause infection by ingestion. Findel found that from 6,000 to 19,000 times as many tubercle bacilli are needed when fed as when inhaled, to produce the disease in guinea-pigs.

Cornet and others have actually found live tubercle bacilli in the dust and upon objects of rooms where tuberculous patients were careless with their sputum. In one of Cornet's experiments forty-seven out of forty-eight guinea-pigs exposed to the dust produced by sweeping a carpet with a stiff broom became tuberculous. The carpet had been purposely infected with tuberculous sputum shortly before. Dust containing tubercle bacilli may also enter the atmosphere from soiled linen, upholstery, handkerchiefs, and other fabrics containing the dried tuberculous sputum. Tuberculous dust may also be stirred up by walking and dancing. Crawling infants and playing children are exposed to especial danger of infection. They get the fresh virulent material on their hands, which are then carried to the mouth. It is now believed that in most cases of tuberculosis the infection is contracted in childhood, but the disease develops later.

Droplet Infection.—When it was found that the danger from dust did not seem to be as great as was supposed, Flügge called attention to the fact that in speaking, coughing, sneezing, and in other violent expiratory efforts the fluids of the mouth and nose are sprayed into the air in the form of a fine mist. These tiny droplets contain tubercle bacilli or germs of any other infection that may be present. Ordinarily these droplets are only carried two or three feet, but under exceptional circumstances may be carried thirty or forty feet or more; however, at these distances the dilution is enormous and the hazard, therefore, much diminished. The tubercle bacilli contained in the droplets sprayed from the mouth are fresh and virulent, and may land directly upon the mucous membranes of the healthy individual or may be conveyed indirectly through food, fingers, and other objects. There is danger from droplet infection, but it cannot be the usual mode of transmission in tuberculosis

from the nature of the circumstances. The danger from droplet infection is increased by close association with the patient in stuffy, ill-ventilated rooms, especially if the individual does not take proper care in coughing and sneezing. For a further discussion of droplet infection see pages 477 and 855.

Ingestion Infection.—Little by little the view gained ground that some cases of tuberculosis, particularly in children, were due to bacilli entering through the mucous membrane of the alimentary canal. Now we recognize that much of the tuberculosis in children comes through the alimentary tract. Many years before the discovery of the tubercle bacillus Chauveau (1868) was inclined to the belief that the alimentary canal may be the portal of entry. Woodward in 1894 maintained that the infecting bacilli might reach the lungs through some part of the alimentary canal. He drew attention to the fact that in many children, and also in animals fed on tuberculous material, the lungs may be markedly affected. He traced the course of the infection through caseous or old calcareous mesenteric glands up through the diaphragm to the posterior mediastinal glands, and so to the lungs. Still in 1899 analyzed 259 fatal cases of tuberculosis occurring in the Hospital for Sick Children, London, and concluded that the infection had occurred through the alimentary canal in 20.5 per cent of the cases. Shennan in 1900, dealing with 316 autopsies at the Royal Hospital for Sick Children in Edinburgh, found this ratio to be 28.1 per cent.

There is no doubt that the lungs are more or less involved in all cases of generalized infection, especially in children, but these are not cases of pulmonary tuberculosis (phthisis) in the usual meaning of the term. Unquestionably children often contract tuberculosis but they rarely have phthisis. It is phthisis or pulmonary tuberculosis which causes 87.5 per cent of all the mortality from tuberculosis and whose mode of origin is still in question.

Recent studies show that primary intestinal tuberculosis infection in the British Isles is relatively frequent. Evidently raw milk containing virulent bovine tubercle bacilli is consumed there to a serious degree. In the United States, however, Opie³ found that healed tuberculosis of the mesenteric glands is seldom seen.

Behring in 1903 maintained that the tubercle bacilli might be taken up from the intestine and pass through the mesenteric glands, so gaining access by the blood stream to the lungs without leaving any lesion in the gut or glands to mark the portal through which they had entered or the route by which they had traveled, and that pulmonary tuberculosis was commonly caused in this way. The tender mucous membrane of babies permits the bacillus to pass readily. The bacilli remain latent in the tissues and acquire increased activity later in life. According to this view tuberculosis of adults is the "end of a song, the beginning of which for the unfortunate patient was sung in the cradle." Behring's theory of the origin of phthisis did not find a ready acceptance. Nevertheless, the belief that phthisis may be caused by

³ *Am. Rev. Tuberculosis*, 1920, 4: 629, 641.

bacilli which have been swallowed and absorbed from the digestive tube gradually gained ground. Vallée in 1904 concluded from his own investigations that ingestion of dust or food infected with tubercle bacilli was the quickest and surest method of infection. A little later Calmette (1905) appeared as a strong supporter of this view and went so far as to assert that the immense majority of cases of pulmonary tuberculosis in man are caused by ingested bacilli and not by inhalation. Whitla, in 1908, and also Symmers repeated some of this work and became converted to Calmette's doctrine. Cobbett (1910) considered that the ingestion theory is based on a slender substructure of experiments from which too sweeping conclusions had been formed. Thus Calmette and his colleagues claimed that even anthracosis is caused not by the carbon particles inhaled, but the particles ingested, which pass through the intestinal mucosa and lodge in the lungs. Cobbett showed the experimental error and demonstrated that India ink intimately mixed with cream is not absorbed in any great amount from the intestine, for the cream reappears of a normal color in the lacteals. He found, however, that feeding finely divided carbon matter caused traces of pigmentation in the lung and bronchial glands when long continued. Heller and Vulcanstein showed that the feeding of large amounts of coal dust never produces that grade of anthracosis which is found after the inhalation of much smaller amounts.

Calmette⁹ recently summed up his belief that too much emphasis has been laid on the respiratory tract as the most frequent path of infection in both man and animals. He refutes the teaching of Flügge and states that tubercle bacilli floating in the air are not often sufficiently numerous to be carried into the fine bronchial ramifications. Small droplets do not as a rule contain tubercle bacilli. Numerous researches have shown that healthy lungs are nearly always germ free. Without denying the possibility of air-borne infection, he takes for granted that the most frequent method is by way of the absorbing membranes: the buccal cavity, the digestive tract and occasionally also the external mucosa, including that of the eyes. Calmette believes there is a lack of logical control in our campaign against tuberculosis. He concludes that a prophylactic campaign must at the present time be confined to the avoidance of overinfection. Further, he thinks that to attempt total eradication is a wasted effort.

There is now sufficient proof to state definitely that tubercle bacilli, when taken in food or drink, may pierce the mucous membrane of the digestive tract and produce lesions in distant parts of the body. It is also demonstrated that the tubercle bacillus may thus travel without leaving macroscopic evidence of its passage in its wake. Fraenkel¹⁰ and others have shown that the tubercle bacilli may pass through the uninjured skin of guinea-pigs, leaving no trace of their passage at the place where they had been rubbed upon the skin, yet causing tuberculosis of the internal organs. Tubercle bacilli may remain alive

⁹ *Tubercle Bacillus Infection and Tuberculosis in Man and Animals*, trans. by W. B. Soper and G. H. Smith, Williams and Wilkins Co., Baltimore, 1923.

¹⁰ *Hyg. Rundschau*, 1910, 20: 817.

in lymph-nodes, as well as other organs, for 104 days (Bartel) without producing gross or visible changes. Ravenel and others have shown that tubercle bacilli may pass through the intestinal wall without leaving a trail behind them. It does not, therefore, necessarily follow that the seat of the primary lesion in tuberculosis is the site of the entrance of the infection.

It is also claimed that, no matter how the tubercle bacillus reaches us, whether in dust or droplets, by kissing, or through fingers, flies, cups, handkerchiefs, or milk, it either passes through the tonsils or mucous membrane of the upper respiratory passages, or is carried into the intestinal tract and absorbed from the intestines. Viewed in this light, the portal of entry even in dust infection may be through ingestion rather than through direct aërogenic infection of the lungs. Experimentally it is easy to prove that tubercle bacilli given by the mouth may produce a generalized and fatal tuberculosis; thus, of one hundred guinea-pigs given one large feeding of a bovine culture by Rosenau and Anderson, ninety-nine died of tuberculosis. That infection by ingestion does not tell the whole story is judged from the fact that primary tuberculosis of the mesenteric nodes in man is not as common as we might expect. On the other hand, it is claimed that the tubercle bacillus may pass these lymph glands, leaving little or no trace behind them. Thus the work of Weichselbaum and his pupils, Bartel, Neuman, and Spieler, strengthens the importance of ingestion as the portal of entry. These investigators found that the tubercle bacillus produces, in addition to the specific tubercles, other lesions of a simple lymphatic hyperplastic character. These early lesions are called the "lymphoid stage" ("lymphoide stadium"). The recognition of this early stage is of importance in determining the point of invasion.

These investigators assume that the tubercle bacillus is carried from the mesenteric or the neck glands either through the lymphatics directly, or through the thoracic duct and the arterial circulation to the lungs and other tissues and organs of the body. The disease usually localizes itself in the lung because the tubercle bacilli are caught by the capillary network of the lungs which are the first to be passed through and because this organ is very susceptible. It is now the general belief that tubercle bacilli reach the lungs through the blood stream rather than through the lymphatics.

The important fact that first infections with tubercle bacilli are quite different from subsequent infections, complicates the story (see page 173). Opie states that first infections may occur by way of the lungs or by way of the gastro-intestinal tract; and the occurrence of one lesion tends to prevent the other (see Superinfection, page 172).

It is clear from the evidence at hand that tuberculosis may arise either by inhalation or by ingestion, and it is becoming increasingly clear that in most cases the bacilli enter the body through the mouth, throat and tonsils, which are common passages for air and food. We practically never know just how or when the infection was acquired, and one cannot even guess at the size of the infection dose in any single instance. Before a final judgment can be given concerning the usual channel of entrance of the tubercle bacillus causing

phthisis, we will need more information on the subject. There are many other things about tuberculosis that are imperfectly understood. The disease needs further study.

Miscellaneous Modes of Transference.—Under certain circumstances flies may readily transfer tubercle bacilli from exposed sputum to fingers, lips, or food. This may account for an occasional infection.

Large quantities of tuberculous sputum that escape disinfection and an additional large number of tubercle bacilli in the excreta finally reach the drinking water. Nearly all persons with tubercle bacilli in their sputum pass some of them in their feces. The tubercle bacillus is particularly resistant to putrefactive processes, and may live a long time in water. The use of contaminated water can, therefore, not be disregarded. Lawrason Brown found tubercle bacilli in the water of the Saranac River into which sewage from a sanatorium was discharged. A study of the vital statistics of Hamburg, Lowell, and Lawrence seems to show a diminution in tuberculosis following a purification of the water supply by filtration (Mills-Reincke Phenomenon, page 1040). It is clear, however, that tuberculosis is not ordinarily a water-borne infection, and this mode of transference is not a major problem. Infected eating utensils and many other unusual modes of transfer are occasionally responsible for the transference of the infection, but these are only accidental and incidental.

Infection in Childhood.—Every child has numerous opportunities to become infected. Beginning with no infection at birth, a very small proportion of infants become infected by the end of the first year, as indicated by the Pirquet skin tuberculin test. At the age of two years, the percentage is 10; at four years, 25 to 30 per cent; from five to ten years, about 50 per cent; and by fifteen years, from 60 to 70 per cent. Pulmonary tuberculosis is rare in childhood; glandular tuberculosis common.

The infant at birth has a clean bill of health. After a year, it becomes a quadruped and on all fours, so that it comes in close contact with sputum on the floor. Soon it drinks cow's milk, much of which contains tubercle bacilli. As the child grows, it continues in close contact with raw sputum in the dust and dirt of floors, streets, gutters and surfaces, so that it soon "eats its peck of dirt." At school, at play, and at home the ingestion of offal and filth of all kinds is repeated. The contact between child and child, and between child and adult is intimate, and of such a nature as to favor the spread of infection. The child at play, at marbles, at ball, at hoop rolling, at top spinning, at rope skipping, and in scores of other ways cannot help getting on its hands the sputum of others. Children have no inherent sense of cleanliness. Hand to mouth infection becomes a hygienic problem of first rank. Children thus have frequent chances of acquiring tubercle bacilli from dust, dirt, milk, droplets, and other sources. Krause believes that the "schmutz and schleim" infection of Volland plays a major rôle in children, and that the adult disease is an expression of childhood infection.

The consensus of opinion now favors the view that the prevalence of tu-

berculosis is due to the frequency of intercourse between children and tuberculous adults in a contagious stage, and this is often dependent on the income. Hence, the disease is stratifying itself in social layers. Tuberculosis, then, is a contagious disease contracted largely by exposure of children to adults with phthisis. In more than one sense it is a house disease.

The *eyes* seem to be channels of entrance for the tubercle bacilli, at least primary disease of the eyes of children is common. Phlyctenular conjunctivitis sometimes antedates the appearance of tuberculous lymph glands or pulmonary disease. Children, then, may owe their minor eye infections to an underlying tuberculosis. Recognition of this fact is important to guide general prophylactic measures instead of simply local eye treatment.

Contact Infection.—The majority of cases of phthisis contract the disease through "contact." Contact infection is a general and convenient term; it implies the rather quick transference of fresh infection in which the bacilli pass from one individual to the other in a brief space of time and through a short distance. Contact infection may be either direct or indirect; through bacilli in the air, or through contaminated food, through soiled fingers or objects; through sputum smeared surfaces, as well as in numerous other ways. The infections transferred through kissing, pencils, pipes, toys, cups, and other objects all come under the convenient category of "contacts." Even infection through droplets is included in the present-day conception of contact infection. The term is a practical one, and implies close association, though not necessarily actual touch, between the sick and the well. Viewed in this sense, tuberculosis is a house disease or a family disease. With this conception it makes little practical difference whether the infection enters the body through the respiratory tract or the digestive tube. Either or both would be possible in regarding the disease as "contagious" in the sense of contact infection.

At all ages, the intimacy and length of exposure are the determining factors. Pollak states that the earlier the infection the more serious the outcome. This receives support from Wallgren's statistics, for out of fifty-one consumptives, fifteen had been exposed during the first five years of life, whereas of the thirteen healthy persons who gave a history of exposure, in but one case had that exposure been before the sixth year. It is now believed that the *infection* is usually received during childhood, but remains latent until adolescence or early adult life, when the *disease* becomes clinically apparent.

Although there is some doubt concerning the exact mode of transmission, and the portal of entry that the tubercle bacillus usually takes, we have sufficient knowledge to guide our preventive measures with every assurance of success. One thing is certain: tuberculosis is an infection spread mainly from man to man, usually because sputum in some form or other enters the mouth; and secondarily from cows, through milk.

Conjugal Tuberculosis.—It has long been a puzzle why husband and wife do not give each other the disease more frequently. Roussel's figures¹¹ are

¹¹ *Médecine*, 1923, 4: 630.

typical: he found the husband alone tuberculous in 423 families; the wife alone in 163, and both husband and wife in forty-nine, but the infection had preceded marriage in twenty-seven in this last group. Consequently, conjugal contagion could be incriminated in only 5.2 per cent of the total 423 families, and the infection was mild. On the other hand, Barnes¹² found that the histories of 229 consecutive widowed patients admitted to the Rhode Island State Sanatorium, 1905-1921, showed that 93, or 40 per cent, lost their consorts by death from tuberculosis, a tuberculosis mortality over three times that of the married people of the community. Ward¹³ takes the view that the great majority of those mates of tuberculous husbands or wives do sooner or later show signs or develop symptoms of tuberculosis, but that the great majority of those infected recover and make a speedier recovery than most tuberculosis patients. It is known that the disease is unlikely to start in adult life, on account of the immunity acquired through prior infections, and in this case enhanced by repeated doses frequently received.

Marriage and Tuberculosis.—A tuberculous woman, as a rule, loses nothing by marriage. It is not likely to affect her disease either way; if it does affect her it is more than twice as likely to improve her condition as to cause deterioration. Pregnancy and parturition, however, are likely to make her worse with a 50 per cent chance for deterioration and a 19 per cent chance for improvement. The children are several times as likely to be tuberculous as those of healthy people.¹⁴ The rule is to advise women with active tuberculosis not to become pregnant and even to interrupt the pregnancy. It seems to be especially fatal with those who have a recently acquired infection, subactive lesions, and who are, at the same time, sensitive to overexhaustion, to menstruation, and especially if they have impaired heart, kidneys, or liver. A tuberculous mother should not nurse her baby, and otherwise must take scrupulous precautions to prevent infection.

IMMUNITY

Man possesses some resistance to tuberculosis. This is shown by the fact that many cases recover spontaneously and that 70 per cent of all individuals who reach the age of sixteen years and who spend most of this time in association with their fellowmen under the usual urban conditions have at one or more times been infected. The resistance to tuberculosis increases after middle life, due perhaps to the immunity which is induced by these prior infections. The rarity of conjugal phthisis is thus explained. There is probably no true racial immunity to tuberculosis. Some races show a smaller incidence to the disease, owing probably to modes of life, habits of nutrition, and conditions of exposure. Jews and Italians are said to have a racial immunity; the Irish are more susceptible. Some of the white races

¹² *Am. Rev. Tuberc.*, 1921, 5: 670.

¹³ *Lancet*, 1919, 2: 656.

¹⁴ Ward, *Lancet*, 1923, 557.

seem to acquire a certain degree of resistance through association with an almost universal infection. All races long removed from civilization are particularly susceptible. In such, tuberculosis runs an acute and fatal course such as is often seen in young children and in experimental animals. The experience of the World War again emphasized the fact that individuals from countries where tuberculosis is rare are much more susceptible than the inhabitants of countries in which the disease has long been widely prevalent. In such susceptible persons, the disease runs an acute and fatal course with lesions resembling the type found in infants.

The human body is capable of taking care of a certain amount of infection without the development of clinical tuberculosis. The dose, that is, the number of tubercle bacilli and their virulence, is, therefore, a very important factor in determining the course of events. This may readily be demonstrated upon susceptible animals and is doubtless true of man. It takes about ten tubercle bacilli of ordinary virulence to infect a guinea-pig. *In man the balance between immunity and susceptibility to tuberculosis is delicately adjusted: there is a small factor of safety.* The resistance to the disease may be increased by attention to personal hygiene, fresh air, and good food; immunity may readily be broken down by any weakening influence; herein lies the keynote of personal prophylaxis.

The immunity to tuberculosis is not sufficiently strong to overcome a large amount of infection. At best it is weak and partial. As in other infectious processes, the strongest and most robust individuals in the prime of life succumb to the disease in a short time if they receive into the tissues a large number of virulent tubercle bacilli. Frequent reinfections occurring at short intervals with small numbers of tubercle bacilli may break down the immunity. Hence the avoidance of infection is one of the most important of our preventive measures.

Romer has shown that actively tuberculous guinea-pigs resist a second small dose of tubercle bacilli. Krause confirmed these results on monkeys, and von Behring and Calmette on cattle. This is confirmed by clinical experience of the disease in man and indicates that a disease which is progressive within the body may not allow another focus of infection to start and gain headway. *Superinfections* may, however, occur under certain conditions. It is believed that tuberculous infection occurs many times but that the disease starts once only during the life of man, and this usually takes place in childhood, although it may break out within a year.

The frequency with which such diseases as measles, whooping-cough, syphilis, and other infections are followed by tuberculosis makes us believe that most acute infectious processes diminish resistance to the tubercle bacillus and serve to lighten up or spread the disease.

The mechanism of the immunity to tuberculosis is probably exceedingly complex. There is no antitoxic immunity. The tuberculins are not true toxins. Phagocytosis and cellular reactions play a very important rôle. Studies upon anaphylaxis throw a certain amount of light upon the mechan-

ism of immunity in tuberculosis. The phenomenon of hypersusceptibility is beautifully illustrated in the action of tuberculin, which is a comparatively harmless substance to a normal individual, but produces a marked reaction in a sensitized individual. This reaction must be useful in protecting the body against the invasion of the tubercle bacillus, and also in guarding it against the spread of the disease after it has become localized. Thus, if tuberculin is placed upon a normal conjunctiva no reaction follows.¹⁵ This first application, however, sensitizes the tissues of the conjunctiva so that, if the application is repeated after the lapse of a few weeks, there is a violent reaction. The same phenomenon doubtless occurs when a tubercle bacillus lodges in a lymph gland or in the lung or some other part of the body. The first time it meets with little resistance; the next time the tissues react immediately and vigorously. All of nature's protecting agencies, such as the germicidal substances in the blood, the phagocytic cells, and antibodies, are concentrated upon the point where they are most needed. In the same way the body protects itself against the extension of a tuberculous focus. The parts surrounding a tubercle become sensitized and react so as to encapsulate the focus with a cellular and fibrous coat of mail. This reaction is probably stimulated by small amounts of tuberculin produced within the tuberculous focus. When the tuberculin is not produced autogenously in sufficient amounts, as in chronic lesions of the bones, or inactive processes of the glands or skin, the specific reaction may be stimulated to advantage by the injection of small quantities of tuberculin. If, however, the tuberculin is given in too large amounts or too frequently, the power of reaction is readily broken down.¹⁶ When this occurs the mechanism of immunity has been destroyed, there is little resistance left to the extension of the infection, and death soon occurs. Clinical experience has demonstrated the danger of large doses of tuberculin or small amounts too often repeated in tuberculosis. The same may readily be demonstrated experimentally in the lower animals. These facts are of fundamental importance in the use of tuberculin both in diagnosis and therapy.

It is quite proper to deny dogmatically the *hereditary transmission* of tuberculosis in educational pamphlets for popular use. The infection is not transmitted hereditarily, although it occasionally passes from mother to fetus congenitally. Tubercle bacilli do not occur in the spermatozoon, and do not appear in the seminal fluid. They are not found in the ovum; in fact, a tubercle bacillus in the ovum would doubtless result in the death of the egg. The bacilli, however, occasionally pass from mother to fetus through the placenta. Warthin shows that placental tuberculosis is more common than is supposed. The lesions in the placenta are not those of typical tubercle formation.

While the tubercle bacillus itself is rarely transmitted from parent to fetus, an *hereditary tendency* or disposition to the disease may be transmitted. We have no definite knowledge as to what this decreased resistance consists in;

¹⁵ Rosenau and Anderson, *J. Am. Ass.*, 1908, 1: 961.

¹⁶ A state of anti-anaphylaxis or negative phase is produced.

it may be a diminished power of reaction. For this view there is analogy in the experiments upon anaphylaxis in guinea-pigs, in which it has been shown that hypersusceptibility to a foreign protein such as tuberculo-protein may be transmitted from mother to young.

A mild infection with bovine tuberculosis in early life seems to leave a certain degree of immunity against the human strain. At least, persons who have glandular tuberculosis of the bovine type in childhood are said to be less apt to have tuberculosis of the lungs in later life. Likewise, the human strain injected into cattle produces a definite immunity against the bovine type. Cattle may be immunized by the intravenous injection of 2 c.c. of a suspension of a pure culture of the human tubercle bacillus. This produces an immunity which probably lasts for one to two years. It should be remembered that the human bacillus under these circumstances remains alive for a very long time, and may appear in the milk provided there is a lesion in the udder. This presents a danger which cannot be disregarded.

Trudeau long ago showed that the only definite immunity that could be induced in experimental animals was through the use of live tubercle bacilli. It is now generally conceded that the only immunity obtainable is through mild infection. "Only the tuberculous are immune to tuberculosis." Webb and Williams¹⁷ have produced a certain amount of immunity in guinea-pigs and monkeys by the injection of small numbers of live tubercle bacilli. This procedure is not practical, even hazardous for man.

Calmette and Guérin reported striking results on immunity to tuberculosis in cattle, produced by vaccination with tubercle bacilli of the bovine type, reduced in virulence by prolonged artificial cultivation. Through 250 successive cultures produced during thirteen years on potato in the presence of ox bile and 5 per cent glycerol, the culture became avirulent even in large doses for animals including the monkey. The loss of the tubercle-producing power seems to be permanent. Their experiments show that from 50 to 100 mgs. of these living bacilli injected into the connective tissue of a calf produce immunity to 5 mgs. of virulent bovine bacilli injected intravenously from three to eighteen months later.

Calmette, Guérin, and Weill-Halle¹⁸ state that the immunity persists as long as the bacilli inoculated remain in the body which is two years in calves and six months in rabbits and guinea-pigs. Weill-Halle and Turpin¹⁹ report that 4,517 children have been vaccinated since June, 1924. The vaccinated children all came from homes with open tuberculosis. Selter is cautious to state that the method will not replace "natural" inoculation, but is to be considered in the case of infants exposed to massive infection in their home environment.

The injection of dead tubercle bacilli and their products long ago proved

¹⁷ *J. Am. M. Ass.*, 1911, 57: 1431. *Trans. Internat. Cong. Tuberc.*, 1908, 210; also, Lieb, *J. Med. Research*, 1910, 30: 3; also, Lawrason Brown, Heise, and Petroff, *J. Med. Research*, July, 1914.

¹⁸ *Bull. Acad. de méd.*, 1924, 91: 787.

¹⁹ *Presse méd.*, 1925, 33: 1655.

valueless. The injection of small doses of virulent bacilli has more than once proved dangerous. The work of Calmette and Guérin points in a promising direction, for there is abundant evidence for the dictum of Krause that infection is a *sine qua non* of immunity in tuberculosis: "No resistance without tubercle." It appears that immunity to tuberculosis is not general, but quite specific.

Resistance of the Virus.—We have no easy method of determining just when the tubercle bacillus dies. The criterion of death depends upon animal experimentation. The tubercle bacillus has no spore and may be classed with other non-spore-bearing organisms so far as its viability is concerned. Its virulence fades before it dies. It is doubtful whether the waxy substances protect the bacillus against external harmful influences to any unusual extent. The thermal death-point is 60° C. for twenty minutes. This is much less than was once considered.²⁰ Failure to recognize the lesions produced by the dead tubercle bacillus is responsible for some of the false conclusions reached by experimenters upon this subject.

From a practical standpoint the resistance of the tubercle bacillus in sputum is of prime importance. Protected from the sunlight, it is now known that they may live in dried sputum for months. All the bacilli do not survive under these conditions, but we lack methods to determine the quantitative reduction.

The tubercle bacillus withstands cold very well. It has a marked resistance against putrefactive processes. It will live a year in water, which is a fact not to be neglected, as many tubercle bacilli finally find their way into drinking water, and occasional trouble from this source is possible.

For the destruction of the bacilli in sputum only very strong germicides or exposure to steam or boiling water should be depended upon on account of the nature of sputum which hinders penetration. Burning is the most practical method for disposing of tuberculous sputum (see page 181). Five per cent carbolic acid is sufficient, provided equal parts of sputum and solution are mixed and the exposure continued for twenty-four hours. Alkaline solutions of cresols are best because they dissolve and penetrate.

Sunlight is one of the best germicides and often destroys tubercle bacilli quickly. In direct sunlight the naked bacilli exposed directly die in a few hours; in diffused sunlight, in a few days. Tubercle bacilli imbedded in sputum masses may be protected against the germicidal action of the sun's rays. The time the bacilli may live under these circumstances is variable.²¹

PREVENTION

Preventive measures are based upon two important facts: that tuberculosis is an infection mainly spread from man to man through tuberculous sputum,

²⁰ The thermal death point of pathogenic microorganisms in milk. M. J. Rosenau, *Hyg. Lab. Bull. U. S. Pub. Health and Mar. Hosp. Serv.*, No. 42.

²¹ Von Bergen, *Schweiz. med. Wchnschr.*, 1920, 50: 1120; also Técon, *Paris méd.*, 1920, 10: 33.

and secondarily from cattle through infected milk. Preventive measures fall into two categories: (1) avoiding the infection, and (2) increasing resistance through personal hygiene. Both are necessary. The infection may be avoided through segregation; the use of pasteurized milk, or milk from tuberculin-tested cattle; education; disinfection; proper disposal of tuberculous sputum; the avoidance of contact with open cases, especially with those who do not use proper precautions; early diagnosis, etc. Increased resistance may be gained through fresh air, good food, rest, and compliance with the dictates of personal hygiene. This part of the subject includes sociologic and economic reforms, without which the warfare against tuberculosis cannot succeed. Im-

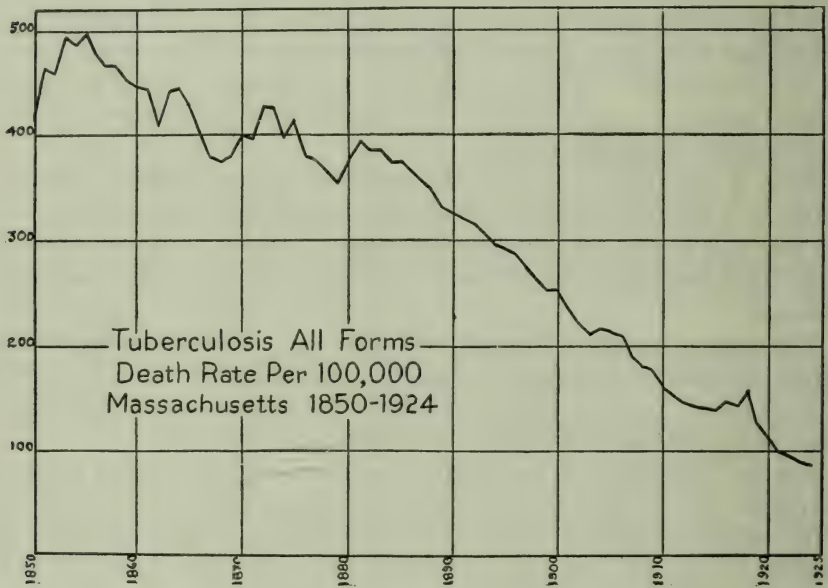


FIG. 10.—CHART SHOWING THE DECLINE IN THE DEATH RATE FROM TUBERCULOSIS.

provement in housing conditions, lowering of the cost of living, increase in the scale of wages, and all forms of uplift help secondarily to diminish the amount of the disease. Furthermore, it will be necessary to consider secondary agencies, as preventive clinics, health insurance, notification, open-air schools, day and night camps, etc.

It is well to remember that tuberculosis has gradually declined in England and the United States and also in Massachusetts since 1850—before the tubercle bacillus was discovered. The decline was gradual from 1850-1885, but quite pronounced since that date (see Fig. 10). In 1922 our death rate from tuberculosis was below ninety per 100,000. A few of our states now equal the low rate of New Zealand, fifty per 100,000.

The causes of this decline have been much discussed. It may be due to better food supply at all seasons of the year, brought about by improved

methods of transportation, and the general use of refrigeration and canning; it may be due to amelioration of social and domestic life; it may be due to better hygiene and sanitation; or, it may be due in part to the special anti-tuberculosis measures. It may also be due to self-protection which is more intelligent and general; early and accurate diagnosis, segregation even though



FIG. 11.—TUBERCULOSIS (ALL FORMS) DEATH RATE PER 100,000 IN ENGLAND AND WALES AND ORIGINAL REGISTRATION AREA IN UNITED STATES, 1900-1922.

imperfect; the pasteurization of milk; improvement in the health of dairy herds; correction of undernourishment in school children; the child hygiene movement; the fact that predisposing diseases such as measles, whooping-cough, typhoid fever, and others are less common also plays a part. On the other hand, the decline may have been little influenced by any of the usually assigned causes, but may be a biological phenomenon indicating a falling off in the virulence of the tubercle bacillus, or, what seems less likely, an increased resistance owing to specific immunization of the population. During recent

years, there has clearly been a lessened mortality, but apparently an increased morbidity. In other words, the infection seems to be more prevalent, but the disease less fatal.

Community Organization.—The individual alone cannot protect himself against infection without the help, coöperation, and organization of the community. A special antituberculosis campaign should be part of the public health work of every community. An active program for the study and prevention of tuberculosis should be encouraged in every locality.

The legislative measures include compulsory reporting and registration of cases; a penalty for tuberculous persons who place others in danger; compulsory segregation of indigent, careless, or irresponsible open cases; anti-spitting ordinances; regulations to protect and pasteurize the milk supply; a tuberculosis clinic for early diagnosis and treatment; a tuberculosis nursing service; special measures for the young, especially the preschool age; medical inspection of school children and special classes for the pretuberculous; laboratories for bacteriology, roentgenology, and operative service; sanatoria for treatment and segregation; and finally education and investigation with follow-up care of every case.

Segregation—Sanatoria.—Tuberculosis is a "contagious" disease, and it is now perfectly plain that one of the important preventive measures in this as in all other communicable diseases consists in isolation. A case isolated is a case neutralized, hence the great value to the community of sanatorium treatment. Isolation in this case refers only to those individuals having tubercle bacilli in their sputum. It should not be used as a terminal measure. Too often the door is locked after the damage is done. The power of forcible removal and isolation of the irresponsible, careless or indigent consumptive is essential to success, but not at present practicable. The isolation in tuberculosis need not go to the extreme practiced in the acute communicable fevers. In fact, we cannot for many years to come object to giving a case of open pulmonary tuberculosis restricted liberty, provided he is careful and cleanly and uses proper precautions in the disposal of his expectoration. When the disease becomes less prevalent, more stringent and arbitrary measures may then be enforced. At present it would encourage concealment.

Special measures must be taken to protect infants and children against the infection.

"Every case of tuberculosis isolated means an average of at least three less new infections." Sanatoria should, therefore, be attractive and as generous as it is practical to run them. Free hospital care for the open cases is necessary, especially for the poor. Tuberculosis has diminished most in those countries where sanatoria are most in use.

Separate quarters in sanatoria ²² should be provided for the incipient cases

²² Edward Trudeau went to Saranac in 1873 as a hopeless victim of consumption. All his friends were filled with horror at the idea of his going practically alone to die, as they believed, in the Adirondack wilderness, in a little town consisting of little more than a sawmill and half a dozen cabins, forty-two miles from a railroad. Trudeau did not die, however, during the winter of 1873, but grew very much better;

and for the advanced cases. A sharp division is not always possible, for an incipient case may develop into an open case within a week, and, on the other hand, open cases may return to latency in a short time. It is better for each locality to have its own sanatorium than to provide large institutions which become unwieldy. Furthermore, tuberculosis, like other widespread infections, is largely a local problem. A distant sanatorium will neither attract, nor keep the chronic cases. Persons with tuberculosis need not necessarily go to a sanatorium with the object of remaining until cured. It is worth while if they go there but for a few months to learn the methods of treatment and the technic of prevention. While sanatoria should be well built and comfortable, extravagance is not necessary. Special police power to restrain the incorrigible consumptive in special detention wards or places is desirable and will be demanded when public apprehension is aroused.

Tuberculosis Dispensaries.—Every community should be provided with a dispensary for diagnosis, treatment, and for teaching the consumptive how to care for himself at home. Each dispensary should have at least one physician and a nurse with special experience in the problem. Much social service work can be done from such a dispensary, especially in following up cases after leaving the sanatorium. Dispensaries are necessary for early diagnosis and education—two of the essentials for treatment and prevention.

Anti-Tuberculosis Associations.—Associations for the cure and relief of tuberculosis are essential parts of the problem, and such associations should be active in every community in order to obtain hospital accommodations for the advanced cases, sanatorium treatment for the hopeful cases, and advice for the incipient cases; to obtain and maintain dispensaries, district nursing and necessary legislation and help in the problem of education. Local branches of the National Tuberculosis Association should be active in every community.

Personal Prophylaxis.—Personal prophylaxis consists in avoiding the infection and in obeying the dictates of personal hygiene—that is, living a clean, normal, and temperate life.

Close association with persons known to have tubercle bacilli in their sputum is hazardous, especially for the very young. This becomes particularly dangerous when the contact is prolonged and intimate, such as working in the same room, especially if it is small and ill-ventilated, or living in the same house, sleeping in the same bed. The more intimate the association and the less care the tuberculous individual takes with the expectoration, the greater is the hazard in communities where infection prevails. The danger diminishes materially with age; infants and children need special protection. The infection may further be avoided by refusing to drink from common cups, by taking care not to place objects in the mouth that do not belong there, by

and some ten years later, as a result of his experience, he founded the Adirondack Cottage Sanatorium, which in its primitive form consisted of a single house in which, with great difficulty, he persuaded two consumptive patients to live. That was the beginning of the demonstration in this country of the fresh-air treatment of tuberculosis, which Brehmer and others had introduced on the other side of the water (Winslow). Read Trudeau's inspiring autobiography.

avoiding dusty atmospheres, and refusing to drink milk that does not come from tuberculin-tested cattle unless it is pasteurized.

Mechanical obstructions to breathing should be corrected, by surgical methods if necessary. Functional lack of proportion in the chest and lungs of young people favor infection, and every effort should be made to help the child to outgrow them. Breathing exercises and outdoor play are especially useful.

A generous diet is one of the best prophylactics against tuberculosis. Good nutrition is fundamental both in prevention and cure of this disease. This is the one infection in which nutrition definitely influences immunity.

Resistance to the disease is increased by adequate rest, fresh air, good food, sunshine, the avoidance of all depressing influences, such as worry, overwork, strain, intemperance, and excesses of all kinds. Attention should be given to slight colds and other conditions known to be predisposing causes to the disease. Tuberculosis is the one disease in which the measures of treatment and of prevention are to a large extent identical.

Conditions favoring tuberculosis are anemia and underweight, continuous overfatigue, recurrent colds, especially recurrent bronchitis, slow recuperation from any acute infection, whether influenza, measles or whooping-cough, prolonged septic processes, or typhoid fever. It is well to remember that maturity of tissues, freedom from trauma, normal nutrition, and the absence of intercurrent disease or toxic influences are the important "factors of safety" on the part of the body.

Personal prophylaxis becomes the keynote of prevention when we recall that most of us become infected during childhood. It is, therefore, our problem to live so that the disease will not break out in adult life.

Education.—The prevention of tuberculosis, like all other widespread infections, depends for its success upon the education of the people. We are now in possession of sufficient information of a precise nature to place the facts in plain words before the public. This has been done in numerous excellent pamphlets and popular articles in the daily press and magazines, through lectures, conferences, moving pictures, exhibits, and meetings, so that there is now a widespread understanding of the problem. The modern message in tuberculosis has been one of hope, in that the disease is curable; and one of fear, in that it is transmissible. The former has been a great encouragement and has added strength to the movement; the latter is also helpful, although it has run to extremes in some quarters. An unwarranted fear of tuberculosis (phthisiophobia) has subjected the tuberculous individual to severe hardships by branding him as a leper. Even cured cases of the disease may find difficulty in obtaining work. A wholesome regard for the infection is useful and helpful in preventive medicine, but an hysterical fear of tuberculosis is quite as unwarranted as a total disregard for the infection.

Notification.—Tuberculosis should be included among the list of diseases requiring compulsory notification. Without this important feature an adequate control of the disease cannot be effected. The objection to compulsory

notification is based largely upon sympathy with the large number of individuals affected and the sensitiveness of the afflicted. Compulsory notification may occasionally result in individual harm, but is necessary for the communal good. The prejudice against notification in tuberculosis is rapidly being worn down, and successful reporting is part of the program of all up-to-date health administrators.

Tuberculosis is required to be reported in Maine, Michigan, Massachusetts (since 1907); many cities: Alameda, California; Asbury Park, New Jersey; Boston, Buffalo, Cincinnati, New York, Salt Lake City, Trenton, Yonkers—also in Washington, D. C., Minneapolis, San Francisco, and Syracuse. The list is growing and the returns are gradually improving.

In England and Wales the notification of all forms of tuberculosis, whether in public or private practice, is obligatory, since February 1, 1913. The information thus received is held as confidential.

Under practically ideal administrative conditions in Framingham, Massachusetts, for 1917-1918, Armstrong found the ratio of reported cases to tuberculosis deaths to be approximately nine to one. He cites the ratio of 4.7 to one for Cleveland in 1920, as better than will be found in many other American cities.

Out of sixty-nine cities for which data is available, ten report less than one case per annual death, thirty report between one and two cases per death, twenty-one between two and three cases per death, and eight more than two cases per death. Chicago and Flint approach the result found by Armstrong in Cleveland, with rates of 4.1 and 4.4 cases per death, respectively. These facts indicate the general inadequacy of case reporting in most cities.

Disposal of the Sputum.—As the tuberculous sputum is the principal source of the infection, it should be disinfected or disposed of so that it will be harmless to others. Perhaps the best way is to receive the expectorated matter into cloths, which may be burned, or the material may be received into one of the various forms of sputum cups and finally burned or disinfected. Persons with pulmonary tuberculosis must be warned against the possible danger to others of coughing without holding the handkerchief before the mouth and nose; under no circumstances should they spit upon the floor. Penalty for spitting upon the sidewalk, upon the floor of public buildings, and in street cars serves a useful purpose in diminishing the spread of tuberculosis as well as other diseases (see Disinfection of Sputum, page 1384).

Disinfection.—Rooms occupied by tuberculous individuals should be kept clean and disinfected from time to time. A thorough disinfection and cleansing should also be practiced before such rooms are occupied by other persons. This may be accomplished by washing all surfaces with hot soap and soda, followed by mopping with the usual solutions of bichlorid of mercury or one of the alkaline coal-tar preparations, and then a thorough airing and sunning. Formaldehyd gas alone cannot be depended upon because it lacks the power of penetration. It is only the merest surface disinfectant. Tubercle bacilli are usually imbedded in particles of sputum not easy to reach. The room may

finally be renovated and refurnished. All fabrics, handkerchiefs, bed and body linen should be boiled or steamed.

Early Diagnosis.—Early diagnosis plays an important rôle in successful prevention; not only does it give the individual the best chances of cure, but at the same time it assures the possibility of maximum protection to others. Through the use of x-rays, tuberculin and other refinements of clinical methods it is now possible to diagnose tuberculosis at a stage when it was formerly not suspected. It is a great mistake, from the standpoint of prevention, to wait until tubercle bacilli appear in the sputum before making a diagnosis of tuberculosis. The symptoms that suggest incipient tuberculosis are rather general in character. It is often necessary to make a diagnosis by exclusion, for it may be impossible for the clinician to state just where the process is located. The symptoms that suggest incipient tuberculosis are loss of weight, rise of temperature in the afternoon, or subnormal temperature with rapid pulse, loss of appetite, languor and lack of energy, anemia, dyspepsia, with or without a cough. Probably many cases of "a slight run-down condition," of transient and irregular febrile attacks, are due to a small focus of tuberculosis hidden from the ken of the clinician. In such cases a course of rest, fresh air, and better food, with a change of scene, may often prevent irreparable damage. The establishment of preventive clinics to look after such cases and the maintenance of well-equipped medical clinics to diagnose and care for the early cases are important adjuncts to preventive measures.

Housing Conditions.—It has long been realized, even before the reasons were understood, that the incidence to tuberculosis diminishes with improvement in housing conditions. This is a common observation in the stabling of cattle as well as the domicile of man. The reasons for this are complex. In addition to raising the standard of living, better houses lessen the chances of contact infection, afford better air and more sunshine, and tend generally to the well-being and uplift of mankind. House infection is after all another name for contact infection. Better housing helps to lessen the opportunity for the infection to develop into the disease. Municipalities do well to enact and enforce stringent laws regulating the construction of houses, offices, stores, and workshops. The congested and squalid slums are both a disgrace and a menace. Germs are social climbers, and many a palace is invaded with an infection from a nearby neglected alley. Philanthropists cannot do better than assist in improving the housing conditions of the poor and thus help the art of hygienic living (see page 503).

Care of the Cases in the Home.—Open cases of tuberculosis should not be cared for in the home, but in case they are, such homes should be visited by a public health nurse.²³ Home visitation is a simple but very powerful means of attacking this and other diseases. It is the surest way of reaching the indifferent and ignorant portion of the public which constitutes the great obstacle in the successful prevention of disease. Home visitation is not merely

²³ That is, a nurse specially qualified in the tuberculosis problem, and with social service instinct and training.

applicable, but almost indispensable in many public health problems, and should become one of the routine methods of a good public health organization. Such a system recognizes the fact that tuberculosis is not merely a bacterial invasion, but a disease of defective civilization. Through this means social relief may teach better standards of living and provide better food, light, air, housing, clothing, and occupation. A study of tuberculosis at close range has taught the lesson of the absolute necessity for individualizing the treatment of each case in order to obtain satisfactory results.

Tuberculosis Nursing Service.—A fairly complete service needs about four nurses per 100,000 of population. Most cities average less than this. The public health nursing especially directed towards tuberculosis is carried out by the public health nurse, the instructive worker, by private and governmental nurses.

Industrial Conditions.—Cornet taught that many a case of tuberculosis is contracted by those who are required to work alongside of a fellow workman who has pulmonary tuberculosis, especially in crowded, poorly ventilated and insanitary workshops. We now believe, however, that most cases are infected during childhood; nevertheless, exposure at any period of life may be hazardous. Allen Joslin helped to suppress the disease within two years at Oxford, Massachusetts, where tuberculosis was unduly prevalent, through medical inspection, aided by a nurse; removing the sick to sanatoria, and by a general improvement in the sanitary and hygienic conditions of the mill. Similar measures have been met with signal success in other industrial centers. Much real good can be accomplished along these lines.

Tuberculosis is the chief of the occupational infections. The disease is promoted by irritating dust, especially silica or metallic particles; by lead and other poisons; by the hours and conditions of work and the workshop.

Tuberculosis in Children.—In one sense tuberculosis is an infection of childhood, for about 70 per cent of all children by the time they reach sixteen years of age react to tuberculin, thus indicating that they have become sensitized or "tuberculized." The hazard to a child living in a house with an open case of consumption is very great. One of the fundamental considerations is to separate babies at once from exposure to such an environment. Children rarely have tuberculosis of the lungs (consumption); when they do it is usually rapidly fatal. It is hazardous to permit such sick children in school rooms or even to remain at home with other children; they should be cared for in sanatoria. It is the anemic, incipient, pretubercular, glandular, or scrofulous type that demands especial attention. Such children are best cared for in preventoria, sometimes called open-air schools. These children are going to furnish a large percentage of the open pulmonary cases in later life. In open-air schools emphasis should be laid on their health, and the teaching given secondary consideration (see page 1292).

Bovine Tuberculosis.—The prevention of bovine tuberculosis consists simply in using milk, cream, and fresh milk products from tuberculin-tested cattle. The cattle should be tested frequently, at least twice a year, for the

disease may develop in the cow in a few months. When milk from non-tested cattle is used, it should be pasteurized, and the same precaution applies to the milk used for making cream, butter, ice cream, and other fresh milk products.

Directions for Testing Cattle with Tuberculin.—Three tests are now used: (A) the subcutaneous test; (B) the intradermic test; and (C) the ophthalmic test. The *subcutaneous* test is carried out as follows:

1. Stable cattle under usual conditions and among usual surroundings, feeding and watering in the customary manner.

2. Make a physical examination of each animal, and give to each one some designation by which the animal will be known throughout the test.

3. Take each animal's temperature at least three times at two or three hour intervals on the day of injection; for instance, at 2, 5, and 8 P.M.

4. At 10 P.M. inject a dose of tuberculin under the skin in the region of the shoulder, using a sterile hypodermic syringe after disinfecting the skin at the seat of injection with a 5 per cent solution of carbolic acid or a similar antiseptic solution.

5. Tuberculin is not always concentrated to the same degree, and therefore the dose, which should always appear on the label, varies considerably. The dose of imported tuberculin is .25 c.c. for an adult cow, and before injection is diluted with sterile water to 2 c.c. The tuberculin made by the U. S. Bureau of Animal Industry is prepared so that it will not be necessary to dilute it, and the dose is 4 c.c. for cattle more than one year old and 2 c.c. for calves up to one year old.

6. At 6 A.M. on the day following the injection of tuberculin commence taking temperatures, and continue every two hours until the twentieth hour after injection, at which time if there is no tendency for the temperature to rise the test may cease.

7. A rise of 2° F. or more above the maximum temperature observed on the previous day, providing the temperature after injection exceeds 103.8° F., should be regarded as an indication of tuberculosis. Those cases which approximate but do not reach this standard should be considered as suspicious and held for a retest six weeks later, giving double the original dose.

The *intradermic* test consists of injecting two minims of undiluted tuberculin into the skin of the caudal fold at the root of the tail. It is therefore called the tail test. When positive, a swelling appears which persists, and should be read in about seventy-two hours. This test requires good technic. It is popular because it is practical and evident.

The *ophthalmic* test consists in placing a tablet containing tuberculin in the conjunctival sac. Susceptible animals show a distinct reaction, consisting of injection, swelling and exudation.

Each of the three tests has limitations. It is often desirable to use two, or all three in special cases.

The Bang Method of Suppressing Bovine Tuberculosis.—More work has been done in Denmark in accordance with the recommendations of Bang

in the suppression of tuberculosis of cattle, and the results achieved are of greater value than in any other part of the world. The system, in short, is as follows: Herds are tested with tuberculin upon the application of the owner of the cattle. After the test, the herd is divided into two parts: (1) the healthy section and (2) the reacting or tuberculous section. These herds are, if possible, kept in separate buildings. If this cannot be done, they are kept in separate parts of the same building, a tight partition separating them. The milk from both sections is used by the creameries, but it is the almost universal practice in Denmark to pasteurize the cream preparatory to ripening it for churning, and the law requires that skim milk shall be heated to a point that will kill the tubercle bacilli before it is returned to the farmers to be used for feeding purposes. The law further provides that the sediment that remains in the separator shall be burned.

The calves from the cows in the reacting sections are removed from their dams immediately after birth and are reared on the milk of healthy cows or on milk of reactors that has been heated. These calves are tested when they are three or four months old, and if they do not react, they are permitted to enter the sound section of the herd. As a matter of fact, reactors are very rare among these calves. Most of them are born healthy, and when cared for as directed, they remain free from tuberculosis. All cows with tuberculosis of the udder are required to be reported and are killed. Some compensation is allowed for them. The appraisement is equivalent to one-fourth of the meat value of the animal. The other tuberculous cattle in the reacting section are examined physically from time to time and killed in public abattoirs under veterinary control. Their flesh is then disposed of in accordance with the recommendation of the inspector. Some of it is seized and destroyed, some of it is sold for food. The law further provides that cattle brought into Denmark shall be kept in quarantine until tested with tuberculin and found free from tuberculosis.

Many herds have been tested in Denmark with negative results, proving that tuberculosis is not a necessary disease among highly developed dairy cattle, and that the tubercle bacilli are not omnipresent.

In the United States the Bang method has never been popular and there are only about 50 such herds in the country at the present time. On the other hand, the method of controlling bovine tuberculosis through accredited herds has met with success.

Health Insurance.—Health insurance, sick benefits, pensions, non-employment insurance, and similar schemes to attempt social justice are helpful in the fight against tuberculosis. The German industrial associations were under government supervision and did more than care for the tuberculous workman. The heavy drains upon the funds of the industrial associations were checked by the establishment of "preventoria." These are attractive country places where the working man can go when he is "run down." This simple measure is a great boon, and prevents the development of many a case of tuberculosis as well as other diseases (see also pages 1156 and 1220).

Organizing a Local Tuberculosis Campaign.—Tuberculosis being a local problem should be attacked by every town throughout the country. The method of organizing a tuberculosis campaign is first to interest a number of different agencies, such as the church, business, doctors, politicians, women's clubs, the press, and the board of health. This beginning can best be done by one person who will devote himself or herself to the work. After the press has printed a few articles on tuberculosis and the pulpit has helped to emphasize the importance of the movement, a local committee should then be formed to invite a tuberculosis exhibit. These exhibits may be obtained either from the National Association for Tuberculosis, or from most of the state boards of health. It will require at least one hundred and fifty dollars to finance such an exhibit. A tuberculosis society should then be formed. The next step is to establish a dispensary with a physician and a nurse. An efficient nurse with social service training is essential for the success of the movement. After this is well under way, a day camp should be started in a modest way at a convenient locality, and, should this succeed, a night camp may be added. A day and a night camp is the beginning of a local sanatorium. This much can be done by voluntary efforts and private subscription, but at this point the authorities should take over the work.

Collateral Benefits.—The collateral benefits of a tuberculosis campaign may be even greater than the effects upon the actual control of the disease. Even if the measures used prevent only a few cases of tuberculosis, they still would be worth while, for they preach the gospel of hygiene. The problem of tuberculosis is closely interwoven with everything that pertains to human welfare.

Outlook.—The control of tuberculosis is complex and difficult. Many of the factors are not understood. The prevention of tuberculosis is no longer solely a medical problem but largely a sociological problem. It is a disease of defective society. Its eradication will, however, take time on account of the chronic nature of the disease and its widespread prevalence. We should be satisfied if we diminish the amount of tuberculosis appreciably in a generation. The momentum thus gained will increase rapidly. The time will come when the comparatively few cases left may be treated by compulsory isolation or other aggressive measures. Persistence along the lines now understood will in time control the disease, which will be the crowning achievement in preventive medicine.

REFERENCES

- The American Review of Tuberculosis*, published monthly by the National Tuberculosis Association, Baltimore, Md.
- VILLEMIN, J. A. *Études sur la tuberculose*, 1868.
- COHNHEIM, J. *Die Tuberculose vom Standpunkte der Infektionslehre*, 1879.
- KOCH, R. "Die Aetiologie der Tuberculose," *Berl. klin. Wchnschr.*, April, 1882; *Mitth. a. d. k. Gsndhtsamte*, Berlin, 1884, 2: 1.
- CORNET, G. *Ueber Tuberculose*. Die Verbreitung der Tubercelbacillen ausserhalb des Körpers, 1890.

STRAUS, I. *La Tuberculose et son bacilli*, 1895.

FLÜGGE, C. G. *Die Verbreitungsweise unter Bekämpfung der Tuberculose auf Grund experimenteller Untersuchungen im hygienischen Institut der Kgl. Universität Breslau*, 1897-1908. A collection of articles by various authors, edited by Flügge.

PIRQUET, C. "Die Allergieprobe zur Diagnose der Tuberculose im Kindesalter," *Wien. med. Wchnschr.*, 1907, 47: 1369.

DIPHTHERIA

Our knowledge of diphtheria is most satisfactory in that we know the cause of the disease and its modes of transmission; we are able to check its spread, and possess specific preventives, a precise measure of susceptibility and a curative agent of great potency.

Diphtheria spreads slowly from person to person and from community to community. It is endemic almost everywhere in the temperate zone. Newsholme points out that diphtheria epidemics and pandemics occur cyclically. The intervals between the years of epidemic prevalence vary greatly. The large American cities formerly suffered severely. In Boston diphtheria was epidemic in 1863-64, 1875-76, 1880-81, 1889-90, and 1894; in New York in 1876-78, 1880-82, 1886-88, and 1893-94; in Chicago in 1860-65, 1869-70, 1876-79-81, 1886-87, and 1890. Within recent years such epidemic outbreaks have not taken place, and the disease should never again be allowed to get out of hand. The death rate in the United States in 1923 was 12.1 per 100,000. Diphtheria in Massachusetts in 1925 was responsible for 7.9 deaths per 100,000; or a total of 329 deaths out of 4,482 cases reported in the state.

The cause of epidemic outbursts is doubtless due to a fortuitous combination of such circumstances as a new crop of susceptible children, a particularly virulent strain of the bacillus, opportunity for contact, and environmental factors favoring the spread of the infection. Just as a spark in a forest may cause a brush fire or a conflagration, depending upon the amount of plant growth, its distribution, its condition as to dryness, the direction and force of the wind, the topography and nature of the soil, and many other conditions, so diphtheria and other infections will smolder or burst into flame, depending upon similar factors.

Outbreaks in congested centers, schools, camps, on board ships, and in other crowded places, are common. Widespread epidemics have occurred in country districts. Washington died of diphtheria. In the tropics diphtheria is not a serious disease. It prevails especially in the temperate zones. Newsholme pointed out that it is more of a continental than an insular disease. The fatality from diphtheria has been greatly lowered since 1894, owing to the use of antitoxin, toxin-antitoxin immunization and to refinements of diagnosis. The case fatality rate, however, in recent years has remained about the same—6 to 7 per cent.

The Disease.—Diphtheria is a specific disease due to the Klebs-Löffler bacillus (*B. diphtheriæ*). It is characterized by a local fibrinous exudate,

usually upon the mucous membrane of the throat, but may affect other mucous membranes or wounds in any part of the body. The bacilli remain in the local lesion, where they grow and multiply. There they produce a soluble poison, the diphtheria toxin, which enters the blood and produces the general symptoms. Diphtheria is therefore a type of the toxemias. The serious nature of the disease may be due either to the local inflammation or the general toxemia. The local inflammation is characterized by coagulation necrosis, fibrinous exudate, and the formation of a membrane. This process is apparently due to the local irritating effect of the diphtheria bacillus and certain of its products, and is attended with congestion and edema. When in the larynx or trachea, death may result mechanically from suffocation. The local process is self limited as a result of tissue reaction against the bacillus and its bacillary products, whereas the toxemia is neutralized with antitoxin.

Diphtheria often begins insidiously without pain or local indications and with little or no fever. The disease may exist several days before it is noticed. Both from the standpoint of prevention and cure, early recognition is of prime importance. The *period of incubation* is from two to seven days, usually two.

Occurrence.—The maximum incidence is between five and seven years. By the time adult ages are reached about 9 per cent have had the disease. It shows no special difference for the sexes, but is more prevalent among whites than among colored. Some races are more susceptible than others.

Diphtheria reaches its maximum prevalence in the autumn of each year, which corresponds to the seasonal prevalence of scarlet fever. Diphtheria is a cold weather disease. The influence of climate is confirmed by its rare incidence in the tropics. An epidemic once established may go on regardless of season, reaching its maximum at the period of highest temperature.

In 1878 Thrushfield published papers illustrating the way in which diphtheria hung about damp houses. A damp dwelling favors sore throats and colds, and may thus open a way for invasion of the bacilli, just as any depressing influence may predispose to the infection. Children with scarlet fever or measles are especially prone to take diphtheria if the infection is around. Any abnormal condition of the mucous membrane of the throat and nose favors the localization and penetration of the diphtheria bacillus, and may thus act as a predisposing cause; therefore, sore throat, foreign bodies, adenoids, as well as dust or any irritant, may be accessory factors if the diphtheria bacillus is present. Formerly, imperfect drains and sewer gas were given as the causes of diphtheria; this is a fetish which dies hard.

Studies with the Schick reaction plainly indicate that two main conditions determine diphtheria: (1) a virulent strain of the bacillus; and (2) susceptibility.

Modes of Transmission.—The diphtheria bacillus enters by the mouth or nose, and the lesions are usually localized in the mucous membranes of the throat, nose, larynx, or upper respiratory tract. The bacillus leaves the body in the discharges from the mouth and nose. Diphtheria occasionally affects other mucous membranes or abraded surfaces, such as the conjunctiva or

vaginal mucous membrane, or open wounds; the discharges from these lesions containing the infective agent.

The bacillus may be transmitted directly from one person to another, as by kissing, or exposure to droplet infection in coughing, speaking, sneezing. The infection may be conveyed indirectly from one person to another in many ways; most common among children, perhaps, are toys, pencils, food, fingers, spoons, cups, handkerchiefs, or other objects that have been mouthed first by the infected child and then by the susceptible child. Experience points clearly to the conclusion that diphtheria is transmitted usually by a direct exchange of the flora of the nose and throat, rather than indirectly through inanimate objects. Bacillus carriers play a rôle in spreading the infection. Milk may become infected and transmit the disease. The diphtheria bacillus is frail and soon dies when dried or exposed to sunlight, therefore air-borne infection is probable only within the radius of the possibility of droplet infection.

Experience clearly teaches that diphtheria is spread mainly by the active cases; recent convalescents; mild and missed cases; and carriers.

The following description by Chapin illustrates how diphtheria and all other infections contained in the secretions from the mouth and nose may be transmitted; it also emphasizes the importance of education in personal hygiene based upon habits of biological cleanliness:

"Not only is the saliva made use of for a great variety of purposes, and numberless articles are for one reason or another placed in the mouth, but, for no reason whatever, and all unconsciously, the fingers are with great frequency raised to the lips or the nose. Who can doubt that if the salivary glands secreted indigo the fingers would not continually be stained a deep blue, and who can doubt that if the nasal and oral secretions contain the germs of disease these germs will not be almost as constantly found upon the fingers? All successful commerce is reciprocal, and in this universal trade in human saliva the fingers not only bring foreign secretions to the mouth of their owner, but there, exchanging it for his own, distribute the latter to everything that the hand touches. This happens not once, but scores and hundreds of times during the day's round of the individual. The cook spreads his saliva on the muffins and rolls, the waitress infects the glasses and spoons, the moistened fingers of the peddler arrange his fruit, the thumb of the milkman is in his measure, the reader moistens the pages of his book, the conductor his transfer tickets, the 'lady' the fingers of her glove. Everyone is busily engaged in this distribution of saliva, so that the end of each day finds this secretion freely distributed on the doors, window sills, furniture, and playthings in the home, the straps of trolley cars, the rails and counters and desks of shops and public buildings, and, indeed, upon everything that the hands of man touch. What avails it if the pathogens do die quickly? A fresh supply is furnished each day. Besides the moistening of the fingers with saliva and the use of the common drinking cup, the mouth is put to numberless improper uses which may result in the spread of infection. It is

used to hold pins, string, pencils, paper and money. The lips are used to moisten the pencil, to point the thread for the needle; to wet postage stamps and envelopes. Children 'swap' apples, cake, and lollipops, while men exchange their pipes and women their hatpins. Sometimes the mother is seen 'cleansing' the face of her child with her saliva-moistened handkerchief, and perhaps the visitor is shortly after invited to kiss the little one.

"Children have no instinct of cleanliness, and their faces, hands, toys, clothing, and everything that they touch must of necessity be continually daubed with the secretions of the nose and mouth. It is well known that children between the ages of two and eight years are more susceptible to scarlet fever, diphtheria, measles, and whooping-cough than at other ages, and it may be that one reason for this is the great opportunity that is afforded by their habits at these ages for the transfer of the secretions. Infants do not, of course, mingle freely with one another, and older children do not come in close contact in their play, and they also begin to have a little idea of cleanliness."

Domestic animals, especially cats, dogs and horses, have been suspected as sources of infection, but the evidence lacks definite foundation of fact. Savage²⁴ reëxamined the whole subject, and concluded that cats do not suffer from diphtheria. He failed to infect young kittens with cultures of the Klebs-Löffler bacillus, and found that these organisms usually disappear within twenty-four hours when implanted into the nasal cavity and upon the throat. He concludes that there is no evidence that cats serve as carriers of diphtheria. On the other hand, Major Simmons²⁵ has isolated virulent diphtheria bacilli from two cats which were pets of a person who contracted a fatal diphtheritic pharyngitis. It is quite possible that domestic animals, especially pets, may occasionally transfer the infection.

Milk-borne Diphtheria.—The diphtheria bacillus grows well in milk without appreciably changing its flavor or appearance. Trask collected twenty-three diphtheria epidemics from the literature between 1895 and 1907. Fifteen of these occurred in the United States and eight in Great Britain. Since then many other outbreaks have occurred. The milk is usually contaminated by cases or carriers occurring on the farm or at the dairy or milk shop. In some cases the diseased person milks the cows or the same person nurses the sick and handles the milk. In two instances the outbreak was supposed to be due to disease of the cow. One instance studied by Dean and Todd is instructive. In certain families supplied with milk from two cows there occurred two cases of clinically typical diphtheria and three of sore throat, whereas in another family using the milk, only after pasteurization, no case occurred. One of the cows had mammitis and furnished a scanty, ropy, semi-purulent, and slightly blood-tinged milk. The Klebs-Löffler bacilli were isolated in all cases and also from the milk of the cow with mammitis. Experiments justified the conclusion that the ulcers upon the udder of the cow

²⁴ *J. Hygiene*, 1920, 18: 448.

²⁵ *Am. J. M. Sc.*, 1920, 160: 589.

with mammitis had become secondarily infected with *B. diphtheriæ*, accidentally from some apparently healthy person. Usually, however, the milk is infected directly from a case or carrier.

The diphtheria bacillus is not killed by freezing. An outbreak at Newport, Rhode Island, and vicinity, due to ice cream, was reported by McCoy, Bolton and Bernstein.²⁶

Virulent and Avirulent Strains.—The virulent and the non-virulent strains of the diphtheria bacillus remain true to type. All attempts to exalt the pathogenicity of the avirulent strains by passage through animals have failed. The avirulent diphtheria bacilli do not produce toxin, do not kill guinea-pigs, do not produce antitoxin and seem incapable of producing immunity;²⁷ the virulent and avirulent strains look alike under the microscope. The virulent strain can therefore only be distinguished by animal tests.

The virulent bacilli may be differentiated from the avirulent strains by injecting pure cultures into guinea-pigs. Zingher and Soletsky²⁸ have improved Neisser's method by using two guinea-pigs for the testing of from four to six different strains. One guinea-pig serves as a control and receives about 200 units of antitoxin intracardially at the time of making the test, or intraperitoneally twenty-four hours before. Both pigs are injected intracutaneously with suspensions of the cultures to be tested. A fresh twenty-four-hour growth from an ordinary Löffler slant is suspended in 25 to 30 c.c. of normal salt solution; 0.1 c.c. is injected into the skin. The results of the tests are noted in twenty-four to forty-eight hours. Virulent strains produce a definite local inflammatory lesion, which shows a superficial necrosis in 48 to 72 hours. In the control pig the skin remains normal. With non-virulent strains no lesions will be found in either control or test animal.

Bacillus Carriers.—It was in the case of diphtheria that the danger of bacillus carriers was first realized. It is now known that persons who come in contact with diphtheria patients are very apt to harbor diphtheria bacilli, though they may remain in good health. It is also now well known that a certain percentage of the population at large harbor the diphtheria bacilli in their nose or throat, even though they have had no known association with the disease. Graham-Smith found that 66 per cent of the members of the family to which the diseased person belonged were infected; the proportion being higher (100 to 50 per cent) in families in which no precautions were taken to isolate the sick, and much lower (10 per cent) when such precautions were taken. Of the more distant relatives examined, 29 per cent were found to be carriers. Bacilli were found in 37 per cent of persons in attendance on the sick. Observations of the inmates of hospital wards and institutions showed that 14 per cent are likely to give positive cultures when diphtheria occurs among them. In infected schools 8.7 per cent of the pupils were found

²⁶ U. S. Pub. Health Rep., 1917, 32: 1787.

²⁷ M. J. Rosenau and G. H. Bailey, *J. Infect. Dis.*, 1925, 37: 97.

²⁸ *Proc. N. Y. Path. Soc.*, 1915, 15, Nos. 1, 2.

to be bacillus carriers. In New York, Scholley examined 1,000 children from the tenement districts, and found 18 with virulent and 38 with non-virulent bacilli. Moss, Guthrie and Gelien found *B. diphtheriae* in 3.61 per cent of 1,217 school children in Baltimore, and 3.48 per cent of 1,290 individuals in the city at large. Only 18 per cent of the positive cultures were virulent. Goldberger and Williams examined 4,093 healthy persons in Detroit in 1914 and found 0.928 per cent to harbor morphological typical bacilli, and 0.097 per cent to be carriers of virulent bacilli. Diphtheria was unduly prevalent at the time. Slack, Arms, Wade, and Blanchard took cultures at the beginning of the school year from about 4,500 pupils in the Brighton district, Boston. Diphtheria was not prevailing at the time. Nevertheless, at least 1 per cent of all these healthy school children were found to carry morphological diphtheria bacilli. In 1921, Doull and Fales examined 7,740 school children in Baltimore, at a time when the prevalence of diphtheria was no higher than normal. Of this number, 5.25 per cent were carriers of morphological and 1.75 per cent carriers of virulent diphtheria bacilli. The carriers were more numerous during the seasons of high case incidence. In the summer of 1922, McGuire and Hitchens made a study of diphtheria carriers among 1,080 young men from seventeen to twenty-one years of age, who were members of the citizens' military training camp at Camp Meade. Of this number, nine were carriers of virulent diphtheria bacilli (0.83 per cent). Schick tests were done of 833 of the men, and 55 per cent of them were found to be susceptible. Three of the men harboring virulent organisms gave a positive reaction. No case developed in the camp. The foregoing figures indicate that carriers of virulent diphtheria bacilli have been found in the general population of different parts of the country in numbers varying from one in seventy-five to one in 1,030.

Diphtheria bacilli from patients with diphtheria and from carriers who have been in contact with such patients are practically always virulent. Diphtheria is kept alive by these virulent strains in immune persons. Carriers with the avirulent strain may be disregarded. None of the children in the Brighton district above mentioned had any known association with the disease, nor did they afterward develop diphtheria. The danger of such carriers is nil. Doull and Lara estimate that the risk is ten times as great for family contacts of cases as for those in simple association with known bacillus carriers. *The dangerous carrier is he who harbors the virulent strain, and this is usually obtained from the patient, convalescent, or from a third person who has come in contact with the patient.* The danger apparently decreases the farther the carrier state is removed from the patient.

Diphtheria bacilli disappear in about 50 per cent of cases by the time the local membrane has disappeared. The bacilli persist in about 5 per cent of persons at the end of two months, about 2 per cent at the end of three months, and approximately 1 per cent continue as chronic bacillus carriers. The virulence is not lessened during the carrier condition.

It is important to remember that diphtheria bacilli may be either in the nose or throat or both. It is therefore essential to take nose as well as

throat cultures in searching for carriers. Two, three or more successive examinations will discover more carriers than one examination.

Cure of Carriers.—Many methods have been used to cure diphtheria carriers. These consist of liquid antiseptics applied as swabs, sprays or gargles; the inhalation of antiseptic vapors; the use of diphtheria vaccine, toxin, antitoxin, and antibacillary serum; also toxins of the *Bacillus pyocyaneus*; and the introduction of cultures of staphylococci, yeast and other microorganisms. Hektoen and Rappaport²⁹ suggested the use of kaolin. Good results have sometimes followed but mostly failed in all the methods so far tried.

The diphtheria bacilli probably do not long persist upon normal mucous membranes, but continue in pockets, folds, crypts of the tonsils, fissures of adenoids, spaces about the turbinates, the sinuses connected with the nasal cavity, and in any irritated, inflamed or ulcerated portion of the mucous membrane. Therefore, the removal of enlarged tonsils, polypi, foreign bodies, and other sources of irritation and inflammation sometimes results in the cure of diphtheria carriers. The first indication is to treat the mucous membrane of the upper respiratory passages so as to get it in a normal condition.

The disappearance of the bacilli from the throat and nose cannot be hastened by the usual injections of antitoxin. Diphtheria antitoxin neutralizes the toxin, but does not harm the bacilli. A serum containing agglutinins has been used. This serum in powdered form is blown into the throat. The diphtheria bacilli are supposedly agglutinated and may then be more readily washed away by gargling and douching. A substance proposed by Emmerich known as "pyocyanase" has been used. This contains a ferment from bouillon cultures of the *Bacillus pyocyaneus*. It is applied locally and acts by its power of bacteriolysis.

Encouraging results have been reported by "over-riding" the throats of diphtheria carriers with suspensions of *Staphylococcus pyogenes aureus*, which are sprayed into the throat and nose. The method was introduced by Schiotz, in 1909, who reported the prompt disappearance of diphtheria bacilli in six carriers. Page, also Catlin, Scott and Day, Lorenz and Ravenel, and others, have reported successful results.

Hewlett and Nankivell, and also Petruschky, report encouraging results in clearing up diphtheria carriers by the subcutaneous injection of a diphtheria vaccine.

We must acknowledge that all these measures often fail. The relief of bacillus carriers is one of the unsolved problems in preventive medicine.

Diagnosis.—The diagnosis of diphtheria often rests upon a combination of clinical symptoms and laboratory findings. Positive cultures alone do not necessarily mean clinical diphtheria, even though sore throat and fever be present. Cases of streptococcal tonsillitis, with follicular patches resembling false membrane, occur in diphtheria bacillus carriers. Such persons give negative Schick reactions, and do not have clinical diphtheria, despite the

²⁹ *J. Am. M. Ass.*, 1915, 64: 1991.

positive findings in cultures taken from their throats. It is important to remember that the diagnosis of clinical diphtheria cannot be made in the laboratory as a result of finding virulent diphtheria bacilli in cultures. Diphtheria often begins insidiously, and valuable time is lost in waiting for false membrane or other symptoms; cultures should be taken of all sore throats in order to exclude or confirm diphtheria.

On the other hand, negative cultures are significant in excluding diphtheria, provided the material is taken from the proper place and good technic is used.

Always give antitoxin in a clinical case of diphtheria at once, without waiting for laboratory confirmation of the diagnosis. No harm will be done and lives will be saved by this rule, for time is the important element in the life-saving properties of antitoxin. In mild cases, subcutaneous or intramuscular injection may be used, but in severe cases intravenous injection is called for.

Resistance.—The diphtheria bacillus has less resistance to adverse conditions than the majority of the spore-free bacteria. It is more readily destroyed by light, heat, and disinfecting substances than the typhoid bacillus. In this regard it corresponds more to the frailer streptococci. Under certain circumstances the diphtheria bacillus resists drying for a long time. When enclosed in the false membrane or other albuminous substances, they may remain virulent for some months. It is not killed by freezing. It is destroyed with certainty at 60° C. for twenty minutes.

IMMUNITY

Immunity to diphtheria is mainly an antitoxic immunity and persists a variable time. Second attacks sometimes occur within a few weeks while the patient is still in the hospital. Reiche³⁰ states that 5.8 per cent of 4,761 cases of diphtheria in Hamburg were known to have had a previous attack. He presents further data to confirm the absence of any lasting immunization by a single attack of diphtheria. A positive Schick reaction may be obtained in children who have recovered from the disease. Graef and Ginsberg³¹ have shown by the Schick reaction that immunity obtained by an attack of the disease lasts from a month to several years, varying greatly in different individuals, and being very brief in children. The reason why one attack of diphtheria does not necessarily protect is that the toxin produced during a non-fatal attack is not sufficient to stimulate permanent antitoxin protection. In other words, it acts very much as one injection of toxin immunization. The fact that healthy persons may harbor virulent bacilli upon their mucous membrane for a long time without contracting the disease shows the high grade immunity enjoyed by many individuals, due to antitoxin constantly in the blood. Persons vary markedly in susceptibility. During the

³⁰ *Med. Klin.*, 1913, 9: 1663.

³¹ *J. Am. M. Ass.*, 1915, 64: 1205.

first few months of life there is a very high grade immunity. Antitoxin is transmitted to the fetus through the placental circulation and to the infant in the mother's milk. This immunity during the early months of life is passive and soon wears off, so that by the end of two years 70 per cent are susceptible. After this period, children begin to manufacture their own diphtheria antitoxin, so that the percentage of susceptible children gradually decreases until the twentieth year, when only 12 per cent give a positive Schick reaction; in other words, 88 per cent of the adult population are immune to diphtheria.

The susceptibility and mortality with reference to age are shown in the following table, based on Park's extensive studies.

Age	SUSCEPTIBILITY, BASED ON 20,000 SCHICK TESTS		MORTALITY, NEW YORK CITY, 1891-1900	
	Average Susceptible, per Cent	Range Schick Positive	Age	Per Cent
At birth	10	0 to 15	Under 6 months	3.0
Under 4 months	15	0 to 15	6 months to 1 year ..	6.0
4 to 6 months	30	20 to 30	1 to 2 years	23.0
6 to 9 "	60	60 to 74	2 to 3 "	21.2
9 to 1 year	75	65 to 75	3 to 4 "	16.1
1 to 2 years	75	60 to 76	4 to 5 "	10.6
2 to 3 "	65	50 to 70	Under 5 "	81.5
3 to 5 "	40	15 to 50	5 to 10 "	17.0
5 to 10 "	30	8 to 40	10 to 15 "	1.3
10 to 20 "	25	5 to 40	Over 15 "	0.2
Over 20 "	20	5 to 42		

Passive Immunity.—This can be induced by injecting antitoxin. The usual immunizing dose is 1,000 to 1,500 units for adults and 750 to 1,000 units for children. The protection thus afforded disappears in three to eight weeks. A second injection of antitoxin renews the immunity, which, however, lasts a still shorter time. Passive immunity, while occasionally of great practical service in individual prophylaxis, cannot be depended upon to control epidemics.

The great advantage of passive immunity is that it is prompt; the disadvantages are that it lasts a brief time, produces serum reactions, and is relatively much more expensive than active immunity.

Active Immunity.—This can be induced by injecting a mixture of toxin and antitoxin.³² It is the toxin and not the antitoxin in the mixture that induces active immunity. The antitoxin is added only to subdue the irritating action of the toxin.³³ The toxin and antitoxin are mixed so that the toxin is not quite neutralized; in other words, there is some free poison in the mixture.

³²This method was suggested by Theobald Smith, but first attempted by Behring in 1912. It has been studied especially by Park and Zingher of the New York City Department of Health.

³³Other methods of modifying the toxin are used, such as the anatoxin of Ramon, or toxoid which is toxin detoxicated with formaldehyd.

Five c.c. of this mixture injected into guinea-pigs does not produce acute symptoms of toxic irritation and poisoning, the result of *toxin*, but causes late paralysis, which begins about the tenth day, the result of *toxon*. The mixture is diluted so that one-tenth of an L₄ dose of toxin partially neutralized is contained in each c.c.

The dose of toxin-antitoxin mixture is 1 c.c. subcutaneously, repeated every week, until three injections are given. Children react less than adults and therefore can be given a full dose. The immunity appears slowly, sometimes in eight to twelve weeks, but once established seems lasting. Park finds that children immunized ten years ago still have antitoxin in their blood as demonstrated by negative Schick reactions. The three injections immunize from 75 to 90 per cent of all Schick positive children. A certain percentage will show a positive Schick reaction at the end of six months, after three injections of the toxin-antitoxin mixture. A second series of injections will develop an active immunity in almost all susceptible persons.

An immunizing dose of antitoxin interferes with the production of an active immunity with toxin-antitoxin mixtures. In other words, an over-neutralized toxin delays or prevents the stimulation of antibody formation.

By actively immunizing all children in the preschool period, we can render the child population immune to diphtheria, and tide it over the period of greatest susceptibility. It should be remembered that in adult life the great majority (70 to 90 and *even 95 per cent*) are naturally immune. The percentage is higher in urban than rural districts, and among some groups than others.

The advantages of active immunity, produced by injecting toxin-antitoxin mixture, are that it is lasting, cheap and simple, and the reactions are slight. Its only disadvantage is that it takes several weeks or months to appear, and this is not important except in the case of youthful contacts.

The Schick Reaction.—The presence or absence of immunity in any individual may readily be determined by the Schick test, which tells whether antitoxin is present or absent in the blood of that individual. The Schick reaction is made by injecting a small amount of diphtheria toxin *into the skin*. The precise quantity used is one-fiftieth of a minimum lethal dose of diphtheria toxin for a 250-gram guinea-pig. This amount of toxin is diluted so that it is contained in 0.1, sometimes 0.2 c.c. of salt solution. The injection must be made *into* and not under the skin. A positive reaction at the site of the injection means absence of antitoxin, that is, a susceptible individual. A negative reaction means the presence of antitoxin, hence immunity.

A *positive* reaction is indicated by redness at the site of injection which appears gradually and becomes distinct in twenty-four to forty-eight hours, reaching its height on the third or fourth day. The reaction then consists of a circumscribed area of redness and slight infiltration, about one to two centimeters in diameter. The degree of redness and infiltration varies with the relative susceptibility of the individual. The reaction slowly disappears leaving a definite circumscribed scaling area of brownish pigmentation, which

persists three to six weeks. A positive reaction represents the effect of an irritating poison upon tissue cells that are not protected by antitoxin. The reaction is sufficiently delicate to indicate less than one-thirtieth of a unit of antitoxin in each cubic centimeter of blood serum. Such persons are susceptible to diphtheria.

The intensity of the reaction varies. A well-marked redness indicates an almost complete absence of antitoxin. Faint reactions point to the presence of very small amounts of antitoxin. All gradations are observed.

In a *negative* reaction, the skin at the site of injection remains normal. The toxin is neutralized by the antitoxin, and therefore causes no irritation. Such persons are immune and need not fear diphtheria. A negative reaction in a child that has reached the age of three years indicates an immunity that is probably permanent. No authentic instance has yet been reported of an individual with a negative reaction contracting diphtheria, even though exposed to the disease, or after becoming a carrier of virulent diphtheria bacilli. Susceptible children sometimes give a false negative because the material injected runs out or through some other fault in technic.

A *pseudoreaction* represents a local anaphylactic response to proteins in the material injected. This reaction is urticarial, appears within six to eighteen hours, reaches its height in thirty-six to forty-eight hours, and disappears on the third or fourth day, when the true reaction is at its height. Control tests should therefore always be made. Pseudoreactions occur only in older children and adults, not in infants.

Combined reactions also occur, that is pseudo and true at the same time. The rule is to retest all doubtful cases or to play on the side of safety by considering them positive reactions.

Control tests consist in injecting toxin heated to 75° C. for five minutes. The reaction at the site of the control will help interpret the reaction at the site of the test injection. The accuracy of the Schick test depends upon the strength of the toxin, the technic of the test, and the interpretation of the reaction.

We therefore possess a ready method of determining who is susceptible and who is naturally immune. Persons who react negatively to the Schick test will not develop diphtheria.

PREVENTION—THE CONTROL OF THE OUTBREAKS

Diphtheria frequently appears in asylums, hospitals, camps, jails, on ship-board, and similar places. Under these conditions of crowding, the disease has a highly contagious tendency. It may, however, be controlled with every assurance of success by the application of well-tried measures.

The most important measure to suppress diphtheria is to isolate all cases and all carriers and immunize the susceptible. Isolation is easier in an institution, or limited compound, than in the population at large. The isolation of both cases and carriers is the first of our preventive measures. The most

radical is the active immunization of all susceptible individuals with toxin-antitoxin mixture.

The following measures are recommended for the control of an outbreak: (1) the recognition and isolation of cases; (2) the finding and quarantining of carriers; (3) the discovery of the susceptible by means of the Schick test; (4) active immunization of all susceptible persons; (5) inspection of all susceptible persons every twelve or twenty-four hours, and the use of anti-toxin upon the first appearance of symptoms; (6) epidemiological control, with disinfection, laboratory facilities, and other administrative measures.

Recognition and Isolation of Cases.—These are the immediate demands. It should be remembered that diphtheria often comes on insidiously and that the patient may have his throat plastered with false membrane of three or four days' standing before feeling ill enough to attract attention. Early recognition of the disease is important both for successful treatment and prevention. In all endemic centers the examination of the throat, especially of children, should be made a routine procedure by practicing physicians. In outbreaks a daily inspection and taking of temperatures morning and evening will detect cases early.

In hospitals, cases may be isolated in cubicles, or separated by sheets. Gauze masks may also be used. Careful nursing technic is essential to prevent cross-infections.

Convalescents should not be released until at least two cultures taken from both the nose and throat are negative.

Finding and Quarantining Carriers.—Throat and nose cultures from all exposed persons and from all persons in an institutional epidemic should be made. All positive carriers based upon morphologic diagnosis should be quarantined. At a later date, when time permits, the carriers of avirulent bacilli may be released. It is sometimes necessary to go through a camp or school twice, thrice, or oftener to detect all the carriers, or to discover new ones.

While it is practical to isolate all the carriers in a limited outbreak, such as a camp, ship, or asylum, this is not always feasible in the community at large. Yet most of the dangerous carriers even in a metropolitan city can be discovered by taking cultures from all persons closely associated with cases.

The bacilli frequently grow in the mucous membrane of the nose and nasopharynx without anything to indicate their localization. Unless cultures are taken from the nose, many carriers will be overlooked, leaving a large loophole in our preventive measures. Ward and Henderson, in a public school epidemic in Berkeley in 1907, found that all attempts to isolate infected children had no effect on the epidemic so long as they made throat cultures alone. When they took both nose and throat cultures and quarantined all the children showing positive cultures, the epidemic stopped.

Good results can be obtained only where care is exercised in obtaining the material and skill used in the technic of the bacteriological examination.

Diphtheria bacilli sometimes leave the body in the discharges from middle ear disease, and occasionally also from infected wounds.

Schick Reaction.—As soon as practical, all those exposed to the infection, whether in a family or in an epidemic focus, should be tested for immunity by the Schick test. In an institution, both the inmates and the administrative force should be included. No special concern need be given those who react negatively, for they are immune and will not contract the disease. Such persons, however, may be carriers of virulent strains, in which case they should be quarantined.

For the population at large, the best time to test susceptibility with the Schick reaction is the preschool age. Between nine months and two years the percentage of positive Schick reactions is largest, and the susceptibility to diphtheria as well as the mortality from the disease is greatest. All those showing positive reactions at this period should be given active immunization.

Active Immunization.—All those who show a positive Schick reaction should at once be given the toxin-antitoxin mixtures. Three successive injections of 1 c.c. at intervals of a week are given. The immunity comes on slowly (two to twelve weeks). During an attack such individuals should therefore be carefully inspected every day, so that the first indications of infection may be met with an injection of antitoxin. Under this system, cases of the disease may arise during the time it takes these persons to develop an active immunity, but with the timely use of antitoxin the mortality will be reduced practically to nil.

This is the most effective method of eradicating diphtheria, both in endemic centers as well as in epidemic foci. The results obtained are permanent. It requires time, and very careful supervision of all the details, with a corps of skilled and trained technicians, to carry it out successfully.

An immunizing dose of antitoxin should not be used to tide over the interval it takes for toxin-antitoxin immunity to become established, because this interferes with the production of active immunity. In other words, the methods of active and passive immunization should not be used at the same time.

Immunization of Children.—Zingher³⁴ recommends the following procedures for the protection of children: (1) All children over eighteen months of age in the entire community should have their susceptibility to diphtheria determined by means of the Schick test, and the reaction which they show should be noted either in institutional records or in the records of the family physician. (2) In infants below eighteen months of age, the Schick test is not necessary because a negative reaction may give rise to a false sense of security. Very young infants may exhibit a negative Schick test owing to the immunity passively acquired from the mother, but inasmuch as this type of immunity is transient, it is safer to assume that no child under eighteen months possesses permanent immunity. (3) All infants below eighteen months of age accordingly should be actively immunized with three doses of 1 c.c. each of diphtheria toxin-antitoxin mixture, irrespective of the reaction

³⁴ Reprint No. 72, Dept. of Health, New York City, Nov., 1918.

to the Schick test which the infant might show at the time of immunization. (4) All children over eighteen months of age who give a positive Schick test should be immunized by receiving three subcutaneous injections of 1 c.c. each of toxin-antitoxin at intervals of seven days. (5) All children immunized by this method should be retested three to six months after the last injection and reimmunized if they should by any chance still give a positive Schick reaction.

The most important and most difficult of all children to reach is the child of preschool age. That this can be done has been shown by Sears in Auburn, New York, where the disease has been practically controlled. The attack was focused upon testing the susceptibility of children with the Schick test and immunizing all those found to be susceptible with the toxin-antitoxin mixture. This campaign, energetically carried out, was a commendable achievement.

The tendency is developing to give the toxin-antitoxin mixture without a preliminary Schick test to all children under ten years of age. Six months after the toxin-antitoxin mixture, all persons should be Schick tested in order to determine whether they are immune.

Passive Immunization.—This consists in injecting diphtheria antitoxin. The customary immunizing dose is 100 units. Schick recommends 50 units per kilogram of body weight. The protection wears off in three to eight weeks, but the immunizing dose can be repeated every two or three weeks until the danger is passed.

Antitoxic immunity cannot be depended upon to stamp out the infection. It has several disadvantages that should not be disregarded. The bacilli remain in the throats of those immunized and the disease continues to crop out from time to time as the antitoxin disappears. Where large numbers are involved the method is very expensive, time-consuming, and the resulting serum reactions often disturbing, especially where repeated immunization is called for. A blind reliance upon antitoxic immunity has proved disappointing and futile in many institutional outbreaks. In the end, the active immunity induced by toxin-antitoxin mixtures does not take much more time and is much more certain.

Antitoxic immunity has a distinct place in personal prophylaxis and in selected individual cases, especially in family practice. Youthful contacts present a special problem. Young children are difficult to examine and diagnose. Further, they not only have a greater liability to the disease, but there is also the danger of laryngeal localization. It is therefore clear that such young children should be given an immunizing dose of diphtheria antitoxin to tide them over the period of incubation. After the danger has passed and the passive immunity has worn off, if Schick positive they should be actively immunized with toxin-antitoxin mixture. In some hospitals for communicable diseases, cases of scarlet fever, if Schick positive, are given immunizing doses of diphtheria antitoxin because such cases are apt to be complicated with diphtheria.

Prevention of Postdiphtheritic Paralysis.—It has been observed that postdiphtheritic paralysis is more frequent since the use of antitoxin than before the days of serum therapy. This is due to the fact that many cases now recover that would formerly have died. It is also due to the fact that diphtheria antitoxin is sometimes used too late, thus neutralizing only the acute effects of the *toxin*, but not neutralizing the after-effects of the *toxon* which acts specifically upon the nerves. The prevention of postdiphtheritic paralysis, therefore, consists in giving sufficient amounts of antitoxin *early* in the disease. The antitoxin does not influence the paralysis after it has once appeared.

Disinfection.—Disinfection has a place in controlling the spread of the infection. The discharges from the patient should be burned. All fabrics, bed and body linen, that have come in contact with the discharges from the mouth and nose, and all objects such as spoons, cups, thermometers, toys, etc., that have been mouthed should be boiled, steamed or soaked in one of the standard germicidal solutions. The hands of the nurse need attention and proper nursing technic should be carried out.

Terminal fumigation is of little avail. A special cleansing and disinfection of floors, walls, door knobs, bed frames, and other surfaces that have been contaminated will suffice. The germicidal solutions available are bi-chlorid of mercury, 1:1000; carbolic acid, 2.5 per cent; 10 per cent formaldehyd solution; or liquor cresolis compositus, 1 per cent. They should be used hot.

Administrative Measures.—Diphtheria requires a well-trained force familiar with the modern problem and skilled in the technical sides of diagnosis and the Schick reaction. Laboratory facilities are indispensable. With an adequate force, including an experienced epidemiologist, diphtheria should never get out of hand in any community, and epidemics should be promptly controlled. Education of the public and profession is part of the program. Personal hygiene and sanitary habits must be taught and encouraged. Care should be taken that the infection is not spread by tableware, handkerchiefs, wash bowls, and articles in common use. Better sanitary control over restaurants and soda water fountains should be maintained. All tableware should be scalded before it is again used. Sanitary habits regarding "hand to mouth infection" and measures to "screen the sneeze" need emphasis in nursery, schools and workshops.

In almost all communities diphtheria is now one of the diseases which must be reported to the health authorities. The houses are placarded and the cases isolated. There is no great objection to treating a case of diphtheria in the household provided the patient and the nurse may also be quarantined from the rest of the household. Under these circumstances and with intelligent care and disinfection at the bedside there is little danger to the rest of the family; but the great menace that some of the members of the family will become bacillus carriers of a dangerous type makes it advisable to treat all cases of diphtheria in a special hospital.

All outbreaks should of course be investigated as to their source. The possibility of milk-borne infection is usually evident. In any case, milk should be pasteurized and care taken to prevent infection in the kitchen or in serving and handling food.

Schools need not be closed during an epidemic of diphtheria; in fact, better results will be achieved by daily inspection, and examination of cultures from the nose and throat of each pupil from time to time. The well children of a household, where a person is ill with diphtheria, should be excluded from school until one week has expired from the date of the last exposure, unless showing two negative cultures from throat and nose. All other members of the household may be allowed to continue their usual occupations, except those who are engaged in the handling of milk.

Responsibility for Diphtheria Deaths.—People still die of diphtheria—usually because the diagnosis is not made early and because diphtheria antitoxin is not given in time.

Diphtheria antitoxin is a specific and sovereign remedy. When given in sufficient amounts during the first twenty-four hours of the disease it reduces the mortality to practically nil. Upon the first appearance of sore throat, fever, or other suggestive symptoms in persons who are exposed to diphtheria, a full dose of 3,000 to 10,000 units should be administered subcutaneously without delay. It is absorbed more quickly when given intramuscularly. In very toxic cases, or for late use, it acts most quickly when given intravenously. In order to obtain the full life-saving benefits of diphtheria antitoxin, it should be given early in the disease. Time is the most important factor. When the damage to the cells has been done, it may be too late. It is not always advisable to wait for bacterial confirmation.

Many unnecessary deaths from diphtheria occur. In New York City alone, over 1,000 deaths occurred annually, approximately twenty per 100,000 of population. Similar rates prevailed in Rhode Island, Pennsylvania, Kentucky, North Carolina, Massachusetts, Michigan, and elsewhere. Wherever the disease is attacked energetically with the immunization of the child population, diphtheria is being controlled. The classic demonstration of Sears, at Auburn, shows what may be done.

In a study of 1,000 deaths made by Carey³⁵ of the Massachusetts State Department of Public Health, it is shown that the useful knowledge and facilities of dealing with diphtheria are still utilized far too little. In 23.1 per cent of the cases, the patient was ill a week before the physician was called. In 4.2 per cent, the patients had been ill from one to two weeks before they received attention. In 7.6 per cent of the deaths, the disease was not recognized during life. In a number of fatal cases, the physician delayed antitoxin treatment by waiting for laboratory confirmation of the diagnosis. In not a single instance was the antitoxin given intravenously. A similar situation was found

³⁵ *Boston M. & S. J.*, 1919, 180: 67.

in New York City—"with a diagnostic laboratory service unsurpassed, with Schick test outfits, antitoxic serum, and active immunization outfits practically at their elbow, the physicians of New York were charged with insufficient or delayed utilization of these aids and with responsibility of continued prevalence of fatal cases of diphtheria." Every death from diphtheria should be investigated and the responsible party brought to task. The facts indicate the further need of education of both the profession and the public, in order to save lives from this and other preventable infections.

Historical Note and References.—A complete summary and bibliography of diphtheria up to 1908 will be found in the system edited by Nuttall and Graham-Smith entitled *The Bacteriology of Diphtheria*, containing articles by Löffler, Newsholme, Mallory, Graham-Smith, Dean, Park, and Bolduan; Cambridge University Press, 1908.

The modern clinical description of the disease is, by common assent, attributed to Bretonneau, of Tours, in 1826: *Traité de la diphthérie. Des inflammations spéciales du tissu muqueux et en particulier de la diphthérie ou inflammation pelliculaire, connue sous le nom de croup, d'angine maligne, d'angine gangréneuse, etc.*, Paris.

The bacillus of diphtheria was first cultivated and adequately described by Löffler, 1884: "Untersuchungen über die Bedeutung der Mikroorganismen für die Entstehung der Diphtherie beim Menschen, bei der Taube und beim Kalbe," *Mittheilungen aus der königlichen Gesundheitsamte*, 2: 451.

The classical article in which Behring and Kitasato announced their discovery of diphtheria antitoxin in 1890 will be found in *Deutsche medizinische Wochenschrift* 16: 1113 ("Ueber das Zustandekommen der Diphtherieimmunität und die Tetanusimmunität bei Tieren").

Ehrlich's important work, in which he laid the foundations of his side-chain theory and established the present satisfactory method of standardizing diphtheria antitoxin, will be found in the following: "Die Werthbemessung des Diphtherieheilserums und deren theoretische Grundlagen," *Klinisches Jahrbuch*, Jena, v, 6 (2) 1897, pp. 299-326. "Über die Constitution des Diphtheriegiftes," *Deutsche medizinische Wochenschrift*, Leipzig, v, 23 (38) 1898, pp. 597-600. Croonian Lecture, "On Immunity with Special Reference to Cell Life," *Proceedings of the Royal Society of London*, v, 66, pp. 424-448, pls. 6, 7.

The official method for standardizing diphtheria antitoxin in this country and the principle upon which it is based are described by Rosenau (1905), "The Immunity Unit for Standardizing Diphtheria Antitoxin" (based on Ehrlich's normal serum), *Hygienic Laboratory Bulletin*, No. 21, P. H. and M. H. S., Washington, Government Printing Office, 92 pp.

The Schick test was first described in the *Münchener medizinische Wochenschrift*, Nov. 25, 1913, p. 2608.

PREVENTION OF SERUM SICKNESS AND ANAPHYLACTIC SHOCK

This subject may appropriately be considered here, although it is a condition that may follow the injection of any alien protein into the system. Serum sickness is a syndrome which frequently follows the injection of horse serum into man.³⁶ The symptoms usually come on about eight or ten days following the injection. The period of incubation is shorter if the person had a previous injection of homologous serum. They consist of various skin eruptions, usually urticarial or erythematous in character; also fever, vomiting, edema, glandular and splenic enlargements, rheumatic-like pains in the joints and muscles; and albuminuria. The eruptions may be either local or general, and sometimes resemble those of scarlet fever or measles. Serum sickness has nothing to do with the antitoxin, but is caused entirely by the foreign proteins contained in the horse serum. It may be produced with normal horse serum as well as with antitoxic horse serum. The studies upon anaphylaxis have thrown much light upon the nature of this complication. The occurrence and severity of the symptoms depend upon the amount of foreign protein injected and the sensitiveness of the individual. If concentrated antitoxic serum is used, the reactions are correspondingly lessened because smaller quantities of the foreign protein are injected, the albumins and certain other proteins having been eliminated by the partial purification. The concentrated antitoxin contains mainly serum-globulin.

Under certain circumstances, however, anaphylactic shock develops a few moments after the injection and may be fatal. H. F. Gillette collected twenty-eight cases of collapse after serum injection, of which fifteen died. Rosenau and Anderson have collected some nineteen cases of sudden death following the injection of horse serum, and they know of more instances which have not appeared in the literature. This unusual and serious complication comes on within a few minutes of the injection, and is characterized by collapse, unconsciousness, cyanosis, labored respiration, and edema. The heart continues to beat after respiration has ceased. The entire picture is an exact counterpart of the anaphylactic shock so readily reproduced by a second injection of horse serum or other foreign protein in the guinea-pig. Contrary to the experimental work on the lower animals, the cases of sudden death in man follow the first injection of horse serum. The serious symptoms and death in these cases are not due to any inherent poisonous property in the antitoxic serum, but result entirely from a hypersusceptibility of the individual. Just how man becomes sensitized in these cases is not always clear. It may be by a previous injection of horse serum, or by eating horse meat, or by the introduction of small amounts of horse protein through wounds of the skin, or through the respiratory tract; finally, hereditary transmission may account for the susceptibility.

³⁶ Pirquet and Schick. *Serum Krankheit*, Wien, 1905; also, Rosenau and Anderson. *U. S. Hyg. Lab. Bull.*, Nos. 29, 36, 45.

Most of the cases occur in healthy persons who give a history of asthma or discomfort when about horses. This is a practical and important point, and should be inquired into before horse serum of any kind is injected. Horse serum should not be injected into such individuals unless the indications are clear, and then only with a statement as to the possible outcome. Bovine serum may be used and after desensitization.

Diphtheria antitoxin may be given without fear of anaphylactic shock in cases of diphtheria. The few serious accidents have occurred only with prophylactic doses in healthy persons. There is no instance on record of diphtheria antitoxin causing fatal anaphylactic shock in a person ill with diphtheria. We now have an experimental confirmation of this, for Bronfenbrenner,³⁷ working in my laboratory, has shown that diphtheria toxin prevents anaphylactic shock in sensitized guinea-pigs. *It is therefore inexcusable to delay or hesitate to use full therapeutic doses of antitoxin in diphtheria.*

Desensitization.—In order to prevent anaphylactic shock, the following precautions are suggested:

1. Except in urgent cases, avoid injecting horse serum into individuals known to be asthmatic, especially those in whom symptoms are brought on by being around horses.

2. If hypersensitiveness is suspected, give at first a very small amount of serum subcutaneously, following it in an hour or so with the rest, injecting it exceedingly slowly and avoiding direct injection into the circulation. Sometimes an intradermal injection is given to determine sensitization; this is followed by small desensitizing doses of the serum at intervals of an hour. There is no necessary correlation between skin hypersensitiveness and general anaphylaxis.

3. In persons known or suspected of being hypersusceptible to horse serum, bovine antitoxin may be used.

It is good practice to inject 0.5 c.c. to 1 c.c. subcutaneously an hour before giving intravenous injections. Intravenous injections should always be given very slowly.

Adrenalin, pituitrin, chloral, chloroform, and atropin in full therapeutic amounts are claimed to ameliorate or even prevent anaphylactic shock, but it must be admitted that none of the above procedures is wholly satisfactory.

VINCENT'S ANGINA

Vincent's angina was first described by Professor Vincent in 1898. It is an important disease in itself, and also because it is very likely to be mistaken for diphtheria. Vincent's angina is due to a fusiform bacillus and an accompanying spirillum (spirochete?). In early cases the bacilli are usually more numerous than the spirilla, but in more advanced cases the spirilla usually predominate. The organisms are best stained with carbolfuchsin or gentian violet. Both the spirilla and the bacilli are usually Gram-negative.

³⁷ *Proc. Soc. Exper. Biol. & Med.*, 1921, 18: 147.

They have been grown by Tunnicliff³⁸ anaerobically in ascitic broth containing a piece of tissue. She regards them as identical for the reason that the spirillum appeared to develop from a pure culture of the fusiform bacillus.

The disease is characterized by slight constitutional disturbance and no fever; the temperature rarely goes over 100° F.; there is pain on swallowing; the submaxillary lymphatic glands are enlarged and tender, the lesions are often unilateral; the yellowish-gray membranous exudate is usually easily removed, leaving a raw, bleeding surface; albumin rarely appears in the urine.

The fusiform bacillus and the long spirillum were first demonstrated in cases of ulceromembranous angina, and later found in cases of ulceromembranous stomatitis; also in gingivitis, noma, hospital gangrene, pyorrhea alveolaris, appendicitis, abscesses, and other morbid processes. Noguchi found these organisms in a case of ulcer of the labia, and Vincent himself reported a case of gastro-enteritis in which large numbers of typical organisms were found. Corbus and Harris have described ulcerative balanitis due to this organism, and they called it "the fourth venereal disease."

This infection is peculiar to man; at least, experiments on animals are negative. Second attacks and recurrent attacks occur, indicating that there is little or no immunity conferred. Local applications of arsphenamine appear useful, both in treatment and for prophylaxis.

Vincent's angina is often mistaken for diphtheria. It must be differentiated from syphilis. The disease is much more common than is ordinarily supposed. Under military conditions the incidence is greatly increased, and it was a common cause of disability in the World War. The disease is favored by any debilitating condition, such as fatigue, chill, exposure, insufficient and improper food, and excessive use of alcohol and tobacco.

The organisms are found in normal mouths, especially in those with poor teeth and lack of oral hygiene. Campbell and Dyas found a few Vincent's organisms in about 50 per cent of all swabs taken from the throats of troops at Bramshott. Reckford and Baker³⁹ found only one carrier in fifty normal individuals, whereas fusiform bacilli and spirilla were found in 90 per cent of the smears from diseased teeth in a dental clinic.

The disease is spread through the discharges from the lesions and by carriers in the same ways that diphtheria is spread. The prophylaxis is similar; special attention must be given to predisposing factors, such as undernutrition, to oral hygiene, and to measures that improve health.

MEASLES

(*Morbilli, Rubeola*)

Measles is usually taken as the type of a contagious disease because it is one of the most readily communicable of all diseases, in this regard ranking

³⁸ *J. Infect. Dis.*, 1906, 3: 148.

³⁹ *J. Am. M. Ass.*, 1920, 75: 1620.

with smallpox and pandemic influenza. As a cause of death it ranks high among the acute fevers of children.

In the registration area of the United States, during the twelve years from 1900-1911, 50,000 deaths from measles were recorded, and it is estimated that over 100,000 deaths were caused by measles in the continental United States during the same period. There were 68,466 deaths from measles in the United States registration area for the period 1912-1922. The number of deaths from measles as compared with those of certain other diseases in the registration area during the years 1910, 1917 and 1922 is shown in the following table:

Disease	1910		1917		1922	
	Deaths	Deaths per 100,000 Population	Deaths	Deaths per 100,000 Population	Deaths	Deaths per 100,000 Population
Diphtheria and croup	11,512	21.4	12,453	16.5	13,659	14.7
Measles	6,598	12.3	10,745	14.3	4,042	4.3
Scarlet fever	6,255	11.6	3,141	4.2	3,256	3.5
Whooping-cough	6,148	11.4	7,837	10.4	5,220	5.6
Cerebrospinal meningitis...	2,272	4.2	6,890	9.1
Infantile paralysis	1,459	2.7	790	.85

It is estimated that 10,000 deaths from measles take place each year in the United States. These figures are conservative, for many deaths from measles are recorded as pneumonia. The measles death rate per 100,000 population in the registration area of the United States varies from 4.3 to 14.3 in different years.

Measles is an infection peculiar to man. Experimental measles with Koplik spots has been produced in monkeys, but the susceptibility is not marked and subject to variations. The virus is contained in the blood, as has been shown by Hektoen, who thus transmitted the disease from man to man. More important from the standpoint of prevention, the virus has been demonstrated in the secretions from the nose and mouth by Anderson and Goldberger. The period of incubation is quite constant (from nine to eleven days), and the rash appears quite uniformly on the thirteenth or fourteenth day after the infection. In Hektoen's two experimental cases the eruption appeared on the fourteenth day. The cause of measles is not established. Tunncliffe⁴⁰ found that monkeys, guinea-pigs and rabbits were susceptible to measles when nasopharyngeal washings were introduced, and that the same symptoms and lesions might be produced in these animals by green-producing diplococci. Ferry and Fisher⁴¹ have described an aerobic green-producing streptococcus which they state produces a soluble toxin specific to measles.

Measles is highly contagious during the præruptive stage, when the nature of the disease is not recognized and when most of the damage is done; it

⁴⁰ *J. Infect. Dis.*, 1925, 37: 193.

⁴¹ *J. Am. M. Ass.*, 1926, 86: 932.

remains contagious for a variable time during convalescence. Recent experimental evidence and clinical experience plainly indicate that the infection of measles soon dies out, and that there is little danger of transmitting the disease after the temperature returns to normal. An isolation of two weeks from the onset of the disease is sufficient in public health work; health officers, however, adopt arbitrary times. For public health purposes the maximum period of incubation is placed at fourteen days.

Measles is malignant in virgin soil. An increased virulence is also observed wherever many cases of measles are brought together. This is explained on the theory of cross-infection with the chief complications—streptococci and pneumococci pneumonia.

Occurrence.—Measles is more or less constantly present in all large cities in the temperate zone; it is less common in the tropics, although it spreads as readily in hot as in cold climates. All races are susceptible; the death-rate is higher in urban than in rural districts.⁴² It is also higher in the north than in the south. Measles frequently becomes epidemic, usually in the cooler months, in this respect resembling influenza, scarlet fever and smallpox. Its greatest prevalence in our climate is in the spring months. The maximum incidence of measles occurs at six to seven years of age. It is usually the six-year-old child that brings measles into the house. The disease is somewhat more prevalent among girls than boys, among the native white than among foreign children, and among white than colored.

Measles is common in army camps, especially among troops enlisted from country districts, who are thus exposed to the infection for the first time. In the World War, measles was a serious cause of disability in our mobilization and training camps. Pneumonia was the common complication, often resulting fatally.

Measles itself is rarely fatal—95 per cent of the deaths are due to pneumonic infection. In this regard, influenza and measles are alike. Both diseases become malignant when introduced into a population where they have not prevailed for a long time. Measles lowers the resistance to diphtheria, pneumonia, streptococci, tuberculosis and noma; it is a common history for tuberculosis to develop after an attack of measles.

Periodicity.—The epidemics recur cyclically, at intervals of two or three years, with considerable regularity in thickly settled communities. In more sparsely populated areas, these waves recur at longer and more irregular intervals. Levy and Foster noticed that in Richmond, Virginia, epidemic outbreaks recurred at intervals of about three years. They were able to predict and warn against an epidemic prevalence of the disease in the winter of 1910. During 1909, forty cases of measles occurred in Richmond, but during this year the disease showed no special tendency to spread. In the middle of February, 1910, eight cases occurred among the pupils of one school and the infection showed a high degree of communicability. According to the

⁴² F. S. Crum, *Am. J. Pub. Health*, 1914, 4: 289.

history of the disease, an epidemic year was due and an epidemic was predicted. Over 2,000 cases occurred with twenty-six deaths. Chapin found a periodicity of two years in eleven well-defined outbreaks in Providence in the last twenty-five years.

Panum's Classic Studies.—Very little has been added to our knowledge of measles since the admirable report of Panum,⁴³ who was sent to the Faroe Islands in 1846 as a member of a medical commission to study an outbreak of measles. Measles had disappeared from the Faroe Islands in 1781, so that they were free of the disease for a period of sixty-five years when the epidemic of 1846 occurred. It was brought into the Islands by a cabinet maker, who left Copenhagen March 20 and reached Thorshaven on March 28 in good health. A few days before leaving Copenhagen he had come in contact with a case of measles, and on the fourteenth day thereafter, and some days after reaching Thorshaven he developed the disease.

Panum demonstrated that the period of incubation of the disease is almost invariably fourteen days; that all ages are susceptible; that one attack produces a definite immunity; that the disease is most readily transmitted at the time of the development of the eruption, but it may be transmitted during the prodromal period and before the appearance of the rash, but in no instance did he find that it was transmitted during the period of desquamation.

Panum found that out of 7,782 inhabitants of the Faroe Islands in 1846, 6,000 had measles and no one who was exposed escaped the infection, except those who had had the disease in 1781 or previously.

Immunity.—One attack of measles confers a definite protection against subsequent attacks; second attacks, however, are more commonly reported than in the other eruptive fevers. Some persons are said to have the disease three or four times.⁴⁴ In a study of 14,744 cases in Providence, Chapin⁴⁵ found 689 or 4.8 per cent who had previously had the disease, and of these, 41 had had it more than twice. The close similarity between rubella (German measles) and rubeola (measles) accounts for many so-called second attacks of measles. As with smallpox, there appears to be no natural immunity to measles—man is exquisitely susceptible to these two infections. Only about 3 per cent escape the disease. There usually is a relative immunity sometimes of a high grade during the first few months of life, although measles occasionally occurs in infants of a month or six weeks. We now know that this is a passive immunity which the baby gets from its mother's blood and milk.

Adults are susceptible to measles, provided they have not had a previous attack. Susceptibility to the infection does not diminish with increasing age; the disease is apparently one of childhood only on account of the chances

⁴³ *Virchow's Arch.*, 1847, 1: 492. This is a condensed summary by P. L. Panum. His original report is entitled "Tagttagelser, anstillede under Maeslinge-Epidemien paa Faroerne i Aaret 1846," *Bibliot. f. Læger*, Copenhagen, 1847, pp. 270-344.

⁴⁴ Wagener (*Monshrift für Kinderheilkunde*, 12: 477) describes mild catarrhal attacks during epidemics in adults who have had measles, and believes these to be atypical cases. He states that the disease is spread by these missed cases.

⁴⁵ *Am. J. Hyg.*, 1925, 5, 635.

of exposure in early life. Before the days of vaccination smallpox was also a disease mainly of childhood.

The following instances demonstrate the susceptibility of adults to measles and also the serious nature of the disease: In 1775 it was introduced into the Sandwich Islands, and in four months 40,000 of the population of 150,000 died. In 1874, Thacombau, the native chief of the Fiji Islands, had measles while on a visit to Sydney. His son and a native attendant sickened on the voyage home, and carried the infection to the islands, with the result that one-fifth of the population (20,000) died. In the epidemic of 1846 in the Faroe Islands, 6,000 out of 7,782 inhabitants were stricken. The virulence of measles under these conditions makes us conclude that a certain amount of resistance is acquired by communities in which the disease has prevailed a long time.

Measles, perhaps more than any other disease, lowers resistance to other infections. Immune bodies diminish or disappear from the blood during and soon after measles. Thus, the tuberculin reaction is in abeyance during and some months after an attack; typhoid agglutinins diminish in the blood. There is a distinct leukopenia.

Convalescent Serum.—Nicolle and Conseil attempted to immunize children with the blood-serum of convalescents and also with virus. The presence of protecting antibodies in convalescent blood-serum has been demonstrated by Park and others. The serum is most active between the tenth day and third month of convalescence. About 5 c.c. of this serum is sufficient to protect a young child against measles. The immunity, being passive, soon wears off and cannot be depended upon for more than a few weeks. If recent convalescent serum is not available, adult serum, or that of a child who has had measles at some previous time, may be used, but in these cases 30 c.c. or more is not always effective.

Resistance of the Virus.—In general the virus of measles is known to be much less resistant than that of scarlet fever and many other infections. The virus does not live long upon fomites, probably less than twenty-four hours. The infection is commonly passed directly from person to person. There is practically no danger of children contracting the infection from the room in which the patient was treated, even though no disinfection was practiced, provided two weeks have elapsed.

Goldberger and Anderson⁴⁶ found, as the result of experiments upon monkeys, that the virus in measles' blood may pass through a Berkefeld filter. In blood-serum it resists desiccation for twenty-five and one-half hours, loses its infectivity after fifteen minutes at 55° C., resists freezing for twenty-five hours, and possibly retains some infectivity after twenty-four hours at 15° C.

From the standpoint of our present knowledge it is evident that any of the ordinary germicidal agents sufficient to kill spore-free bacteria will serve

⁴⁶ *J. Am. M. Ass.*, 1911, 57: 1612.

as effective disinfectants for measles. Aside from the few scientific observations upon the viability of the virus of measles, epidemiological observations have long pointed out the fact that the virus of measles is frail and soon dies in the convalescent as well as in the environment.

Modes of Transmission.—The virus of measles is contained in the nasal and buccal secretions. While it is possible that the virus may leave the body in other secretions, it is highly probable that the discharges from the nose and mouth are the means of transmitting the infection in the vast majority of cases. We are less certain concerning the modes of entrance into the body, although it is presumed that the virus also enters by the mouth and nose. The evidence is clear that measles is usually transmitted by direct contact.

Mayr⁴⁷ showed in 1852 by experiments on the human subject that the buccal and nasal secretions were infective. Anderson and Goldberger⁴⁸ have demonstrated by experiments upon monkeys that the nasal and buccal secretions of uncomplicated cases of measles may be at times, but are not always, infective. Hektoen⁴⁹ in 1905, as well as Goldberger and Anderson, 1911, demonstrated that the virus of measles is also contained in the circulating blood. The virus appears in the blood at least twenty-four hours before the eruption appears, and begins to diminish about twenty-five hours after the first appearance of the eruption. Nicolle and Conseil, also Lucas and Prizer have produced experimental measles in monkeys. Blake and Trask's results are convincing.⁵⁰

It had long been assumed that the virus of measles is carried in the fine bran-like desquamating epithelium, which is one of the characteristics of the disease. Panum in 1847 found that measles was not transmitted during the period of desquamation. Mayr long ago failed in his attempts to inoculate children with measles by using the desquamating epithelium. Anderson and Goldberger also obtained negative results in three experiments, in which it was shown that the "scales" were not infective for monkeys. These authorities believe that it is highly probable, if not altogether certain, that the desquamating epithelium of measles in itself does not carry the virus of the disease. This conclusion is warranted by epidemiological evidence.

Measles is so readily communicable that clinicians receive the impression that the virus is "volatile." It has long been suspected that the virus is contained in the expired breath, but this is very doubtful. In fact, it may now be stated with confidence that measles is not air-borne, in the sense in which this term is usually understood. In any case, the radius of danger through the air is confined to the immediate surroundings of the patient—that is, within the danger zone of droplet infection. Droplet infection is quite possible, as the virus is contained in the secretions of the mouth and nose; furthermore, it evidently requires an exceedingly minute quantity of the virus to reproduce the disease in man, who is exquisitely susceptible to this infection.

⁴⁷ *Ztschr. d. k. k. Gesellsch. de Aertze z. Wien.* 1852, 1: 13, 14.

⁴⁸ *J. Am. M. Ass.*, 1911, 57: 1612.

⁴⁹ *J. Infect. Dis.*, 1905, 2: 238.

⁵⁰ *J. Exper. Med.*, 1921, 33: 385, 413.

Chapin has collected important evidence indicating that the infection of measles is not air-borne. Experiences at various hospitals indicate that the danger of aërial infection in measles is much less than is generally supposed (see also page 855).

The infection of measles is usually transmitted more or less directly from person to person by means of the excretions from the mouth and nose, and most often during the early stages of the disease. Measles may be transmitted by third persons or by fomites only when the time interval is short; such instances are rather exceptional. Carriers are not known. Mild atypical and unrecognized cases of measles occur, but are far less numerous than such cases in scarlet fever, diphtheria, and typhoid.

Prevention.—The suppression of measles is one of the most difficult problems we have to face, for the reason that the disease is one of the most highly communicable of all infections, and for the further reason that it is most contagious during the preëruptive stage. To the student of preventive medicine the problem of measles is very similar to that of smallpox and influenza, and the final control will probably have to await a specific prophylactic measure. Improved sanitation, better hygiene, and the general advance of civilization, which have made such a marked impression upon typhus fever, relapsing fever, leprosy, typhoid fever, and other “filth” diseases, have no influence whatever upon such infections as measles, smallpox or influenza.

Measles is such a common disease that parents are prone to take little pains to avoid the infection; they even sometimes purposely expose their children. This is a mistaken attitude. Special care should be exercised especially during the first five years of life, as over 90 per cent of the fatal cases occur in this period. While it may be almost hopeless to lessen the morbidity in measles, it is quite possible materially to decrease the mortality by simply delaying the age incidence. This may be done by sanitary isolation during the pre-school age. Furthermore, many cases of measles can be prevented, delayed or modified by the use of convalescent serum (page 210). This is particularly useful for the younger age group. While the use of convalescent serum has little effect upon morbidity, it may greatly lessen mortality. It will pay communities to organize a convalescent serum service under a competent epidemiologist. The problem consists first in obtaining a supply of convalescent serum, and secondly in using it promptly in selected situations. Where the supply is limited, it should be used to protect youthful contacts of the pre-school age. The method has some practical difficulties, which, however, are by no means unsurmountable, and the results are quite worth while.

Clinical experience plainly indicates that fewer people die of measles if properly cared for. The mortality may, therefore, be decreased by careful nursing and protection, especially from streptococci and pneumococci, which are the cause of the most dangerous complications. Newman sums up the matter of prophylaxis when he states that “the prevention and control of measles, like that of whooping-cough and tuberculosis, is largely in the hands of the public themselves.”

In the present state of our knowledge the control of measles rests almost entirely upon one measure—isolation, which in this case is highly unsatisfactory. Koplik spots are valuable signs to detect cases early. They appear two or three days, and sometimes six days before the rash. Exposed persons may be quarantined or watched fourteen days. Chapin believes that isolation has been a failure in measles. This is because of the unrecognized but infectious preëruptive stage. “No amount of isolation after the disease is recognized can atone for the harm done before the diagnosis is made.” Isolation, however, accomplishes at least the prevention of further damage. Isolation, as carried out in our large cities, has had no apparent effect upon the prevalence of the disease. In Aberdeen, restrictive measures apparently protected only 7 to 10 per cent of the population.

Cases should be at once reported to the health officer, the house placarded, and visiting prohibited. Quarantine should not be raised nor should the child be permitted to return to school until the manifestations of the disease have disappeared. Home treatment with individual care gives the patient the best chance of recovery. Herman advises against sending children having measles to the hospital on account of the danger of pneumococcus and streptococcal pneumonias. It is difficult under ordinary circumstances to prevent the spread of the disease to the other children in a household. If the case be treated at home, the children who have not had the disease may be sent away, but kept under observation and also under conditions that will not endanger other children in case the disease develops. Convalescent serum finds its chief use in this situation.

Measles patients must be carefully protected against common colds, diphtheria, pneumonia, streptococcal and other infections; also against exposure and other depressing influences.

Terminal fumigation is of no value in preventing the spread of measles. After the patient is released from isolation a general cleaning with boiling of fabrics should be practiced, especially if healthy children are soon to occupy the playroom or bedroom. However, if from 2 to 3 weeks have elapsed, there is practically no danger in a well-ventilated, sunny, and clean room. All bedding, towels, handkerchiefs, and other fabrics that have been exposed should be boiled or otherwise disinfected.

Closing the schools has little effect in preventing the spread of measles. If the school is closed at the beginning of an outbreak and the disease continues to spread after two weeks, little more will be gained in keeping the school closed, for it must then be evident that other factors are at work in spreading the infection. As the disease is mainly spread in the preëruptive stage, it is sufficient to examine the children each morning *before* they enter school for symptoms of a cold, injection of the eyes, running at the nose, cough, sore throat, fever, but especially Koplik spots. All such cases should be sent home to await further developments.

McVail suggests that when a child develops measles all the children exposed may be allowed to continue at school eight or ten days, and then excluded

for a week to ten days, when those who do not develop the disease may be allowed to return. This is a rational plan used in certain districts in England. When measles breaks out in an orphan asylum, a public institution, or an encampment, there is little chance of checking the spread of the disease.

The control of measles is a good example of one of the failures of preventive medicine—a failure, however, that is probably only success postponed.

GERMAN MEASLES

(*Rubella, Rötheln*)

German measles is a distinct disease. It is usually mild and without special complications. It is distinguished from measles by the absence of Koplik spots, the slightness of the prodromal symptoms, the mildness or absence of fever, the more diffuse character of the rash, its rose red color and the early enlargement of the cervical glands. The incubation period is longer than in measles—fourteen to twenty-one days, average seventeen. The two diseases do not protect against each other. German measles is very communicable and extensive epidemics occur.

The cause of German measles is not known. Prevention is the same as for measles.

Clement Dukes described two forms of German measles, one of which he considers a distinct disease somewhat resembling mild scarlet fever, rather than measles. It goes by the name of Dukes' disease, or "the fourth disease."

SCARLET FEVER

Scarlet fever is an acute specific infection, characterized by sudden onset, with fever, sore throat and vomiting and a generalized punctate eruption followed by desquamation. The fever lasts about four days and declines by lysis. The rash usually appears within forty-eight hours, starts about the neck and spreads downward. The cheeks are flushed and the area about the mouth presents a dead white pinched appearance (circumoral pallor), which is striking and characteristic. The disease varies greatly in severity; mild and missed cases and carriers are common and spread the infection.

The period of *incubation* is short but variable: from two to seven days, usually three to four days; occasionally it is less than twenty-four hours. In a few instances in which individuals have been inoculated with the blood of scarlet fever patients, three to four days elapsed before the onset of symptoms. The period of incubation was two days in a laboratory worker who accidentally sucked the cultures into her mouth;⁵¹ it was forty-four hours following swabbing of the throat with cultures in Dick's case.⁵² For public health purposes, the maximum period is placed at eight days.

Occurrence.—Scarlet fever is widespread throughout the world, especially in the temperate climes. It is rare in the tropics, and when intro-

⁵¹ *Arch. Int. Med.*, 1914, 13: 909.

⁵² *J. Am. M. Ass.*, 1923, 81: 1166.

duced soon dies out. Southern races, however, are susceptible, for when they come to northern zones they may have the disease just as severely as the native population. This is an interesting example of the effect of environment upon disease. In the United States the incidence is definitely lower in the southern cities.

Scarlet fever is endemic in every city of considerable size in the temperate regions. In smaller communities it tends to die out after epidemics, and may be entirely absent until a new infection is introduced. The infection is kept alive largely through the atypical, mild and unrecognized cases and carriers. Scarlet fever varies greatly in intensity in different outbreaks. There is about twice as much scarlet fever in the city as in the country; the urban mortality rate is about 10, the rural rate 5.8 per 100,000.

Etiology.—Scarlet fever is due to certain hemolytic streptococci. This was settled in 1923 by Dick and Dick⁵³ whose convincing work closed forty years of doubt and discussion as to whether streptococci were the real cause of the disease or only secondary invaders. This achievement, however, represents the culmination of a mass of careful work by patient scientists, in many laboratories in various parts of the world. The story is an interesting one.

Streptococci are so numerous in the throat in scarlet fever that they early attracted attention. As long ago as 1885, Klein,⁵⁴ an English bacteriologist, isolated a streptococcus from a case of scarlet fever which he called *Streptococcus scarletinae*. The streptococci form a group which were among the first to be studied by pioneer bacteriologists. It was soon learned that they were associated with a great variety of disease conditions. Pasteur in 1879 described cocci in chains as the cause of puerperal sepsis; Fehleisen in 1883 discovered a similar organism, the *Streptococcus pyogenes*, to be the cause of erysipelas; and streptococci were also found associated with abscesses, septicemia, cellulitis, lymphangitis, endocarditis and inflammations of all parts of the body. Streptococci are allies in smallpox, measles, influenza, pneumonia, rheumatic fever, sinusitis, bronchitis, etc. *Streptococcus epidemicus* is the cause of septic sore throat. There are differences among the various pathogenic streptococci which are being studied. One of the reasons given to discredit streptococci as the cause of scarlet fever was that erysipelas and other streptococcic inflammations give no immunity, while scarlet fever does. Another reason was bound up in the fact that we had no way of distinguishing the scarlet fever streptococcus from pyogenes and others in the group. In 1900, Baginsky and Sommerfeld⁵⁵ reported the constant presence of streptococci in the throat in 700 cases of scarlet fever. In 1902, Moser,⁵⁶ of Vienna convinced that one of the streptococci was probably the true cause of scarlet fever, prepared a polyvalent antitoxic serum by injecting killed broth cultures repeatedly into horses. The serum from some horses gave good results

⁵³ *J. Am. M. Ass.*, 1923, 81: 1166.

⁵⁴ *Rep. Med. Off., Local Govt. Bd., London*, 1896-7, 263.

⁵⁵ *Bert. klin. Wehnschr.*, 1900, 37: 588.

⁵⁶ *Wien. klin. Wehnschr.*, 1902, 15: 1302.

when administered early in the disease. This was confirmed by Schick.⁵⁷ Savchenko,⁵⁸ in 1905, proved that a filtered broth culture of streptococcus from scarlet fever contained a strong toxin. He prepared an antitoxic serum. Next, Gabritschewsky,⁵⁹ another Russian bacteriologist, prepared a "vaccine" from broth cultures of streptococci killed by heat and phenol. This vaccine was found to have prophylactic value, but his promising lead received scant attention outside of Russia. Gabritschewsky, in fact, was working with the toxin of scarlet fever, for he found the surprising fact that about 15 per cent of the children who received subcutaneous injections of 0.5 c.c. of the toxic culture fluid without live cocci developed the scarlet fever rash and a few sore throat and the strawberry tongue. At the same time, he found that those who had recovered from scarlet fever did not develop this rash, and also that the streptococci obtained from cases of erysipelas did not produce the rash. Gabritschewsky prepared a vaccine, which when tested in some Russian villages gave protection, but these promising results were not followed up after his death, and the work fell out of the literature. A long interlude then followed. In 1918 Schultz and Carlton⁶⁰ discovered that convalescent scarlet fever serum when injected into the inflamed skin of a case of scarlet fever caused a blanching, which has since been called the Schultz-Carlton reaction.

Numerous attempts to produce the disease experimentally in man and animals either failed or gave inconclusive results. Krumwiede, Nicoll and Pratt,⁶¹ in 1914, observed an accidental infection of a laboratory worker, who sucked into her mouth a mixture of living streptococci. Efforts were made to infect monkeys, but without success.

In 1920, Dochez and Bliss⁶² and also Tunnicliff⁶³ showed by agglutination reactions that hemolytic streptococci of scarlet fever form a distinct group. Dochez⁶⁴ next produced an antitoxin for scarlet fever by injecting the streptococci into a mass of agar jelly previously injected into horses. The method was successful, and an antitoxic serum was obtained which had beneficial effects in the treatment of toxic cases of scarlet fever.

George Dick and his wife, Gladys Dick, in Chicago, in a series of papers⁶⁵ (1921-1924) for the first time reported the production of experimental scarlet fever with pure cultures. They inoculated volunteers with pure cultures of hemolytic streptococci isolated from scarlet fever. They showed that not all of the hemolytic streptococci associated with scarlet fever are of the same cultural type. They further confirmed the fact that the hemolytic streptococcus is found in the throat and is seldom present in the blood in the disease. It is therefore evident that the rash, fever and other toxic symptoms

⁵⁷ *Deutsch. med. Wchnschr.*, Dec. 28, 1905.

⁵⁸ *Russk. Vrach.*, 1905, 4: 797.

⁵⁹ *Russk. Vrach.*, 1906, 10: 467; *Berl. klin. Wchnschr.*, 1907, 44: 556.

⁶⁰ *Ztschr. f. Kinderh.*, 1918, 17: 328.

⁶¹ *Arch. Int. Med.*, 1914, 13: 909.

⁶² *J. Am. M. Ass.*, 1920, 74: 1600.

⁶³ *Ibid.*, 1386.

⁶⁴ *J. Am. M. Ass.*, 1924, 82: 542.

⁶⁵ *J. Am. M. Ass.*, 1921, 77: 782; 1923, 81: 1166; 1924, 82: 265, 301, 542.

of scarlet fever are produced by a soluble poison. They obtained this toxin by filtering broth cultures of hemolytic streptococci. Suitable amounts injected into susceptible persons give symptoms of general malaise, nausea, vomiting, fever, sore throat and a generalized scarlatinal rash. In other words, the Dicks again showed that the sterile toxin alone is capable of producing characteristic symptoms of scarlet fever, including the rash. These symptoms appear within a few hours after injection of the toxin, and disappear within forty-eight hours. The toxin is heat resistant and is neutralized by convalescent scarlet fever serum. The Dicks then demonstrated that this soluble toxin may be used to immunize susceptible persons. One interesting feature of this poison is that minute quantities will produce symptoms in susceptible human beings, but laboratory animals are comparatively insusceptible to it. The next step was to develop a skin test for susceptibility to scarlet fever, which is now called the Dick test. This was the critical point, for by the skin test the Dicks were able to demonstrate the specificity of the scarlet fever streptococcus. Finally, an antitoxin was produced by repeated injections of the toxin into horses. This serum has specific therapeutic value in neutralizing the symptoms due to poisoning, but has little or no effect upon the streptococci themselves. There seem to be more than one strain of scarlet fever streptococci which may require homologous serum or special skin typing for practical use. The subject is still in the experimental stage.

There is still a suspicion in the minds of some students of the subject that scarlet fever is due to a filterable "virus" and that streptococci play a secondary role, but proof of this assumption is lacking. An hemolytic streptococcus appears the true cause of the disease.

Modes of Transmission.—Scarlet fever is commonly spread by direct contact. The streptococci in the vast majority of instances leave the body in the secretions of the mouth and nose and enter by the same channel. Running ears, abscesses and other discharges may remain infective. The streptococci may occasionally enter through wounds, as in surgical and puerperal scarlet fever.

Scarlet fever is less readily communicable than most of the common infectious diseases. Less than half of those exposed contract it, even at the age of greatest susceptibility. Virulence, dose and ability to find lodgment, and the condition of the mucous membrane with reference to penetration and invasion determine the course of events.

There is no evidence that scarlet fever is infective during the incubation period. It is certainly contagious from the beginning of symptoms, and most communicable during the period of eruption. The danger seems to be in direct proportion to the amount of discharge from the mucous membranes of the nose and throat, and the number and virulence of the streptococci contained. The period of infectivity varies greatly, but in uncomplicated cases is probably not over by four weeks. Cases with complications, such as otorrhea, rhinorrhea or other discharges, remain infective for months. There is ample evidence that convalescent carriers spread the disease.

Scarlet fever is often communicated by mild unrecognized cases or by carriers, and occasionally by milk. Fomites occasionally transmit the infection, for the streptococci may live on toys, towels, handkerchiefs and other things contaminated, for weeks and even months, but only under favorable conditions.

Desquamation is not dangerous. It was formerly accepted and taught that the scales carry the infection, and it is now difficult to correct this error. Experience has shown that desquamating patients may as a rule be safely released from quarantine in the fourth week of their attack, provided they have no mucous complications or other discharging sequelæ. Certain convalescents remain infective carriers long after desquamation has ceased. This fact has been emphasized from a study of the so-called "return cases." Thus convalescents are released from hospital and permitted to return home; soon another case appears in one of the members of the household, who in turn comes to the hospital. Neech in a study of 15,000 cases found that the percentage of return cases was 1.86 in those cases who submitted to an average period of isolation of forty-nine days or under. With an average period of fifty to fifty-six days the percentage was 1.12; where the isolation extended to between fifty-seven and sixty-five days the percentage of return cases was one. McCollom stated that in the South Department of the City Hospital, Boston, the children were kept fifty days, and no patient was released who had a discharge from the nose or an abnormal condition of the throat; nevertheless of 3,000 patients discharged from the scarlet fever ward, 1.7 per cent of return cases occurred.

Many cases of walking scarlet fever present little further evidence than a passing sore throat. These missed cases spread the disease. Third persons may occasionally transmit the infection. Scarlet fever is not air-borne. The radius of danger is limited to droplet infection. Experience indicates that the striking distance is not great.

Infected milk occasionally gives rise to widespread and spectacular outbreaks of scarlet fever. Such an epidemic occurred in Boston and the surrounding towns in April and May, 1910. A total of 842 cases were reported, most of them on the route of a large milk contractor. The source of the infection was traced—a "missed" case on one of the milk farms. Milk-borne scarlet fever is, so far as known, always due to infection from human sources, and with the more general employment of pasteurization in the large cities of this country is becoming relatively infrequent. Ice cream and other fresh milk products may carry the streptococcus. Two outbreaks of scarlet fever due to ice cream have been reported. One took place in South Kensington, England, in 1875, following a large dinner where the dessert was frozen pudding.⁶⁶ A clear-cut outbreak due to ice cream occurred in Flint, Michigan, in July and August, 1924, involving forty-one cases and extending over seven days. The ice cream was infected by the maker who had a mild case of scarlet fever.⁶⁷

⁶⁶ *Suppl. to Rep. Loc. Govt. Bd.*, 1875, 72.

⁶⁷ *Am. J. Hyg.*, 1925, 5: 669.

Seasonal Distribution.—Scarlet fever is a cold weather disease. The seasonal prevalence resembles that of diphtheria. In this climate scarlet fever increases in the autumn and reaches its greatest incidence from January to March. From that point, the curve of incidence declines during the spring and reaches its minimum with striking consistency in July or August. In London, over a period of thirty years, the mode of incidence fell in October and the minimum in April. In New Zealand, where the seasons are practically reversed, the greatest prevalence is from April to June and the lowest from November to February. Scarlet fever recurs with the regularity of the crops. The influence of climate is striking upon its prevalence, but the seasons have no effect upon its virulence. Thus, in Providence, over a period of forty years, on the basis of reported cases and deaths, the case fatality rate remained practically constant throughout the year.

Susceptibility by Age and Sex.—As in diphtheria, infants show a relatively high immunity to scarlet fever, but when the disease occurs under one

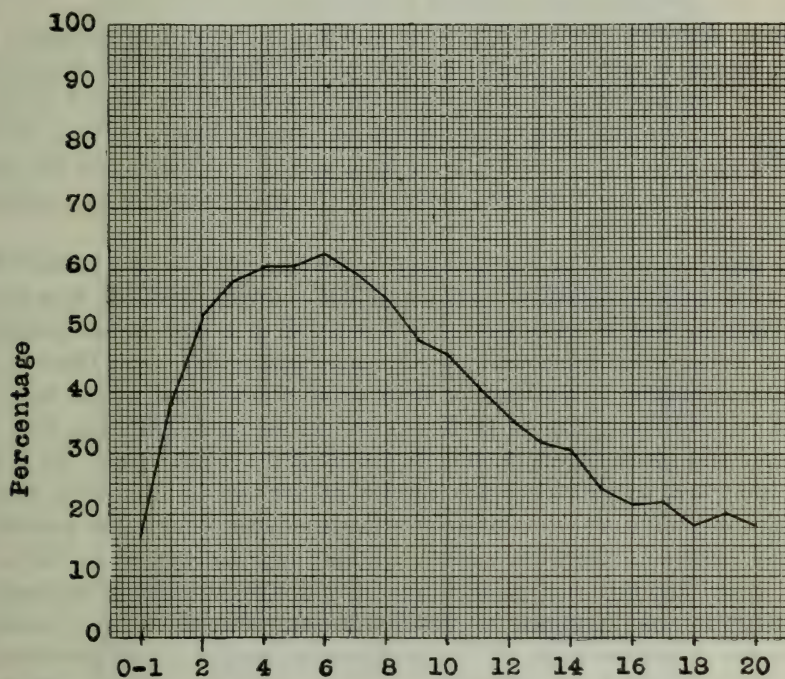


FIG. 12.—SCARLET FEVER, PROVIDENCE, 1887-1920. ATTACK RATES AMONG SUSCEPTIBLES EXPOSED IN THE FAMILY.

Rate at given age (17,625 cases). (From Pope, *Am. J. Hyg.*, 1926, 6: 389.)

year it is very apt to be fatal. After the first year, the susceptibility rises sharply and reaches its height at the sixth year. For the next three years it falls rapidly, and then more gradually to adult life. Approximately 75 per cent of all cases occur under ten years and 90 per cent under fifteen years. In the United States registration area for the decade 1910-1919,

the highest mortality from scarlet fever occurred during the third year of life. Fifty-two per cent of all deaths were under five years and 80 per cent under ten years of age.

Donnelly's figures, based on several million cases in American, European and English cities, show slightly higher morbidity rates among males during the first five years of life. At all ages above seven there is a definite excess of cases among females. Among 1,600 cases in Providence, the attack rates from seven to twenty years were 10 to 50 per cent higher among females; and over twenty years, approximately twice as high as among males. On the basis of these studies, it seems probable that the higher morbidity among females above the age of seven is due to two factors: (1) a physiological difference in susceptibility and (2) prolonged and intimate exposure in the household. On the other hand, while the disease is more prevalent among females, it is more malignant among males. Donnelly⁶⁸ found the case fatality rates approximately 20 per cent higher among males than among females, and this difference constant at all ages.

Second Attacks—Immunity.—One attack usually confers immunity for life, but second attacks do occur. A study of Chapin's⁶⁹ data for Providence shows that among 22,105 cases, 271 were reported as second attacks, approximately one per cent. The excess of females among second attacks above the age of seven is much greater than among first cases, and the study indicates that prolonged intimate contact is the chief factor in the incidence of second attacks.

The susceptibility of an exposed population to scarlet fever is indicated by attack rates from Providence. Among 20,000 individuals of all ages giving no previous history of scarlet fever and exposed to a case in the family, 8 per cent developed the disease. At the most susceptible ages, three to six years, approximately 30 per cent of those exposed developed scarlet fever.

The natural immunity to scarlet fever varies, then, with age and sex. Immunity and susceptibility to this disease is also a family trait. It is well known that lack of resistance has carried off all the children of a family, seemingly overwhelmed with toxemia, sometimes even during the prevalence of a mild infection. Acquired active immunity may be induced by the injection of toxin; and transient passive immunity by the injection of antitoxin.

Change in the Virulence of Scarlet Fever.—One of the most striking phenomena in epidemiology is the progressive change in the character of scarlet fever. There has been no noticeable change in its incidence, but it has lost much of its virulence. Scarlet fever is now much milder than formerly, although there is about as much of it. This is the general experience everywhere. The figures from Providence are representative: From a mean mortality of forty per 100,000 for the decade 1875-1884, the rate had fallen

⁶⁸ *Am. J. Dis. Child.*, 1916, 12:228.

⁶⁹ *J. Prev. Med.*, 1926, 1: 1. Charles V. Chapin, Supt. of Health, Providence, R. I., has an admirable collection of data carefully brought together under his personal supervision during a long career. See also Pope, *Am. J. Hyg.*, 1926, 6: 389.

to two per 100,000 for the decade 1915-1924. For the past forty years the case fatality has shown a negative trend of 5 per cent per year. During

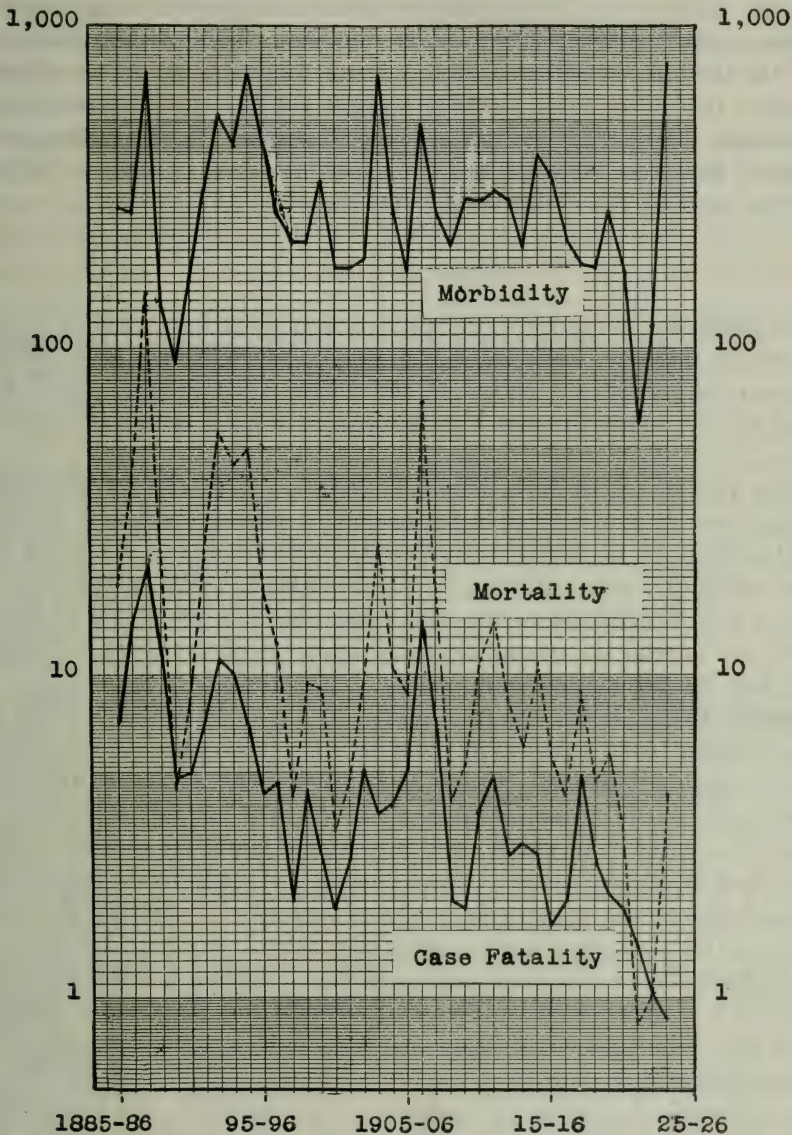


FIG. 13.—SCARLET FEVER, PROVIDENCE, 1886-1924. MORBIDITY, MORTALITY AND CASE FATALITY RATES BY EPIDEMIC YEARS, AUGUST 1-JULY 31.

Chart shows great decline in the severity of the disease but practically none in prevalence. (From Pope, *Am. J. Hyg.*, 1926, 6: 389.)

this same period the case fatality rates have varied greatly in different epidemics, ranging from 0.86 to 21 per cent. Wide excursions from year to

year are characteristic of scarlet fever epidemics in all countries, but the downward trend is general.

Uncertainty in the diagnosis of a disease so protean as scarlet fever, especially the mild atypical form now prevalent, and general improvement in reporting make it difficult to express the prevalence with mathematical precision. On the basis of reported cases in some fifty large American cities for the period 1890-1920, there has been a slight but hardly significant decrease in incidence. Several cities show a marked increase. In Providence, where the figures have been collected under Chapin's personal supervision for forty years, the decrease is not statistically significant.

PREVENTION

The prevention of scarlet fever follows clearly defined lines: early recognition, prompt isolation, disinfection and specific prophylaxis. It is comparatively easy to prevent the spread of the disease from known cases. Much damage is done before the case is recognized, or often because not recognized at all.

Every case of scarlet fever should be isolated. There is no objection to treating a case of scarlet fever in the home if a suitable room and an intelligent attendant can be provided—preferably, someone who is not doing cooking or caring for other children in the household. With proper care the infection may be confined to the sick room, but it is preferable to take no chances and to send the susceptible individuals out of the house. The nurse should take the precautions described for diphtheria.

Specific Prophylaxis.—Specific prophylaxis may be achieved either with antitoxic serum (passive immunity) or with toxin (active immunity). Passive immunity may be produced either with antitoxic horse serum or with convalescent human serum. Passive immunity is transient but lasts long enough to tide over the period of incubation and is therefore useful for youthful contacts and to check family outbreaks. Active immunity is conferred by injecting the toxin subcutaneously in suitable amounts, spaced at intervals of a week. Ordinarily this produces a definite immunity which is probably lasting.

The Dick Test.—The Dick test consists in injecting a small quantity of toxin (0.1 c.c.) into the skin. The toxin consists of a filtrate of a broth culture of the specific hemolytic streptococcus. This is diluted so that 0.1 c.c. contains just enough to produce the typical action, and this amount is called a skin test dose. A positive reaction indicates susceptibility and a negative reaction immunity to scarlet fever. When positive, the redness appears in about six hours, reaches its height in eighteen to thirty-six hours and subsides promptly. The reaction is a circular area of erythema, sometimes accompanied by swelling of the skin. It gives the impression of an anaphylactic reaction.⁷⁰ Most children with scarlet fever react positively to this test, until about the

⁷⁰ The Schick test comes on later, lasts longer and is clearly the result of the irritating action of the diphtheria toxin.

sixth day of the disease, when they become negative. A few remain positive and susceptible. The Dick test therefore has diagnostic significance.

Pseudoreactions also occur, but these are so difficult to distinguish from true reactions that control tests are usually omitted. If controls are desired for teaching or scientific purposes, control tests should be made with toxin-antitoxin mixtures instead of with the heated toxin as in diphtheria, because the scarlet fever toxin is very resistant to heat.

Acquired Active Immunity.—Protection against scarlet fever may be induced by the subcutaneous injection of the toxin. The Scarlet Fever Committee recommends five subcutaneous injections of 500, 1,500, 15,000 and 20,000 skin test doses of toxin as a minimum, spaced at intervals of one week. The first injection must not exceed 500 skin test doses, since the Dicks found ⁷¹ that initial injections of more than this amount frequently give quite severe reactions, with headache, nausea and vomiting, and also a scarlatiniform rash. Young and Orr ⁷² recommend three injections of 500, 5,000 and 30,000 skin test doses, respectively, with an interval of two weeks between injections.

It is too soon to speak positively of the degree and duration of immunity conferred. In a series of retests Park has found that in a group of immunized children the Dick test was still negative at the end of a year. At the end of eighteen months some of the same children showed a faintly positive Dick test with a strong toxin. From these results Park believes that it is safe to assume a conferred immunity of at least two years. The method is useful in epidemics, in institutions, in families and in special emergencies, but is hardly ready to broadcast for general public adoption.

Passive Immunity.—Temporary protection against scarlet fever in susceptible contacts may be secured by the injection of scarlet fever antitoxin, similar to the passive immunity conferred by prophylactic doses of diphtheria antitoxin. In family contacts and persons who have been exposed to scarlet fever and cannot be kept under close observation, this is a measure of some value. It is probable that such passive immunity does not last over two or three weeks. On account of this short duration it seems better practice in institutions to test all children for immunity to scarlet fever and to immunize with scarlet fever toxin those who show a positive Dick test. Then if any contract scarlet fever during the process they can at once be treated with antitoxin.

Specific Treatment.—Scarlet fever antitoxin if given early and in sufficient amounts is of distinct value in the treatment of scarlet fever. The temperature drops promptly, the rash fades, and the patient experiences great relief from the toxic symptoms. It has not been shown that the use of antitoxin has any effect on complications that have already occurred or on post-scarlatinal sepsis, but patients treated early with antitoxin seem less likely to develop complications than cases not so treated.⁷³ For the best results the

⁷¹ *J. Am. M. Ass.*, 1925, 84: 1477.

⁷² *J. Am. M. Ass.*, 1926, 86: 1340.

⁷³ F. G. Blake and J. D. Trask, *Boston M. & S. J.*, 1925, 193: 659.

full amount of antitoxin required should be given in one dose and as early as possible in the course of the disease. No benefit can be expected from the use of antitoxin after the acute stage of the disease is over. Scarlet fever antitoxin may be given intramuscularly or intravenously, depending upon the urgency of the case.

Quarantine and Isolation.—Although there is little definite evidence that quarantine and isolation have materially reduced the incidence of scarlet fever, they should be continued as the main line of defense. Baker⁷⁴ has shown that in rural districts and small cities prompt and effective quarantine seems to have a marked effect in limiting the spread of infection. In large cities, with the much greater opportunity for contact, it is doubtful whether quarantine has had any appreciable effect on the prevalence of scarlet fever. The reason for this is that in most cases opportunity for infection has occurred before the diagnosis is made. With the mild type of scarlet fever now prevalent, it is practically impossible to recognize all cases, and with the most rigid quarantine there are always enough missed cases and carriers to keep the fires burning.

Period of Detention.—The period of detention varies greatly. Among sixty-four of the larger American cities, twenty-two specify a period of thirty days, and nineteen a period of twenty-eight days; twelve have a detention period of only twenty-one days, and four still require six weeks; three merely state "till all discharges and desquamation have ceased." In general, the short period has followed a low incidence of scarlet fever, as in the South, while the longer period seems to be an effort to combat greater prevalence. In spite of the lack of any evidence that the desquamation is infective, twenty-four of these cities still hold all convalescents until desquamation is complete. An unduly long detention is a hardship upon the patient and the family and fosters the concealment of cases; on the other hand, a scant period is hazardous to the community. For administrative purposes four weeks is practical. Cases with rhinorrhea, otorrhea, sore throat or discharging abscesses should receive special care, as the secretions from these parts are known to remain infectious for a long time. Any unhealed area of the body surface, either cutaneous or mucous, may discharge the virus. Bacteriologic technic is not sufficiently developed upon which to release convalescents and carriers.

Hospitalization.—Hospitalization is of distinct value in the management of scarlet fever, especially where the patient cannot receive the proper supervision and medical attention at home. Under such conditions, hospital treatment unquestionably helps to keep down the mortality from scarlet fever, but there is no evidence that it has been an important factor in reducing the incidence of the disease. In English cities, some forty years ago, contagious disease hospitals were built on an extensive scale with the idea that hospitalization would control the acute infectious diseases. A number of cities hospitalized 90 to 95 per cent of their scarlet fever cases, but without any signi-

⁷⁴ *Ann. Rep. Mich. State Bd. of Health*, 1898, 207.

ficant decline in the morbidity rate from scarlet fever. In Leicester⁷⁵ the hospital was closed temporarily and all patients sent to their homes, with an actual decrease in the number of reported cases.

Closing of Schools.—The answer to the question whether the schools should be closed during an outbreak of scarlet fever varies with the circumstances. In the country districts, where the children can be kept apart, this is advisable, but in the cities little can be gained. Better results can usually be obtained by careful daily inspection of all pupils than by closing the schools. Where a person is ill with scarlet fever, all children in the household who have not previously had the disease should be excluded from school for one week from the last possible exposure. All other members of the household may be allowed to continue their usual occupations except those engaged in handling food or milk and those brought in close contact with children.

From studies made in my department, it is clear that scarlet fever (like diphtheria and measles) is usually brought into the home by a six-year-old school child; and then spreads to the younger brothers and sisters.

Pasteurization of Milk.—Milk-borne outbreaks may be entirely avoided by pasteurization. The streptococci are killed with certainty at 142-145° F. for thirty minutes (page 732). Properly pasteurized milk has never been known to transmit scarlet fever. The Boston epidemic of 1910 was promptly checked by pasteurization (page 723).

Complications and Their Prevention.—Scarlet fever is often a serious disease, not only on its own account, but also because of complications which are numerous and varied. Complications of greater or less extent occur in about 40 per cent of all cases. Some of the most damaging are otitis and mastoid disease, acute nephritis, arthritis, heart lesions, bronchopneumonia, pleurisy with serous effusion and noma. In institutions for children and hospitals for contagious diseases scarlet fever is often complicated by other infectious diseases, particularly diphtheria, measles and erysipelas. This is a serious problem.

The prevention of damage to the kidneys consists in absolute rest in bed during the whole course of the disease, at least three weeks, in a room well ventilated but free from drafts. Children should wear flannel nightclothes. We have no proof that a nitrogenous diet may be a factor in the production of nephritis, but it seems a part of wisdom to place as little work on the kidneys as is reasonably possible. The early administration of antitoxin bids fair to prevent many of the complications of scarlet fever.

Disinfection, *not* Fumigation.—The physician should wash and disinfect his hands carefully after each visit and take special pains to purify his thermometer and other instruments. The discharges from the mouth, nose, respiratory passages or other mucous surfaces should be collected upon pieces of gauze or paper and burned. Bed and body clothing, dishes and other

⁷⁵ *Pub. Health, London*, April, 1901.

objects exposed to contamination must be disinfected, by boiling if possible. Fabrics that cannot be boiled or steamed may be disinfected by immersion in a germicidal solution, such as bichlorid of mercury or phenol. Care must be taken concerning remnants of food that come from the sick room.

Terminal fumigation of the sick room with formaldehyd gas was long considered essential, but Chapin⁷⁶ has shown conclusively that the proportion of recurrences following scarlet fever is no higher when terminal fumigation is omitted. Practically all of the more progressive cities in this country have now abandoned terminal fumigation. Formaldehyd has the merest surface action and is otherwise not dependable. Careful bedside (concurrent) disinfection during the course of the disease and a thorough washing of surfaces with a germicidal solution of hot water and soap, with good sunning and airing of the room, are effective.

Scarlet Fever and Diphtheria Contrasted.—These two diseases have remarkable resemblances despite the fact that they are distinct entities. They were formerly confused. Sydenham, the distinguished English clinician, in 1675 gave the first full account of scarlet fever, which he called *febris scarletina*. It was not until about 150 years afterwards (1826) that Bretonneau, of Tours, described diphtheria.

Both diseases are due to microorganisms which largely remain localized in the throat and usually do not invade the blood and internal organs. The symptoms of toxemia are due to a soluble poison that enters the blood and pervades the tissues. The toxin of each infection is used for the skin test, which is a practical index of susceptibility and immunity. The toxins are also useful in producing an active immunity. Specific antitoxins are available in the treatment of each disease.

Age incidence, seasonal prevalence, degree of communicability, geographic distribution and other epidemiological features are strikingly alike. Both diseases also have similar methods of personal prophylaxis and community control.

WHOOPING-COUGH

(*Pertussis*)

Whooping-cough occurs in epidemics, which vary greatly in virulence, intensity, and mortality. The disease is more frequent and severe in cold and changeable climates. The seasonal prevalence, however, is in the spring with a tendency to prevail in the warm month of August. The cause of whooping-cough is a small bacillus, described by Bordet and Gengou, recognized by agglutination and complement fixation tests.⁷⁷ This bacillus is found most readily in the early or catarrhal stage in the mucus from the depths of the bronchi brought up during the paroxysms. In this exudate, which is white, thick, and rich in leukocytes, the bacilli exist in considerable numbers and

⁷⁶ *Sources and Modes of Infection*, 2d Ed., 1912, Wiley, N.Y., p. 250.

⁷⁷ *Ann. de l'Inst. Pasteur*, 1906, 20: 731.

sometimes in almost pure culture. They usually disappear after the fourth week.

Whooping-cough has not been given the study that its importance deserves. Much of our knowledge is assumed. Here is a good field for research into almost every phase of the subject. Whooping-cough presents some interesting anomalies to the class of respiratory diseases in which it is commonly grouped. It has an unusual seasonal prevalence and is more prevalent in rural communities than in cities.

Occurrence.—Whooping-cough has a world-wide distribution except for a few regions remote from the general stream of travel. It is endemic almost everywhere; epidemics are frequent and sporadic cases crop out. It is generally stated that the disease is rarer in tropical and subtropical countries, increasing as we go north. While this statement lacks confirmation, it is undoubtedly true that the disease is more severe, with fatal complications, in colder climates.

The figures from Denmark and Massachusetts show a tendency to periodicity about every four years. The seasonal prevalence is somewhat different from other infections of the upper respiratory tract. In this country the morbidity curve has two peaks, one in April or May, and the other in August. The disease is less prevalent in September or October and in July. The mortality curve also shows several marked rises; a small one in February, the highest in April, and a third in August. In Denmark the seasonal prevalence is highest in March and April.

The disease is largely independent of economic conditions and seems to be more prevalent among colored than whites; among the native white than among the foreign born population; and among girls than boys.

Age Distribution.—The maximum incidence of whooping-cough occurs at five to six years of age. Luttinger gives the following figures based on 10,000 cases in New York City:

<i>Age</i>	<i>Per Cent</i>
Under 1 year.....	19.5
Between 1 and 2 years.....	20.0
From 2-5 years.....	40.0
From 5-15 years.....	18.0
Over 15 years.....	2.5

From this we see that about 80 per cent of the cases occur under five years of age. In Massachusetts from 1913-1919, Henry brings out the point that 33 per cent of the cases occurred under three years.

Mortality.—The dangerous nature of whooping-cough is not generally realized. Whooping-cough almost everywhere causes more deaths than scarlet fever. Many deaths registered as bronchitis and bronchopneumonia are secondary to whooping-cough; during the summer, gastro-intestinal complications are serious in young children. In the United States the death rate

per 100,000 is around 10.5. In Glasgow the annual mortality from whooping-cough for 40 years, 1855-1894, was 13.5 per 100,000 inhabitants and exceeded that from any other acute communicable disease. In England and Wales in 1891 more deaths occurred from whooping-cough than from measles, diphtheria, scarlet fever, or typhoid fever. The mortality figures would be still higher if all the deaths directly or indirectly due to it were completely reported, for the fatal termination is usually due to complications and sequelæ which occur in one-fourth to one-third of all cases. As a result of these complications the original disease is frequently lost sight of entirely in the vital statistics.

In the United States registration area for the years 1911-1922, inclusive, the death rates were higher in rural districts than in cities of 10,000 or over, the ratio being 119 to 100. This corresponds to the rural-urban distribution of infantile paralysis.

In the United States registration area from 1900-1921 the average annual mortality rate was about 10.5 per 100,000 population. In Massachusetts, from 1906-1924, the death rate was nine per 100,000. In Denmark, from 1890-1922, the average annual mortality rate was higher than for this country, but it has shown a marked downward trend. Madsen⁷⁸ gives the following comparative table of deaths in Denmark, England and Wales, and the United States:

PERCENTAGE OF DEATHS FROM ALL CAUSES

Cause	Denmark, per Cent	England and Wales, per Cent	United States Registration Area, per Cent
Whooping-cough	1.4	1.3	0.7
Diphtheria	0.7	0.9	1.2
Measles	0.5	1.9	0.6
Scarlet fever	0.4	0.3	0.4
Typhoid fever	0.2	0.3	1.0

In New York City, from 1866 to 1915, 51 per cent of the deaths from whooping-cough occurred under one year of age, and 27 per cent between one and two years of age, making a total of 78 per cent of deaths under two years. In Massachusetts, from 1913 to 1919, 90 per cent of the deaths occurred in children under two years old. In Denmark, from 1910 to 1914, 62 per cent of the deaths occurred under one year of age and 38 per cent between one and five years.

Mode of Transmission.—Whooping-cough is assumed to be transmitted directly from person to person in the same ways that diphtheria and other infections contained in the secretions of the mouth and nose are spread. Handkerchiefs, toys, drinking cups, roller towels, and other objects recently contaminated with the infective secretions may act as vectors. It is also trans-

⁷⁸ *Boston M. & S. J.*, 1925, 192: 50.

mitted by droplet infection, kissing and close contact. In 60 per cent of the cases studied in New York, the source of infection was given as coming from a neighbor (Luttinger).⁷⁹ Parents are prone to contract the disease from their children.

Whooping-cough is apparently not contagious during the period of incubation, but is communicable from the appearance of the first symptom, and is most contagious during the early stage before the whoop develops. It may be transmitted in the late stages and after convalescence. While the virus is known to be in the secretions from the respiratory tract, all secretions from the mouth and nose (including vomitus) must be regarded as infective.

Convalescent carriers are believed to be rare, and practically a negligible factor. Healthy carriers are not known. Missed cases consist of the so-called abortive type, in which the disease reaches only the catarrhal stage without assuming the characteristic whoop. Such persons doubtless spread the infection broadcast.

Whooping-Cough in Animals.—Jahn and others called attention to the fact that domestic animals may be affected by whooping-cough, and that they may be the means of transmitting it to children. Coughs of a paroxysmal nature have been observed in dogs, and also in cats. Klimenco⁸⁰ and Fraenkel⁸¹ were able to produce what seemed like typical pertussis in monkeys, and Inabo⁸² showed that injection of the bacillus in an ape gave rise to a typical whooping-cough with an incubation period of thirteen days. Kittens and puppies do not take the disease under ordinary circumstances and for practical purposes of prevention are usually disregarded. Mallory and Horner⁸³ have shown that masses of *B. bronchisepticus* are found in the superficial layer of the trachea, thereby mechanically paralyzing the cilia. Some of the whooping-cough of animals may be confused with this infection, for the two organisms resemble each other closely (Ferry and Noble).⁸⁴

Immunity.—There is no natural immunity to whooping-cough; all are susceptible. Whooping-cough at one time or another affects almost every member of a community; by the time adult age is reached, about 78 per cent have had it. It causes 10,000 or more deaths yearly in this country alone. The greatest susceptibility is between six months to five years. Very few cases occur during the first months of life. The largest number of deaths occur in the second half of the first year. After five years the susceptibility decreases with age. One attack confers a definite and prolonged immunity; second attacks are rare.

There is a higher incidence and mortality among girls (56 per cent) than boys (44 per cent). Whooping-cough is the only communicable disease of childhood that shows this peculiarity.

⁷⁹ *Bull. No. 49*, Dept. of Health, New York City, Sept., 1916.

⁸⁰ *Centralbl. f. Bakteriol.*, 1908, 48: 64.

⁸¹ *München. med. Wchnschr.*, 1908, 55: 1683.

⁸² *Ztschr. f. Kinderh.*, June 15, 1912.

⁸³ *J. Med. Research*, 1912, 27: 115.

⁸⁴ *J. Bacteriol.*, May, 1918, 193.

The death rate for whooping-cough in the United States in 1923 was 9.7 per 100,000.

Prevention.—The incubation is probably two to three weeks, but the time is indefinite, owing to vagueness of the onset of symptoms. If sixteen days have passed without symptoms the danger may be considered as having passed. For practical purposes of prevention fourteen days will cover most cases. The long-drawn-out nature of the disease, the difficulty of diagnosis in the early stages when it is most contagious, and the fact that patients sometimes continue to spread the infection for four to six weeks after apparent recovery, make the control of whooping-cough an exceedingly difficult problem. Effective measures must await specific prophylactic methods.

The disappearance of the bacillus after about four weeks, the fixation of complement in the third week, together with clinical observations, have led many to believe that the contagious period of the disease is practically restricted to the catarrhal stage; that is, during the time its true nature is not recognized. There is doubt and uncertainty on this point, and in some places isolation is not attempted, while in others it ranges from three weeks to three months, but ineffectively enforced.

Whooping-cough should be reported, houses placarded, and the patient isolated, but the isolation in this case need not include strict confinement to a room. This, in fact, may be an unnecessary hardship to the patient, who does better out of doors. If the patient is permitted to take the air, he must avoid contact with his fellowmen and not go to school, theater, church, public assemblies, nor ride in street cars or public vehicles. Children should go out only when accompanied by an intelligent caretaker as a protection to others. It has been suggested that children with whooping-cough who are permitted their liberty should be plainly labeled with a red cross on their arm, or a yellow flag, or the word "whooping-cough" conspicuously displayed on their clothing, to serve as a warning to others. Children who have been exposed should be isolated as soon as they show catarrhal symptoms or begin to cough, not waiting for the characteristic spasmodic whoop. Each community should provide a hospital-farm for children with whooping-cough who lack proper care.

Early diagnosis and prompt reporting of cases are essential. Other catarrhal diseases may cause paroxysmal cough, hence a positive diagnosis can only be made from cultures. Luttinger states that only about 10 per cent of the cases of whooping-cough in New York City are reported. The disease is most communicable during the catarrhal or early stages, when the infection can be recognized only by bacteriological methods.

Patients should not be released from quarantine until at least four weeks have elapsed after the onset of symptoms. The duration of isolation varies in different cities; thus it is six weeks in Montclair, New Jersey; on recovery in Providence; as long as the cough lasts in Boston. In Michigan the disease is considered infectious three weeks before the whoop and four to six weeks after apparent recovery. The Danish regulations permit release from quaran-

tine after four weeks and two negative cultures.⁸⁵ The State Board of Health of Michigan requires disinfection of the clothing and premises before the patient is released, and forbids public funerals in deaths from whooping-cough. Standard procedures must await the results of scientific studies.

Individual prophylaxis consists in avoiding the infection. The greatest care in this regard should be taken with children before the age of five years. Ninety-six per cent of the 6,324 deaths from whooping-cough in the United States in 1906 were in children under five years of age. Dogs, cats, and other domestic animals should be kept away from the patient, and the possibility of conveying the disease in this way must be guarded against in the susceptible.

The Bordet-Gengou bacillus is frail and soon dies in the environment; therefore, terminal fumigation of the room is not necessary, although cleaning and airing of the premises are in order. Handkerchiefs, fabrics, toys, and other objects that have come in contact with the secretions from the mouth and nose should be boiled or saturated with a strong germicidal solution. The sputum and vomitus should be burned or disinfected as for tuberculosis.

Whooping-cough usually runs a favorable course in healthy children over five years old, and after puberty it is rarely fatal. The most important thing then is to keep babies and young children from having the disease. Delaying the age incidence will materially decrease the mortality. Particular care should be taken during and for several months following convalescence to prevent the development of tuberculosis.

Schools need not be closed on account of whooping-cough. Daily inspection of pupils may achieve better results. The well children of a household, where a person is ill with whooping-cough, may be excluded from schools unless they have had the disease or until two weeks have elapsed since the last exposure, and then only provided they are free from catarrhal symptoms. Other members of the household may be allowed to follow their usual occupations.

Hess, Madsen and others have used vaccines of the Bordet-Gengou bacillus as a prophylactic with seeming success. The evidence so far shows no striking protective power of these vaccines. The immunity produced is slight if any. The subject is being studied.

The gravity of whooping-cough is scarcely appreciated, either by the physician or the public, and there is much heedless neglect with reference to the prevention of this infection.

The control of whooping-cough is a matter which is largely in the hands of the public itself. The dangerous nature of this infection should be emphasized, and people taught that it is contagious both before and after the "whoop." Mild cases which do not have the characteristic whoop spread the disease; this is especially common in adults.

⁸⁵ Chievitz and Meyer, *Ann. de l'Inst. Pasteur*, 1916, 30: 503.

MUMPS

(*Epidemic Parotitis*)

Mumps usually occurs between the ages of five to fifteen years; the maximum incidence is seven to nine years. There is decreased susceptibility both before and after this time. It is a disease of children and young adults. As a military problem, mumps frequently occurs in men between twenty-one and thirty-one years. In the soldier and sailor the infection is dreaded because it is disabling and unmanageable. In 1918, there were 5,756 cases of mumps among 18,000 men at Camp Wheeler, an incidence of 32 per cent. Most cases occur in spring and autumn, and the disease is endemic in most large centers of population. Mumps is about as prevalent among girls as boys; is more prevalent among the native white than among the foreign-born children; and possibly more prevalent among colored than white. One attack usually confers immunity, but second attacks occur, and third attacks are sometimes reported. The disease may occur in civil institutions as well as military establishments as epidemics which usually develop slowly and last a long time. It is difficult to trace and eradicate. Orchitis is a frequent and painful complication and when both testicles are involved may cause sterility. Other complications are: great prostration; a tendency to develop mania, or wild delirium, or a comatose state resembling uremia; meningism, mastitis, otitis media, tonsillitis, and pneumonia also occur. Mumps is contagious before the symptoms appear, and for some time, even six weeks, after symptoms have disappeared. It is probably transmissible as long as the glands are swollen. The parotids are most frequently involved, next the submaxillary, and last the sublingual. The lacrimal glands escape. The disease is usually spread by direct contact; rarely by indirect contact or by a third person. It is not air-borne. The virus is contained in the secretions from the mouth and perhaps the nose. The incubation is variously stated at from eight to thirty days; it is variable and sometimes prolonged. Most cases develop in from eighteen to twenty days. For public health purposes the maximum period of incubation is placed at twenty-one days.

Granata in 1908, and Nicolle and Conseil⁸⁶ in 1913 obtained indications that mumps may be due to a filtrable virus; Martha Wollstein⁸⁷ in 1918 showed that the salivary secretion in mumps contains a virus, which when filtered and injected into the parotid glands and testicles of cats, causes pathological changes resembling mumps in human beings.

The virus is detected most readily in the saliva during the first three days of the disease, less easily on the sixth, and not at all on the ninth day. It is also present in the blood of patients showing marked constitutional symptoms, but not in the cerebrospinal fluid.

Prevention depends upon the usual practice of isolation and disinfection.

⁸⁶ *Compt. rend. Acad. d. sc.*, 1913, 157: 340.

⁸⁷ *J. Exper. M.*, 1916, 24: 265; 1918, 27: 337.

Hess⁸⁸ injected 6 to 8 c.c. of blood of convalescents intramuscularly into seventeen children. None of these children developed mumps, although exposed to it in an institution where the disease was epidemic. This procedure could be made use of in the home as well as in institutions. Regan⁸⁹ found that from 2 to 4 c.c. of convalescent serum protects.

Orchitis occurs as a complication in as high as 25 per cent of the cases. It is rare before puberty. There is some support for the old suggestion that the infection is conveyed to the testicles by hand-urethral inoculation, but more likely the virus is brought to the testicles by the blood stream. Traumatism of the testicles predisposes to orchitis, hence injurious palpation and injuries however slight should be avoided. Rest in bed is also a preventive.

LOBAR PNEUMONIA

The term pneumonia includes a group of varied diseases due to different causes, but all producing an inflammation in the lung tissue. Pneumonia may be primary or secondary; it may be lobar (croupous) in type or it may be lobular (bronchopneumonia). Classifications according to the anatomic pictures and the causative agents do not run parallel, but the latter are more significant from the standpoint of prevention and cure.

While the pneumococcus is the most common cause of pneumonitis, other pathogenic organisms may be responsible, such as streptococci, Pfeiffer's bacilli, Friedländer's bacilli, plague bacilli, typhoid bacilli, etc. A pneumonic condition is also a frequent terminal state especially in the young and in the old. Because of the difficulties of classification, changing concepts of etiology and different standards of diagnosis, there is much confusion in the literature as well as in the morbidity and mortality records concerning pneumonia.

Lobar pneumonia is the most clear-cut variety of the group clinically and histologically and studies have shown that the pneumococcus is the responsible agent in about 95 per cent of the cases. This is an acute febrile infection with consolidation of lung tissue which, in typical cases, ends by crisis. The pneumococcus is found not only in the lungs and the respiratory tract but often invades the blood. This latter complication increases the severity of the disease.

The pneumococcus is a Gram-positive diplococcus, lance-shaped, with a capsule and further characterized by the fact that it is soluble in bile, ferments inulin and responds to specific agglutinins and precipitins.

Occurrence.—Pneumonia is one of the most prevalent and fatal of all acute diseases. As a cause of death it rivals and sometimes exceeds tuberculosis. The figures, however, are unsatisfactory on account of the difficulties of diagnosis and registration. Ten per cent of deaths from all causes in the United States registration area, 1900-1920, were due to pneumonia, all forms, of which 6 per cent were lobar and undefined pneumonias, and 4 per cent

⁸⁸ *Proc. Soc. Exper. Biol. & Med.*, 1915, 12: 144.

⁸⁹ *J. Am. M. Ass.*, 1925, 84: 279.

bronchopneumonia. It is impossible to say whether pneumonia is increasing or declining. According to the United States Mortality Statistics pneumonia appears to be on the decline, but this may be more apparent than real on account of errors and incompleteness of registration and diagnosis. The following figures indicate some of the changes taking place in the United States Mortality Statistics:

CAUSE OF DEATHS	DEATH RATES PER 100,000 FOR THE UNITED STATES REGISTRATION AREA			
	1914		1922	
	Rate	Percentage of Total Rate	Rate	Percentage of Total Rate
Pneumonia, all forms	127.0	100.0	102.1	100.0
Bronchopneumonia	48.7	38.5	45.2	44.2
Lobar pneumonia	55.9	44.0	50.4	49.5
Undefined pneumonia *	22.3	17.5	6.5	6.3

* A campaign on the part of the Bureau of the Census for exact statement has significantly reduced this item in recent years.

Pneumonia occurs in all *climates*: it is prevalent in tropical as well as in cold countries. Like other communicable infections, it shows geographic differences: thus, it is more virulent in the United States than in England. This is said to be due in part to the dry, overheated air of our homes, offices and work rooms.

Pneumonia shows a distinct *seasonal prevalence*: it is most frequent in the winter and spring months. This is not necessarily accounted for by the chilling effects of cold moist air, for Greenberg has shown that a low relative humidity is conducive to a high death rate. Cold, wetting and chilling are generally regarded as important predisposing causes. They may favor auto-infection through inspiration, or may lower the resistance of the bronchial and pulmonary tissues. Fatigue, or some unusual exertion, also plays a part here. Pneumonia is often preceded by an ordinary catarrhal inflammation. There are other accessory factors which predispose to pneumonia, such as alcohol, trauma, irritating dusts and gases, and diet. The influence of diet is clearly brought out from susceptibility to pneumonias in scurvy and rickets. Cold and crowding also favor the spread of other respiratory infections, such as pneumonic plague, common colds, sore throats, etc.

Pneumonia is fatal among negroes to a greater extent than among whites; and is more frequent in males. It attacks all ages. The incidence is marked at both extremes of life. It is common in children under six years; between the sixth and fifteenth year the predisposition is less marked, but for each subsequent decade it increases. Osler says, "Pneumonia may well be called the friend of the aged. Taken off by it in an acute, short, not often painful illness, the old escape those 'cold gradations of decay' that make the last stage of all so distressing." Pneumonia often attacks the strong and robust in early

adult life, but under these circumstances the chances of recovery are good.

Pneumonia ordinarily does not seem to be a contagious disease. The infection, however, is spread rather directly from man to man. The pneumococcus does not thrive in the outer world, and man therefore must be regarded as its source and reservoir. Pneumonia clearly belongs to the great group of contact infections spread by secretions from the respiratory tract; it shows, however, marked individualities, the chief of which is its feeble tendency to focal concentration, a tendency so characteristic of diseases spread by personal contact. The broadcast distribution of the disease finds its explanation in part in the prevalence of pneumococcus carriers, and in part in the fact that susceptibility, predisposing causes and accessory factors play an important rôle in determining the disease.

Types of Pneumococci.—Four groups or types of pneumococci are now recognized, based upon immunological reactions. In three of the groups these reactions are fixed and specific. This grouping is important both from the standpoint of treatment and prevention.

INCIDENCE OF VARIOUS TYPES OF PNEUMOCOCCI IN HEALTHY PERSONS, IN CASES OF PNEUMONIA, AND RESULTING MORTALITY *

Type	Carriers among the General Population, per Cent	Percentage of All Pneumococ- cic Lobar Pneumonia	Case Fatality, per Cent
Type I.....	1.8	33.	25.
Type II.....	5.1	31.	32.
Type III.....	8.4	12.	45.
Type IV.....	41.8	24.	16.

* For data see Powell, Atwater and Felton, *Am. J. Hyg.*, 1926, 6:4. Also Monogr. No. 7, Rockefeller Inst., 1917, p. 33.

Types I and II together cause about 65 per cent of the lobar pneumonia in the United States. These types are rather strictly limited to the case and its immediate vicinity, appearing in the throats of case contacts more frequently than among the general population. Type III carriers are found more frequently than either Type I or II. Type III is the *Pneumococcus mucosus*, formerly confused with the *Streptococcus mucosus*. It is distinguished not only by specific immunological reactions but by the fact that it has a large capsule and the colonies on solid media form an abundant, sticky, mucoid growth. Group IV comprises all pneumococci that do not belong to Types I, II or III. This is a heterologous group of many different strains. They are frequently found in normal mouths. These organisms have a comparatively lower virulence, with a case fatality rate of 10 to 15 per cent. They cause about 20 per cent of cases, but under certain conditions strains of Group IV may have an exalted virulence as in the Rand miners in South Africa.

Types I, II and III seem quite stable, show little tendency to vary, and are therefore properly spoken of as fixed types. The percentage of incidence and

the death rates naturally vary with time and place; however, the figures given above, which are for the United States, have been fairly consistent from year to year, and the figures obtained by others have not varied widely from those stated.

Resistance of the Virus.—The pneumococcus is a frail organism; it does not multiply in nature outside of the body and indirect transmission is not likely except with fresh infectious material. Even upon artificial culture media the life of the pneumococcus is brief; it must be transplanted frequently in order to keep it alive; it is customary in laboratories to pass it through a susceptible animal, such as a mouse or rabbit, from time to time, in order to maintain its virulence.

The pneumococcus is readily destroyed by heat; 52° C. for ten minutes is sufficient. On the other hand, it withstands low temperatures very well. The ordinary germicidal agents destroy it quickly and with certainty. It may live for months in dried sputum, in which it also maintains its virulence. It has been found in the dust of rooms occupied by cases or carriers, and may persist for weeks.

Modes of Transmission.—The pneumococcus leaves the body mainly in the discharges from the mouth and nose, and enters the system through the same channels. It is assumed, therefore, that this represents the mode of transmission. It must, however, be admitted that there are many factors and features concerning pneumonia that are obscure and puzzling. Evidently the pneumococcus is spread from mouth to mouth by contact with cases or carriers. Our extensive search in Boston⁹⁰ failed to trace the connection between one case of pneumonia and the next, but showed an increased prevalence of the fixed-type carriers among normal persons associated with cases.

Pneumonia as a complication of influenza is one of the chief causes of death when that disease is pandemic. Pneumonia may be considered as recurring in epidemic form during the cold weather in almost every place having a cold winter season. Otherwise, extensive epidemics of pneumonia do not occur independent of influenza, measles, etc. Outbreaks have been described in hospitals, houses and institutions, in camps and on shipboard. Ordinarily, the disease shows little tendency to develop in those immediately in contact with cases. The excessive prevalence of pneumonia so commonly found in camps, barracks and among working people, as at Panama and the African mines, is believed to be due primarily to crowding of a particularly susceptible group in the presence of specific virulent pneumococci.

Stillman⁹¹ found pneumococci in the dust of houses and hospitals, usually correlating with the type present in the case.

Carriers.—Pneumococci are frequently found in the mouths and throats of healthy persons. Sternberg in 1880 first demonstrated a pneumococcus in his own saliva. Netter found it in 20 per cent of the persons whom he examined, and the New York Commission reported its presence in from 48 to

⁹⁰ M. J. Rosenau, L. D. Felton, R. M. Atwater, *Am. J. Hyg.*, 1926, 6, Nos. 3, 4.

⁹¹ *Monogr. No. 7, Rockefeller Inst. M. Res.*, 1917, 96.

85 per cent. In other words, the majority of persons seem to be pneumococcus carriers. One fortunate feature of this situation is the fact that most of the common mouth pneumococci belong to Group IV, which has a comparatively lower virulence.

From our Boston studies it may be taken as established that practically every person becomes a carrier of a fixed type pneumococcus at some time during the course of a year.

Pneumococci of Types I, II and III usually disappear from the mouths of convalescent cases within a few days or weeks, the longest persistence noted in the literature being eighty-three days. Healthy carriers have been shown by our studies to carry these fixed type organisms usually not more than thirty days, though instances of persistence for sixty, ninety and even for 120 days have been observed.

Type III pneumococcus occurs among normal persons more frequently than any other of the fixed type pneumococci, yet this organism is responsible for but a small part of the pneumonia due to fixed type organisms; about 12 per cent. This is only one of the paradoxes that puzzle us in the study of pneumonia.

The best we can say at present is that pneumonia occurs when there is a conjunction of susceptibility and the presence of one of the specific cocci. No known measures will relieve pneumococcus carriers.

Immunity.—One attack of pneumonia does not confer a high or lasting immunity. Man, however, must possess a certain degree of resistance to the pneumococcus, else the disease would be more prevalent than it is, and recovery would probably be less frequent. Recurrence is more common in pneumonia than in any other acute disease. Instances are on record of individuals who have had ten or more attacks. Rush gives an instance in which there were twenty-eight attacks. The pneumococcus seems to be in a class with the streptococcus of erysipelas and the streptococcus of rheumatism so far as recurrences and failure to produce immunity by one attack are concerned.

All kinds of animals, even the most susceptible, may be rendered actively immune to pneumococcus infection by the previous injection of non-lethal doses of living pneumococci, or even by the injection of the dead cocci. The serum of such actively immunized animals, in many cases, possesses protective and even curative power.

The mechanism of the immunity to this infection is not at all understood. Phagocytosis may play a prominent rôle. Protective antibodies, especially lysins and agglutinins as well as precipitins, play a part. The pneumonia attack, especially the crisis, resembles an anaphylactic reaction. The mechanism of immunity in this infection is complex. It is quite specific for type.

Accessory Factors.—Many weakening diseases diminish resistance to the pneumococcus. Pneumonia is frequent in alcoholics and is commonly brought on by exposure to cold, to trauma, or to local irritation. It is a frequent complication of influenza, measles, whooping-cough, typhoid fever and other infections. Pneumonia often closes the scene in chronic heart disease, pulmonary phthisis, Bright's disease, diabetes and other debilitating affections.

Immaturity and old age, as well as other enfeebling conditions, may act as a predisposing cause by lowering immunity. Other factors which predispose to pneumonia are sudden changes in temperature, trauma, alcohol, irritation caused by aspiration of foreign substances, or the inhalation of dust or irritating vapors such as ether. Unbalanced diets, especially those that induce scurvy and rickets, predispose to infections of the lungs and upper respiratory tract.

It should be remembered that pneumonia, like other communicable infections, frequently attacks the strong and robust.

Fatigue, exposure and overexertion have long been recognized as contributing causes of pneumonia. Fatigue and overexertion favor the inspiration of infectious material into the trachea. Blake and Cecil⁹² have produced pneumonia in monkeys simply by injecting small amounts of a pure culture by means of a fine needle into the trachea. Kinyoun and Rosenau, also Meltzer and his colleagues have produced pneumonia by intratracheal insufflation of large amounts into dogs.

The pneumococcus is particularly virulent when it attacks races in which the disease has not been prevalent. This was the case with the laborers on the Panama Canal and the miners on the Rand. Such circumstances indicate that a certain amount of racial resistance is acquired through long conflict with the pneumococcus.

Prophylactic Vaccines.—Vaccines have been tried as a prophylactic against pneumonia. Theoretically, we might expect it to be a hopeless task to produce by artificial methods a useful immunity to a disease which leaves little or no natural immunity. On the other hand, a high degree of protection can easily be induced in susceptible animals to virulent cultures of pneumococci when injected into the peritoneal cavity, the blood stream, or subcutaneously. The results of preventive inoculation upon man are not encouraging.

Following the promising but inexact experiments of Wright in South Africa, Lister⁹³ carried out prophylactic immunization in a large number of Rand mine workers, using a composite vaccine made from pneumococcus types prevalent in that region. He found that subcutaneous injections protected against the types used in the vaccines.

Cecil and Austin⁹⁴ inoculated some 12,000 seasoned troops (20 per cent of the command) at Camp Upton, with a saline pneumococcus vaccine containing Types I, II, and III. Three or four doses were given at weekly intervals, the first containing three billion organisms, and the final doses from six to seven and one-half billions. While the period of observation after the inoculation was only ten weeks, evidence of protection was made apparent by the fact that no cases of pneumonia of Types I, II or III occurred in the vaccinated groups, while twenty-six cases due to these types originated in the unvaccinated

⁹² *J. Exper. M.*, 1920, 31: 403, 445, 499, 519, 657, 685; 1920, 32: 1, 401, 691, 719.

⁹³ *So. African Inst. for Med. Research*, 10, 1917.

⁹⁴ *J. Exper. M.*, 1918, 38: 19.

groups. Only seventeen cases of pneumonia of all types developed among the vaccinated as contrasted with 172 cases among the unvaccinated men.

Cecil and Vaughan⁹⁵ immunized 80 per cent of the command at Camp Wheeler. The conditions here differed materially from those at Camp Upton in several respects: (1) A larger number of the troops were recruits, and hence more susceptible;⁹⁶ (2) the situation was complicated by the influenza epidemic; (3) a lipovaccine was substituted for the saline vaccine. This vaccine contained the three types of pneumococci in a dosage of ten billion organisms, and only one injection was given. During a period of three months, 363 cases of pneumonia of all varieties occurred among the men vaccinated (80 per cent of the command), and 327 cases among the unvaccinated troops (20 per cent of the command). Only eight cases of Types I, II, and III developed among the vaccinated men, and these were all secondary to severe cases of influenza. This excellent opportunity could not be followed up, for it was in a complicated situation, and is therefore not conclusive.

Pneumococcic immunity is strictly specific. Vaccines protect only against the pneumonia caused by those groups represented in the vaccine. Much more work will have to be done before a statement can be made concerning the prophylactic value of these measures. Studies made by the Influenza Commission of the Metropolitan Life Insurance Company⁹⁷ upon groups involving many thousands in insane asylums in Massachusetts and New York failed to demonstrate protective value. This study disclosed the interesting fact that the recent inmates or newcomers into these institutions were especially liable to contract pneumonia. Antipneumococcus serum, particularly the refined and concentrated antibody solution, is useful in the treatment if given early, but not as a preventive.

Preventive Measures.—The prevention of pneumonia is still baffling for lack of a better understanding of the fundamental factors in the epidemiology of the disease. We cannot boast of success with an infection which is one of the chief causes of death.

We must keep in mind that the disease called pneumonia is a group of closely related infections which may require somewhat different methods of control. In Types I and II, the emphasis must be placed upon direct contact with cases and carriers, and also dust; in Types III and IV, these factors also obtain, but personal prophylaxis to prevent auto-infection may be especially important. Until we have more precise knowledge, our measures should be general enough to include all members of the group.

Upon the Isthmus of Panama pneumonia was unduly prevalent owing to overcrowding, which favors contact infection. The same was found by General Gorgas among the workmen of the African mines. The prevention consisted

⁹⁵ *J. Exper. M.*, 1919, 29: 457.

⁹⁶ E. L. Opie *et al.*, *J. Am. M. Ass.*, 1919, 72: 108, 556.

⁹⁷ The Commission for the Study of the Cause and Prevention of Influenza and Pneumonia of the Metropolitan Life Insurance Company, consisting of: Lee K. Frankel, A. S. Knight, W. H. Park, W. H. Frost, G. W. McCoy, E. O. Jordan, and M. J. Rosenau, Chairman.

in scattering the workmen, giving them separate homes in place of barracks. Allaying street dust and house dust removes one of the predisposing causes of pneumonia and other respiratory infections.

It should become common knowledge that anything which tends to reduce vitality predisposes to pneumonia, such as dissipation, loss of sleep, overwork, worry, poor or insufficient food, lack of exercise, alcohol, colds, or excesses of all kinds; the atonic effect of living in overheated rooms, and the injurious effect of excessively dried and warmed air, and sleeping in warmed rooms. Cold baths, regulation of temperature and ventilation, sleeping with open windows or in the open air, as well as oral hygiene, are assumed to be useful prophylactic measures for pneumonia as well as tuberculosis, "colds," and a large group of diseases.

More attention should be given to the minor acute respiratory infections such as ordinary "colds," so-called influenza, bronchitis, and sore throats. These catarrhal inflammations are often associated with pneumococci and predispose to pneumonia. Persons suffering with these "minor" infections should be isolated in bed during the acute stage and at least as long as there is fever. Exposure, overexertion, and fatigue under these circumstances may be hazardous.

The virulent pneumococcus should not be lightly regarded as a normal inhabitant of the mouth, throat, and nose. Because the pneumococcus is very widely spread and the disease is ubiquitous, and because the associated factors which determine infection seem complicated and not well understood, is no reason for a supine and hopeless attitude. The problem of tuberculosis has been attacked with vigor with scarcely better understanding of some of the fundamental problems at issue. If pneumonia were a new or exotic disease, it would be feared and quarantined, and aggressive measures taken to stamp it out. Each case of pneumonia should be regarded as a focus for the spread of the infection. We should think of pneumonia very much as we think of whooping-cough and influenza,—as an infection which is spread from man to man through the secretions of the mouth and nose. The isolation of cases of pneumonia could not be expected to be more than partially effective in controlling the disease, although it should decrease the number of responsible carriers.

The details for the control of the acute respiratory infections transmitted by the discharges from the mouth and nose are given under the prevention of diphtheria and the prevention of common colds.

INFLUENZA

(*La Grippe—Grip*)

Influenza is an acute, highly communicable, febrile disease, characterized by great pandemic outbreaks. Clinically, it is characterized by sudden onset, fever lasting about three days, and depression. There is a special tendency to bronchial and pneumonic complications. The cause of influenza is not known;

we therefore lack a criterion by which to recognize the disease. The term "influenza" has become both popular and vague. It is applied to common colds, acute catarrhal inflammation, brief febrile attacks of unknown origin, and even to nervous indispositions.

When influenza sweeps over the world in pandemic form, it becomes the most serious and furious of epidemics on account of the large numbers attacked in a short time. There is a high incidence, with a comparatively low case fatality rate, but the mortality is high on account of the great number of cases. Influenza itself probably never kills. Death is always due to some complication, usually pneumonia. Pandemic influenza acts something like measles in a virgin population.

In the world-wide pandemic of 1918-1919, it is estimated that there were over 200,000,000 cases and that upwards of 10,000,000 deaths occurred in less than twelve months; in the United States alone there were more than 20,000,000 cases and about 450,000 deaths in less than six months. In India, 4,933,132 deaths from influenza were reported from June to November, 1918.

The early history of influenza is necessarily veiled in obscurity. Hirsch in his great work⁹⁸ gives a summary of periods when influenza was pandemic. There is an account of an epidemic, probably influenza, recorded in 1173. Many outbreaks of "plagues," some of them doubtless influenza, occurred long before this. The first authentic outbreak was described in 1510 by the famous physicians Willis and Sydenham. There have been about eighty epidemics, more or less authentic, since 1173. Fourteen pandemics have been recorded since 1510; they are those of 1510, 1557, 1580, 1593, 1729, 1732, 1762, 1788, 1830, 1833, 1836, 1847, 1889 and 1918. The disease appears to have been epidemic in North America in the years 1627, 1647, 1729, 1732, 1737, 1762, 1782, 1789, 1811, 1832, 1850, 1857, 1860, 1874, 1879, 1889-90, 1916 and 1918. The relation of sporadic and interepidemic outbreaks to pandemics of influenza is not clear. Scarcely a year passes, particularly during the past century, without news of the epidemic occurrence of a disease popularly called "influenza" at some point or other of the earth's surface. There is confusing lack of uniformity in the registration of cases and deaths from this disease.

Etiology.—Influenza is not a clean-cut clinical entity. The manifestations are extraordinarily complex. The fever lasts three days; if prolonged, some complication should be suspected, especially bronchitis or pneumonia. The attack often starts abruptly: hence the term "grip." The prostration is out of all proportion to the fever and lesions. Cough and pains in the head, back and limbs are almost always present. There is a leukopenia. The disease itself is rarely, if ever, fatal, death being due almost always to pneumonia. The clinical features of each outbreak vary; usually the respiratory form predominates. In the 1889-90 epidemic, neuritis and the "nervous form" were common. A gastro-intestinal form is described. In the recent pandemic, pneumonic complications were conspicuous and the disease was unusually viru-

⁹⁸ *Handbook of Geographical and Historical Pathology*, London, 1885. See also Jordan, E. O.: *Epidemic Influenza*, Am. Med. Assn., 1927.

lent in pregnant women. The diagnosis of influenza from the clinical standpoint is not trustworthy. There is no criterion by which the infection may be recognized. Until the cause is established, we cannot know the relationship between influenza and the ordinary cases of "catarrhal fever," "grip," common colds, and "influenza colds."

The cause of influenza is not determined. Filterable viruses have been described⁹⁹ and denied.¹⁰⁰ In 1892, Pfeiffer¹⁰¹ discovered the *Bacillus influenzae*—a small, Gram-negative, hemoglobinophilic, frail bacillus. This organism is found in about 30 per cent of normal throats and is practically always present in measles and whooping-cough. The percentage of times it is found in influenza varies with the observer, many bacteriologists finding it almost constantly in the recent outbreak. The etiological relation to the disease is doubtful; however, the recent experience has greatly raised our respect for Pfeiffer's bacillus, for if it is not the cause of influenza, it may become a virulent secondary invader, and the cause of serious complications. Olitsky and Gates cultivated filtrable bodies as the cause of influenza.¹⁰²

Mode of Infection.—It is assumed that the virus leaves the body in the secretions from the mouth and nose, and enters through the same channel. Therefore, it is probable that the infection is contracted through direct and indirect contact and droplet infection. Lynch and Cumming¹⁰³ believe that indirect contact and hand-to-mouth infection are the chief modes of spread. Water, milk, and food are not known to carry the virus. Rosenau, Goldberger and McCoy¹⁰⁴ were not able to transmit the disease to volunteers.

Occurrence.—Influenza attacks all ages and both sexes. During the 1918-1919 pandemic it was most fatal during the age period twenty to thirty, and seemed to have a predilection for strong, sturdy, robust young men. More females than males were affected, but the death rate was higher among males. It spares neither class nor race; it takes the rich and poor alike, the strong and the weak, the clean and the dirty. Hygiene and sanitation therefore have practically no effect in controlling diseases like influenza, measles and smallpox.

Influenza occurs as great pandemic waves at irregular intervals of a generation or so. These are followed by years of epidemic and sporadic prevalence—the etiologic relationship is not established. A pandemic cycle occurs in succeeding waves lasting about five years. The secondary waves, according to Brownlee,¹⁰⁵ occur at intervals of thirty-three weeks, or multiples thereof.

⁹⁹ Nicolle and LeBailly, *Compt. rend. Acad. d. Sc.*, 1918, 167: 607. Dugaric de la Rivière, 606. P. K. Olitsky and F. L. Gates, *J. Exper. Med.*, 1921, 33: 125, and others.

¹⁰⁰ M. J. Rosenau, *et al.*, "Some Interesting though Unsuccessful Attempts to Transmit Influenza Experimentally," *U. S. Pub. Health Rep.*, 1919, 34: 33; also *Hyg. Lab. Bull.* No. 123, Feb. 1921. A. W. Williams, M. Nevin, and C. R. Gurley, *J. Immunol.*, 1921, 6: 5. S. E. Branham, and I. C. Hall, *J. Infect. Dis.*, 1921, 28: 143.

¹⁰¹ *Deutsch. med. Wchnschr.*, 1892, 2: 28. *Ztschr. f. Hyg.*, 1893: 13.

¹⁰² *J. Am. M. Ass.*, 1921, 76: 740. *J. Exper. M.*, 1921, 33: 3.

¹⁰³ *Mil. Surgeon*, Dec. 1918; other references, page 251.

¹⁰⁴ *U. S. Pub. Health Rep.*, 1919, 34: 33; also *Hyg. Lab. Bull.*, No. 123, Feb. 1921.

¹⁰⁵ *Lancet*, Nov. 8, 1919.

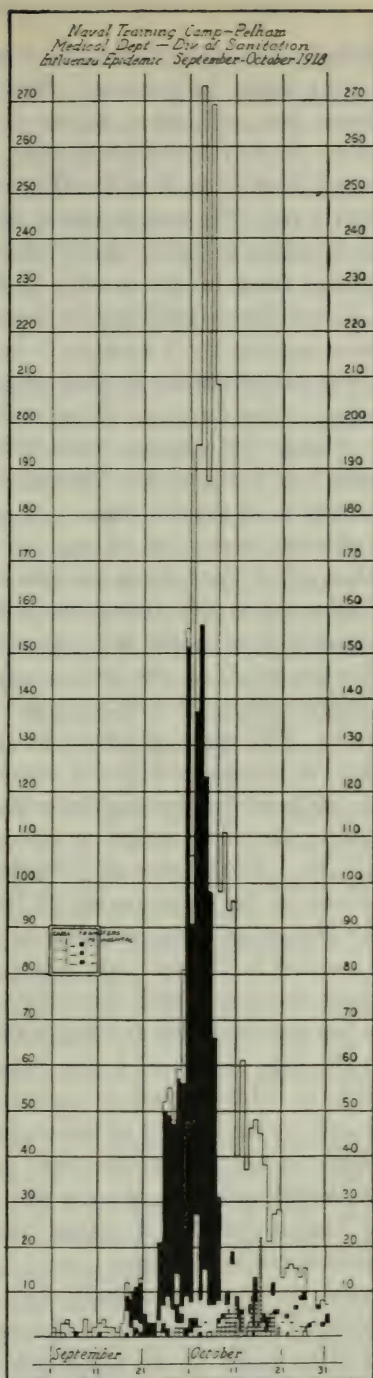
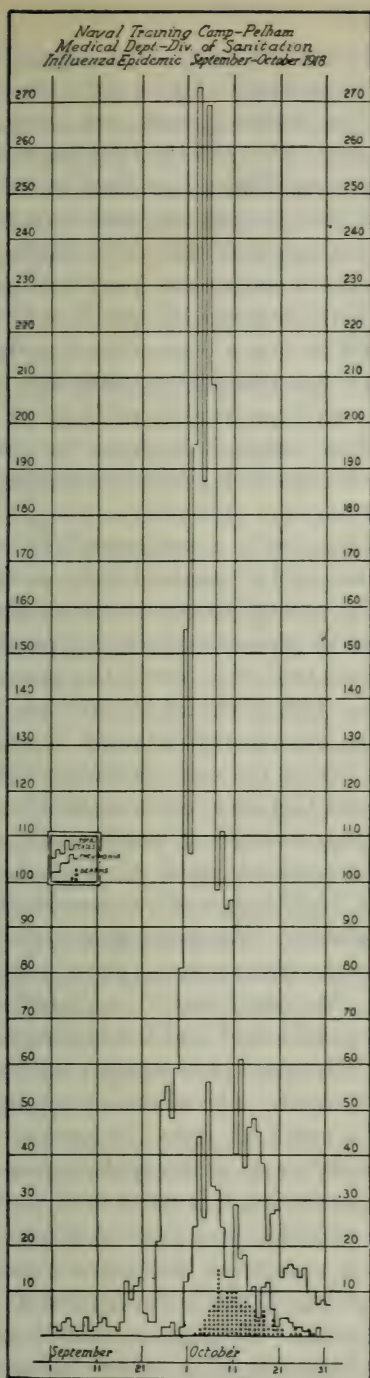


FIG. 14.—CURVE OF THE INFLUENZA EPIDEMIC IN THE NAVAL TRAINING CAMP AT PELHAM PARK, NEW YORK, SEPTEMBER AND OCTOBER, 1918; SHOWING (1) CASES OF INFLUENZA, (2) CASES OF PNEUMONIA, AND (3) DEATHS.

Note the steeple-like appearance of the curve, its symmetrical character, the short duration and explosive nature of the outbreak.

When pandemic, the disease attacks 13 to 53 per cent of the population, averaging about 30 per cent. The incidence on board ship or other limited localities runs as high as 84 per cent.¹⁰⁶ The disease spreads with amazing rapidity. It travels ordinarily from east to west: the 1889-1890 pandemic was supposed to start in Russia. Other world-wide epidemics have been traced to the Far East. The last pandemic was called Spanish influenza because it first came to notice in Spain, but probably did not originate there. It started in malignant form in this country in Boston on Commonwealth Pier, then used as a Naval Receiving Ship, in September, 1918, and rapidly spread over the entire country. By November it was difficult to find a community anywhere that was not affected. By January it was on the wane, and by March it had subsided. The duration of an epidemic in any one locality is from six to eight weeks. In compact communities, such as camps, it may run its course in from four to six weeks. No other disease attacks such a large proportion of the people in so short a time.

Influenza occurs at all *seasons* of the year, with a preference for cold weather. Del Pont, from an historical review of 125 pandemics, shows that fifty occurred in the winter, thirty-five in the spring, sixteen in the summer, and twenty-four in the fall. It is conveyed by human contact, independent of climate, wind, or weather, except that secondary waves avoid the summer. Epidemics break out with *explosive violence*. This is one of its chief characteristics. The curve is apt to be steeple-like (see chart), although it shows marked variations in different communities during the same pandemic. Epidemic outbursts of this character usually mean that some common medium is spreading the virus which is infecting a large number of people about the same time. In the case of influenza, the explosive character of epidemics is explained by the short period of incubation, high degree of communicability of the disease and susceptibility of the population. The rapid spread is further assisted by the fact that many who have the disease do not go to bed, but continue to mingle with their fellow men. Secondary waves in a pandemic cycle are less explosive and the curve is apt to be longer and less symmetrical than the first onrush of a new pandemic. The curve of a primary outbreak is uniform, while secondary waves may be variable. The disease is apt to be mild and uncomplicated in the beginning of a pandemic cycle. It increases in virulence to the peak of the wave and again decreases as the epidemic wanes.

Although influenza spreads with amazing rapidity, it is not known to outstrip human travel. It moves from place to place in a pair of shoes. It sometimes seems to outrun travel; at least it is reported to break out simultaneously in widely separated parts of the globe. Thus, in September, 1918, it appeared simultaneously in Boston and Bombay. The vagaries of epidemics have given point to the name influenza, referring to some hidden influence.

No other disease is more *disabling* to community life, for the reason that so many are placed upon the non-effective list at the same time. Trade and

¹⁰⁶ U. S. S. Yacoma in 1919: 80 of 95 persons stricken within about one week.

travel become temporarily paralyzed; there is a shortage of doctors and nurses; hospital facilities are inadequate. The lesson of the past pandemic should teach us to mobilize the medical, nursing, hospital, and social service facilities in anticipation of such emergencies.

The *incubation period* is usually short—twenty to forty-eight hours. The disease is presumably most communicable during the early stages. *Carriers* may spread the infection and are suspected. If the *Bacillus influenzae* is the cause of the disease, then bacillus carrying is exceedingly common. We do not know how long a person remains infective and little is known concerning carriers.

The question of *immunity* is not settled; it does not last a year. Second and third attacks were said to occur in 1889-1890, but were uncommon in 1918-1919. Some persons have recurring "influenza-like" attacks yearly. Vaughan's studies in Boston¹⁰⁷ and Jordan's studies at Camp Grant¹⁰⁸ indicate that immunity does not last more than seven months.

During epidemics of influenza, other diseases do not prevail to the same extent. On the other hand, influenza is supposed to depress resistance so as to favor or hasten certain infections such as tuberculosis; of this, however, we lack proof. There seems to be an epidemiological relationship between influenza and cerebrospinal fever. The influenza bacillus is sometimes found in pure culture in meningitis, which may also occur as a complication of influenza. *Encephalitis lethargica* also bears an epidemiologic relation to epidemics of influenza.

A disease called influenza was prevalent and fatal among horses in 1919, and the cause of much trouble to the army supply service. It also prevailed as an epizootic in this country in 1889-1890. Other distempers among animals resemble influenza. The relationship to man is not known.

Crowding and human contact seem to favor its spread, although it travels with surprising rapidity through rural communities.

Vaccines.—Two classes of vaccines are used to protect against influenza: (1) influenza bacilli, (2) mixtures of pneumococci (Types I, II, III and IV), hemolytic streptococci, staphylococci aurei, influenza bacilli, and other microorganisms.

Contradictory results have been reported with both types of vaccines. My own observations at Monson, Pelham Park Naval Training Station, and elsewhere lead me to conclude that vaccines made with dead cultures of Pfeiffer's bacillus have no protective value whatever. Whether the polyvalent vaccines prevent pneumonic complications has not been established.

Administrative Measures.—*Isolation* is possible, but not practical during epidemics. In 1889-1890 some large institutions were saved through a strict quarantine, which of course is impracticable for cities. Once within the walls, it soon gets beyond control. In 1918-1919, the infection was kept out

¹⁰⁷ *Am. J. Hyg.*, Monog. No. 1, July, 1921.

¹⁰⁸ *J. Infect. Dis.*, 1920, 26: 463.

of Goat Island in San Francisco Harbor, but finally got in. Several other localities had a similar experience.

Patients for their own good should remain in bed during the febrile stage. This one measure would help control the spread of influenza as well as common colds. It is quite worth while to isolate the first case in a household in order to prevent a house epidemic. This may be done on lines precisely parallel to those described for diphtheria.

Masks.—During the 1918-1919 outbreak, face masks were compulsory in San Francisco, Sacramento, and a few other cities. They did not lessen the incidence nor shorten the course of the epidemic.

Closing of schools, theaters, and other places of assembly, and regulations to prevent crowding were enforced in many cities during the epidemic of 1918-1919, but so far as can be judged had no favorable effect whatever.

Hospitalization is not always advisable, for the condensation of many cases seems to favor the number and severity of complications. This same phenomenon is seen with measles. It is believed that pneumonic complications are sometimes contracted in large hospitals.

There may be unknown factors in the spread of influenza so that our preventive measures lack precision and confidence. We have no specific prophylactic. So far as can be judged, the measures taken during the epidemic of 1918-1919 did not control the disease to any appreciable degree.

The details for the control of the acute respiratory infections transmitted by the discharges from the mouth and nose are given under the prevention of diphtheria and the prevention of common colds.

COMMON COLDS

More people probably suffer from common colds than from any other single ailment. Vital statistics give no hint of the prevalence and importance of these minor affections because the mortality is nil and the morbidity records are notoriously imperfect and difficult to collect. Could the sum total of suffering, inconveniences, sequelæ, and economic loss resulting from common colds be obtained, it would at once promote these infections from the trivial into the rank of the serious diseases.¹⁰⁹

Between 10 and 15 per cent of the population at large are afflicted with colds at one time. Most persons have from one to three or more a year. There is a marked difference in individual susceptibility.

The common colds here considered are a group of acute infections of the mucous membranes of the nose and throat, often extending into the larynx, trachea, or larger bronchi. A common cold is not merely a congestion, it is an acute infectious disease. The sinuses are frequently involved. The infection may localize itself in the mucous membrane of the nose, rhinitis; in

¹⁰⁹ Townsend has reviewed the literature on common colds to 1924. *U. S. Pub. Health Rep.*, Suppl. No. 48, 1924.

the tonsils, tonsillitis; in the pharynx, pharyngitis; in the larynx, laryngitis; in the trachea, tracheitis; in the sinus, sinusitis; etc.

Congestion and inflammation of the mucous membrane of the upper respiratory tract frequently occur as a result of irritants other than bacteria. Thus, chemical and mechanical irritants will produce a congestion or inflammation; an increased acidity causes a flaring up of the mucous membranes, especially of the nose; and many other local and reflex causes lead to acute or chronic catarrhal conditions of these membranes, which may become exquisitely sensitive and sometimes hypersusceptible. In the absence of the proper microorganism, however, these conditions do not develop into infectious colds, and are, therefore, not communicable.

The popular fallacy of colds being due to exposure to drafts, sudden changes of temperature, and chilling of the body clings persistently in both the professional and lay mind. These are predisposing causes but they cannot generate the specific microorganism: that would be spontaneous generation. The bacteria usually found associated with these catarrhal infections are: staphylococci, streptococci (viridans and hemolyticus), pneumococci, influenza bacilli, the Gram-negative cocci classed together as members of the micrococcus catarrhalis group, diphtheroid bacilli, and other microorganisms. The etiological relationship between these organisms and the disease is not at all clear. Many of the above-mentioned bacteria are also found normally upon the mucous membranes of the nose, mouth, throat, and upper respiratory passages; auto-infections may, therefore, be common, and if this be so, predisposing factors which diminish resistance have special importance.

Tunncliffe¹¹⁰ described a *Bacillus rhinitis* associated with acute and chronic rhinitis. Experimental inoculations of pure cultures produced colds.

Foster¹¹¹ working in my laboratory, confirmed and extended Kruse's¹¹² observations, showing the presence of a filtrable virus in the secretions from the nose in common colds prevailing in Boston in 1915. This filtrate, as well as subcultures grown in selective media, when dropped into the nostril of healthy persons, produces a cold. Evidently there are different types of colds, due to different viruses, or to the associated bacteria. A number of negative results have also been recorded.¹¹³

Colds are contracted from other persons having colds, just as diphtheria is contracted from diphtheria. Arctic explorers exposed to all the conditions ordinarily supposed to produce colds do not suffer from these ailments until they return to civilization and become reinfected by contact with their fellow-men. A campaign to prevent the spread of the common cold would have much collateral good in aiding the suppression of tuberculosis and causing a diminution of pneumonia and other infections. Common colds occur in

¹¹⁰ *J. Infect. Dis.*, 1913, 13: 283.

¹¹¹ *J. Am. M. Ass.*, 1916, 66: 14.

¹¹² *München. med. Wchnschr.*, 1914, 61: 1547.

¹¹³ W. H. Park, A. W. Williams, and C. Krumweide, *J. of Immunol.*, 1921, 6: 1, and others.

epidemics and have all the earmarks of a contagious disease. Colds are apt to go through all the members of a household, and outbreaks in schools, factories, and other places where people are closely associated, frequently occur and result in considerable loss of time and money.

While common colds are never fatal, the complications and sequelæ are serious. These are rheumatic fever, pneumonia, sinusitis, nephritis, endocarditis, myocarditis, pericarditis, and a depressed vitality which favors other infections and hastens the progress of organic diseases.

Common colds are perhaps most contagious during the early stages. If persons would isolate themselves by remaining in bed during the first three days of a cold, they would not only benefit themselves, but would largely prevent the spread of the infection. The contagiousness and severity of colds vary greatly in different epidemics and in different seasons of the year, depending upon the particular microörganism involved and other factors not well understood.

Drafts and Chilling.—Drafts in themselves cannot produce an infectious cold. The first symptom of the disease is a chill, which is not the cause, but the effect, of the infection. It is a common belief that the cold is caught when the chill occurs. The rigor frequently consists of only a transient chilliness, and it is during this time that the individual is sensitive to drafts which he thinks are producing his cold (see page 805).

A large number of investigators, including Lipardi,¹¹⁴ Lode,¹¹⁵ Pasteur,¹¹⁶ Kline and Winternitz,¹¹⁷ the New York State Commission on Ventilation,¹¹⁸ Miller and Noble,¹¹⁹ and others, have experimented variously with animals and man, and all are in accord with the general conclusion that exposures to sudden changes of temperature lessen the resistance of animals to infection. Negative results are also found in the literature. It seems evident that the exposure of a portion of the body to cold is more likely to be followed by acute respiratory infections than when the entire body surface is subjected to the same low temperature, and the popular idea that drafts predispose to respiratory infections in some individuals is therefore not without foundation.

Chilling causes vasomotor contraction of the capillaries of the skin, which is doubtless designed to conserve body temperature; coincidently there is turgidity of the erectile tissue of the mucous membrane of the turbinates, which is probably a defensive action. This congestion partly closes the nose and causes snuffling and increased secretion, which is ordinarily called a cold. A great variety of mechanical, chemical and even psychic stimuli will produce congestion of the cavernous tissue over the turbinate; in fact, the mucous membrane of the nose may become very sensitive, even hypersusceptible. Anaphy-

¹¹⁴ Morgagni, 1888, 30: 523, 575, 651.

¹¹⁵ Arch. Hyg., 28: 344.

¹¹⁶ R. Vallery-Radot, *Life of Pasteur*, New York, 1902, Vol. II, p. 61.

¹¹⁷ *J. Exper. M.*, 1915, 21: 304.

¹¹⁸ *J. Am. M. Ass.*, 1916, 1201.

¹¹⁹ *J. Exper. M.*, 1916, 34: 223.

lactic reactions to pollen and proteins are common manifestations of the nasal mucosa.

It has always been assumed that the contraction of the capillaries of the skin caused by chilling is attended with congestion of the internal organs, but Mudd and Grant ¹²⁰ have shown that chilling of the skin causes vasomotor contraction and ischemia of the mucous membrane of the tonsils, palate, and pharynx, as well as the skin, with a drop in temperature and a subsequent increase in the bacteria on these parts. On rewarming the subject, the tonsils tend quickly to recover their blood supply, in some instances actually becoming hyperemic; the skin returns to about its normal condition, but the palate and pharynx remain somewhat ischemic. This may explain why chilling so often seems responsible for sore throat, which in this case would be an auto-infection.

Chilling has a great variety of effects. In some persons it causes diarrhea, in others neuralgic or muscular pain; sometimes it seems to be responsible for nephritis, bronchitis, pneumonia, etc. It brings out a latent malaria (page 288). Chilling is much less apt to cause harm if accompanied with active muscular exercise, which probably helps by keeping up the body temperature and vasomotor tone.

Drafts are much less apt to injure persons in good physical tone. They are, however, injurious to infants, the aged, and to susceptible individuals. Drafts are particularly apt to harm persons accustomed only to still, warm air. "It is not the engine drivers and firemen of trains that catch colds, but the passengers in the stuffy carriages." Coddling renders one susceptible to drafts, partly for the reason that the vasomotor impulses which contract the blood-vessels of the skin are not sent out by the nervous mechanism, and consequently undue cooling of the part blown upon, and perhaps of the blood itself, takes place. Normally, when the wind blows upon the skin the vasomotor contraction reduces the supply of blood and the tendency to cooling is further met by a stimulus which increases heat production. While it is true that a draft can no more cause an infectious cold than it can cause diphtheria, nevertheless it is true that a draft may increase the severity of a cold or be the predisposing cause by which immunity is lowered, thus favoring auto-infection. It is noteworthy that colds so contracted have little or no contagious tendency.

It is a mistake to think that the skin alone is involved in the question of drafts. The "hardening" of the skin in order to prevent colds is, therefore, a misnomer. The good effects of cold baths, exercise, fresh air, sunlight, and wholesome food do not consist in "hardening" the skin, but in improving the nutrition, stimulating the metabolism, helping the control of the nervous system, improving the tone of the vasomotor system, strengthening the musculature, and enriching the blood. In preventing the ill effects of drafts, therefore, the entire organization of the body must be considered, and not the skin alone.

¹²⁰ *J. Med. Research*, 1919, 40: 53; *Exper. M.*, 1920, 32: 87.

Prevention.—The prevention of colds consists, first, in avoiding the infection, and, secondly, in guarding against the predisposing causes. Contact should be avoided with persons who have colds, especially in street cars, offices, and other poorly ventilated spaces where the risk of persons coughing or sneezing directly in one's face is imminent. Contact with the infection may further be guarded against by a careful self-education in sanitary habits and cleanliness based upon the modern conception of contact infection.

Colds, like other diseases conveyed in the secretions from the nose and mouth, are often transferred by direct and indirect contact through lack of hygienic cleanliness and a disregard of sanitary habits. Kissing, the common drinking cup, the roller towel, pipes, toys, pencils, fingers, food, and other objects contaminated with the fresh secretions will transmit the disease. Spitting ordinances should be enforced, ventilation and overcrowding of street cars corrected, and dust allayed.

The predisposing causes of colds include a number of conditions that depress vitality and thereby diminish resistance. The principal predisposing factors in "catching" cold are: vitiated air, dust, drafts, sudden changes of temperature, exposure to cold and wet, overwork, loss of sleep or insufficient rest, improper food, and other conditions that lower the general vitality of the body. On the other hand, it must be clearly kept in mind that vigorous persons in prime health are apt to contract a cold if they receive the infection. Some persons enjoy a specific immunity, and others lack this in high degree. It is a matter of common observation that some persons practically never contract a cold, while others have a distressing succession of them. One attack evidently does not protect against subsequent colds; in fact, there is epidemiological evidence which suggests that persons have a succession of four, five or more colds before they build up a resistance which lasts several years, when the process repeats itself. Resistance may be strengthened by good hygienic habits, especially diet, rest, sunshine and fresh air.

Other important predisposing factors to colds are mechanical defects in breathing, or the filtering power of the upper respiratory passages, also local pathological conditions, such as adenoids, polypus, enlarged tonsils, deviation of the septum, chronic catarrhal conditions, all of which should receive appropriate treatment.

One of the important predisposing factors to cold is breathing vitiated and dusty air. Good ventilation, therefore, with air not too dry nor too warm, and the allaying of dust would prevent many a cold. The bacteria accompanying colds are frequently found in the mouth, nose, throat and teeth of persons in good health. Cleanliness and care of these parts is, therefore, a consideration in the prevention of the complications of common colds.

The prevention of the other diseases in this group spread through the secretions from the mouth and nose follows the lines laid down for common colds. During epidemics individuals should avoid theaters, mass meetings, closed and crowded cars, and close contact with their fellow men, especially those who have catarrhal symptoms or fever. Hand-to-mouth infection should

be remembered, and the hands washed before eating and the fingers kept away from the mouth and nostrils.

Man is the source and fountainhead of these infections. Each case is a focus for their spread. Isolation and disinfection of discharges are the first indications. Limiting the number of persons who come in contact with the patient will lessen the number of carriers. These infections are also spread by mild and missed cases, and particularly during the early stages of the severe cases.

As carriers doubtless play an important rôle in disseminating these infections, the education of the public concerning certain sanitary habits should be actively continued. These include the danger of spitting promiscuously and of kissing; the proper care to be exercised in sneezing and coughing; the peril in the common drinking cup, the roller towel; and the habit of placing unnecessary things in the mouth, especially the fingers. Restaurants, hotels and soda fountains should be required to scald all glasses, cups, spoons, etc., every time they are used. Lynch and Cumming¹²¹ emphasize the importance of the sputum-borne diseases through hand-to-mouth infection and the scalding water used to wash messkits, and dishes in hotels, restaurants, etc.

Crowding, especially of the kind that favors the spread of the buccal flora from mouth to mouth, must be discouraged. Persons who sleep, work and play together, wash from the same bowl and eat with the same tableware have every opportunity of spreading infections of the upper respiratory tract. This is why acute infections of the mucous membrane of the upper respiratory tract are common and often epidemic in camps, schools, prisons, institutions, ships and industrial establishments. The factor of crowding and close personal contact needs emphasis and attention.

This group of infections should be added to the list of diseases requiring compulsory notification. Cases should be isolated at least in the same sense that diphtheria is isolated—the discharges from the nose and throat should be burned or disinfected. If the patient is treated at home, the house should be placarded in order to discourage visiting and as an educational measure.

Isolation in hospitals may be accomplished by proper nursing technic, including the disinfection of discharges and all objects soiled with the infectious materials, especially fabrics, hands, spoons, cups, thermometers, etc. Sanitary isolation may be favored by the use of cubicles or screens between beds, and also by the use of masks by patient, doctor, nurse and attendants. Hospitals should take active measures to prevent cross-infection and institutional outbreaks. It appears that pneumonia complicating measles, influenza and other diseases may possibly spread in hospitals, unless cases are isolated and guarded with special care. Convalescence is often protracted, and subject to complications. Ample time should therefore be given to full return to health.

Health officers may assist by disseminating knowledge concerning the

¹²¹ *Mil. Surgeon*, Dec., 1918; *Am. J. Pub. Health*, 1919, 9: 25; *J. Lab. & Clin. Med.*, 52, No. 6; *Mil. Surgeon*, Oct., 1919; *Am. J. Pub. Health*, 1919, 9: 849; *Mil. Surgeon*, Feb., 1920; *Am. J. Pub. Health*, 1920, 10: 849.

disease, by enforcing antisputting regulations, by proper cleansing and oiling of streets, by requiring a stricter compliance with building and housing laws, and by the regulation of the ventilation and condition of the air in theaters, schools, street cars, and public buildings, as well as the crowding of such places; also by providing free facilities for laboratory diagnosis, to aid in the search for carriers, to assist diagnosis and serum therapy, and to trace epidemics.

CEREBROSPINAL FEVER

Cerebrospinal fever is an infection with the meningococcus (*Diplococcus intracellularis meningitidis*, Weichselbaum). The essential lesions of the disease are chiefly focused upon the meninges of the brain and cord. The disease occurs both in localized epidemics and sporadically.

It is characterized by a clinical course of great irregularity, and a high case fatality rate. Recovery may be attended by distressing sequelæ. On account of the eruption which is often present, cerebrospinal fever was formerly called spotted fever, petechial fever, and malignant purpuric fever.

A clear distinction should be drawn between cerebrospinal fever and cerebrospinal meningitis: the former is caused by the meningococcus, the latter by a great variety of organisms, such as the tubercle bacillus, the pneumococcus, streptococcus, influenza bacillus, the colon bacillus, the typhoid bacillus, the bacillus of bubonic plague, of anthrax, and of glanders. The gonococcus may also cause meningitis as a secondary complication. The epidemic form, or cerebrospinal fever, is always due to the meningococcus.

The first epidemic outbreak of cerebrospinal fever was reported by Vieusseux in Geneva in 1805. The next year James Jackson, Thomas Welch, and J. C. Warren investigated an outbreak in Massachusetts. Since then numerous epidemics have occurred. In the New York epidemic of 1904-1905 there were 6,755 cases and 3,455 deaths. This was part of a pandemic that started in Europe and within five or six years spread over the entire globe and then became quiescent, but was again stirred to renewed activity by the war.

Cerebrospinal meningitis is spread chiefly by carriers. The period of incubation is difficult to determine; it is stated to be from two to ten days, commonly seven. It may be one day in the young. It is extremely variable in some instances, as when carriers infect themselves.

Occurrence.—Epidemics of cerebrospinal fever are usually localized, and rarely widespread. They almost always occur under crowded conditions, as in camps and on shipboard. Mining districts and seaports have suffered most severely. On the other hand, rural districts have also been afflicted, and widespread outbreaks have occurred in the tropics.

Cerebrospinal fever shows a distinct *seasonal prevalence* for the colder months of the fall and winter. In this respect it resembles other infections spread by the secretions of the mouth and nose, as pneumonia, scarlet fever, measles, diphtheria, etc. This is in sharp contrast to the seasonal prevalence of infantile paralysis which is much more prevalent in the summer time.

Children and young adults are most susceptible. Cerebrospinal fever is called a disease of children and soldiers. The soldiers who suffer most are those living in barracks, garrisons, towns and camps, rather than those on the march or in the field. Crowding, which favors contact infection, and fatigue, which predisposes to this disease, explain the special liability under these conditions. Seasoned troops rarely suffer from the disease.

Predisposing factors play an important rôle in depressing immunity and facilitating infection with the meningococcus. Overexertion, depressing mental and bodily surroundings, catarrhal inflammations, the misery and squalor of tenement life, and the sad conditions of some mining and laboring camps have long been recognized as predisposing factors. It is not the hardened soldier but the recruit in the making who is susceptible. The emergencies of the World War again showed the hazard of haste in training the soldier and the sailor.

Weed has shown by experiments on animals that diminution of the pressure of the cerebrospinal fluid facilitates infection of the meninges with meningococci or any other microorganism that may be in the blood stream. Experimentally the pressure of the cerebrospinal fluid may be decreased by puncture, by pressure on the vessels of the neck, and other ways. Decreased pressure due to natural causes may prove an important predisposing factor.

Cerebrospinal fever has long been recognized as a war disease. No great epidemics of the disease occur among troops, but the affection prevails more or less in all armies. Sharp localized outbreaks occurred here and there. Between 1837 and 1850 a very widespread epidemic occurred in France, the disease being carried from place to place by the movements of the 18th Regiment of Infantry, in which the infection had established itself.

Cerebrospinal fever is not highly communicable, but spreads slowly and irregularly. It is usually difficult to trace connection between one case and the next—its movement shows curious pranks. This is now explained through carriers. Except in sharply localized outbreaks, it is rare to have more than one or two cases in a home, even where precautions are not taken. Sporadic cases are apt to crop out almost anywhere, at any time of the year. Only a small proportion of those who receive the infection develop the disease; the meningococcus is passed on from one to another until a susceptible individual is reached who develops meningitis.

The case fatality rate is usually high, about 75 per cent, occasionally being as low as 20 per cent in some epidemics. The use of serum has reduced the death rate to about 25 per cent.

The immunity produced by one attack is not lasting. Councilman reports five instances in which the same individual is reported to have had the disease twice.

Modes of Transmission.—It is probable that the meningococcus enters the system through the mucous membrane of the nasopharynx. From this position it may reach the meninges directly through the lymph channels, but usually indirectly through the circulating blood. The experiments of Flex-

ner¹²² in the monkey indicate that when the meningococcus is introduced into the cerebral cavity it escapes by a reversed lymphatic current, so that under these circumstances it may be found in the mucous membrane of the nasopharynx. It therefore seems clear that cerebrospinal fever is spread through the discharges from the nose and mouth. It is a contact infection, due in the great majority of cases to rather direct association with cases or carriers. Indirect transfer is quite possible through fingers, dishes, pipes, handkerchiefs, toys, and other objects contaminated with fresh discharges. Droplet infection also plays its rôle. Crowding and close personal contact favor the spread of the infection. The meningococcus is so frail that our environment does not remain infective very long.

While carriers are chiefly responsible for the spread of cerebrospinal fever, it has recently been recognized that there are mild and abortive cases of the disease. These "missed" cases spread the virulent infection quite as actively as carriers. Cases of pharyngitis associated with and perhaps due to the meningococcus have been observed to occur frequently in endemic centers and in epidemic times. The theory of the disease is that it is ordinarily mild and prevalent, and that only occasionally does it cause meningitis.

The meningococcus may pass through the cribriform plate of the ethmoid bone, but probably reaches the meninges in almost all instances by the blood stream. The disease is primarily a bacteremia.

The Meningococcus.—The meningococcus is a strict parasite and is unknown in nature outside of the human host. It is a frail, Gram-negative microörganism, resembling the gonococcus. Both are biscuit-shaped cocci; both grow feebly on artificial media. They are readily killed by drying, heat, and other unfavorable conditions. They live a strict parasitic existence and cause diseases peculiar to man, with lesions which resemble each other, both as far as the character of the inflammation and the distribution of the cocci within the cells are concerned. These two microörganisms are usually distinguished by the source from which they are obtained. Otherwise the differentiation depends upon their relation to the fermentation of sugars, agglutination, and complement fixation. The medium for the growth of the meningococcus must contain hemoglobin. Blood agar or liver agar is suitable. Involution, or "ghost" forms, appear in young cultures and after twenty-four hours autolysis is very active.

All bacteriologists now recognize the fact that there are very many different strains of meningococci, distinguished mainly by their reaction towards specific agglutinins. As many as fifty-two different strains were used to inject a horse for the purpose of preparing a polyvalent serum. Gordon recognized four groups; French observers¹²³ speak of meningococci and parameningococci, the latter being commonly associated with the disease in the United States. A classification around definite types, as in the case of pneumococci, has been attempted, but is not yet definite. The Hygienic Laboratory

¹²² *J. Am. M. Ass.*, 1917, 69: 639.

¹²³ *Dopter. Compt. rend. Soc. de biol.*, 1909, 67: 74.

of the United States Public Health Service recognizes twelve strains, which comprise four agglutinating and five tropin groups, including all known types.

The meningococcus has been found in the blood almost constantly in the early stages of the disease. The modern notion of cerebrospinal fever is that it is primarily a septicemia with a predilection to localize in the meninges. Other structures are often attacked, especially the joints and the heart valves. In fulminating cases, the septicemia may overshadow the meningitis. Death may occur in twenty-four hours, or less.

Resistance.—The effect of sunlight on the meningococcus is rather surprising, the organism being very resistant to it. Elser and Huntoon¹²⁴ found that some strains survived eight or nine hours' exposure; Foster and Gaskell,¹²⁵ seven hours' exposure. All strains tested survived one or two hours' exposure to full sunlight. Drying, on the other hand, has a marked effect in killing meningococci. No growth could be obtained from cover slips with meningococcus cultures after five or ten minutes' drying in a sulphuric acid desiccator. When dried on glass in ordinary air, twenty-four hours' exposure has been found by various observers to destroy the organism. Elser and Huntoon found that under certain circumstances the meningococcus can survive somewhat longer. It is very sensitive to germicides and heat.

Carriers.—Healthy passive carriers outnumber the cases of cerebrospinal fever 10 or 30 to 1. Persons directly associated with the patient rarely take the disease, but often become carriers and thus transmit the infection to others, and these to others and so on indefinitely. Only a small percentage of carriers is susceptible and develops the disease. Cerebrospinal fever is disseminated almost wholly by carriers.

The meningococcus is found quite constantly in the nasopharynx during the first five days of the disease. It then begins to disappear and is gone after four weeks or a few months of convalescence. Only rarely are chronic active carriers found. On the other hand, a large proportion of the healthy persons who come in immediate contact with a case or a carrier become carriers. The healthy passive carrier therefore is the chief factor in disseminating the infection.

The number of carriers varies with circumstances. In Boston in 1917, we found from 1 per cent among persons who had no known contact with the disease, up to 80 per cent among intimate contacts under military conditions. On account of the carrier state in this infection, Gordon considers all persons who have come within three yards of a case under conditions favorable for infection as "contacts."

A mother who nurses a meningococcic infant is sure to become a carrier, and vice versa. Of sixteen roommates of a soldier stricken with meningitis, ten were carriers. The meningococcus has been found in as many as 100 per cent of those in the immediate entourage of a case. It was a common experi-

¹²⁴ *J. Med. Research*, 1909, 20: 494.

¹²⁵ *Cerebrospinal Fever*, Cambridge University Press, 1916.

ence during the World War to find 20, 30 and 40 per cent of a command, in the army or navy, to be carriers. The percentage increases with the degree of crowding and the intimacy of contact. Where people eat, sleep, work, play, and wash together as in camps and on shipboard, there is every facility for interchange of the flora of the mouth and nose.

The number of carriers varies with factors other than crowding. There are more in the cold months than in summer, more when an epidemic is fully developed than during the waxing and waning stages, and more in severe epidemics than in mild.

Most carriers are temporary; at least, the meningococci cannot be found after a month or two. Hundreds of carriers observed in Boston during the World War cleared up promptly with the coming of warm weather. This is a common experience.

Almost pure cultures of meningococci are obtained from the nasopharynx of many carriers; in others only an occasional colony. All intermediate grades occur. Only a very small percentage of the carriers develops the disease. This may be due to an immunity or to lack of facilitating factors which determine penetration of the meninges. Most carriers are adults, whereas the disease has a special predilection for children. Differences in the anatomical structure of the lymphatic system may account for the resistance of adults. The quantity of carriers does not seem to influence the incidence of the disease. During the World War the number of carriers discovered was so great that it became impractical from a military standpoint to isolate them. In some places separate training camps for meningitis carriers were established.

There is no evidence that chronic carriers develop the disease. I cannot escape the conviction that many of the carriers of meningococci are not dangerous, either to themselves or their fellow men. This may be on account of the lack of virulence of some strains of the organism. Perhaps we have a similar situation with meningococcus carriers that we have in diphtheria carriers, but we have no method at present of distinguishing true or virulent carriers of cerebrospinal fever from the harmless varieties.¹²⁶

Many measures have been tried to cure carriers. Local applications of germicidal substances such as chloramin in 0.5 to 1.0 solutions (Gordon), peroxid of hydrogen, various cresol preparations, permanganate of potash, and zinc sulphate have been tried. The difficulty is in reaching the meningococci. Warm, moist rooms, with and without germicides added to the air, have been tried. Vaccines do not relieve carriers. Those with tonsillitis and catarrhal inflammation of the nasopharynx lose the meningococcus more slowly than those with normal mucous membranes. No measure so far tried has proven effective.

Recognition of Meningococcus Carriers.—The specimen should be taken from the roof of the nasopharynx, with a sterile cotton swab, guarded by a

¹²⁶ Ponder, *Brit. M. J.*, Sept. 18, 1920, 427.

glass tube, as shown in the illustration (Fig. 16). It is important to prevent contaminations by organisms in the mouth and saliva.

Proper technic in obtaining the specimen and in seeding the plates can only be gained by practice.



FIG. 15.—WEST SWAB TUBE.

The meningococcus is very frail, and therefore the plates should be made immediately. It is very sensitive to acid and is soon destroyed by the activity of contaminating organisms. Sodium chlorid is also toxic to the meningococcus, and therefore salt solution should not be used as a diluent, and salt is commonly omitted from the culture media.

The meningococcus may be grown upon sheep-serum dextrose agar, ascitic agar, blood-serum agar, pea-extract trypsin agar, or liver agar.

The sheep-serum agar is made with one part of sheep serum and three parts of double distilled water. The sheep serum is obtained by clotting and centrifuging. This diluted serum is then sterilized fractionally in the Arnold for thirty minutes on three successive days. The agar is prepared separately with 2 per cent agar and 1 per cent peptone, reaction 0.2 to phenolphthalein. The plates are made by adding 1 c.c. of the diluted sheep serum to 5 c.c. of the liquefied agar at 40° C.

The Petri plate is seeded with the swab by simply touching the tip to one place on the plate. From this spot the material is spread by means of a loop or flat wire. The plate is then incubated over night, at 37° C. Watch for small moist colonies with regular outline; then let the plates stand at room temperature (about 25° C.) for several hours. This permits the colonies of *Micrococcus catarrhalis* to grow and affords a ready method of differentiation, for the meningococcus does not multiply at room temperature.

Young meningococcus colonies are colorless, translucent, have a regular contour (lens effect), uniform granular structure, mix easily in salt solution, and make a homogeneous suspension. Suspicious colonies may be tested directly by the drop method on a

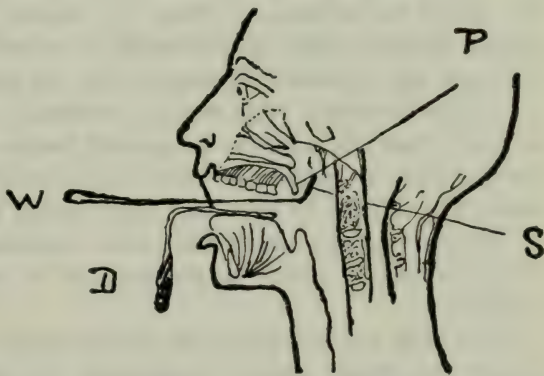


FIG. 16.—DIAGRAM ILLUSTRATING THE METHOD OF TAKING MATERIAL FROM THE NASOPHARYNX BY MEANS OF A SPECIAL SWAB.

W—Wire holder of S—swab; P—soft palate; D—tongue depressor. (After Dopter.)

microscopic slide, fished to sheep-serum dextrose agar slants or other suitable media, incubated over night, and tested for macroscopic agglutination. The cultures for the agglutination test should be less than twenty-four hours old, for by that time involution forms appear, and the older colonies make less satisfactory suspensions.

The agglutination is best done macroscopically at 56° C. and the tubes are allowed to stand at this temperature for twelve to twenty hours before taking the final reading.

Cultures that are agglutinated with a dilution of 1:100 with a polyvalent serum, but that fail to agglutinate in normal control serum in half this titer, may be regarded as presumptively positive. After the presumptive test, careful study of the culture must be made in order to identify it.

The meningococcus must be carefully distinguished from *Micrococcus flavus*, *Micrococcus crassus*, *Micrococcus pharyngis siccus*, *Micrococcus catarrhalis*, and other confusing organisms. For details see Olitsky's method¹²⁷ and Krumwiede's macroscopic slide agglutination test.¹²⁸

Prevention.—A better knowledge of the facts concerning cerebrospinal fever has shaken our confidence in the practical value of preventive measures. Theoretically the case is plain; practically, very difficult. The wide prevalence of the infection, the large number of carriers, the existence of mild and abortive cases, all add to the complexity of the administrative control of the disease. It is not clear that any of the measures so far taken have either materially influenced the course of epidemics or prevented the spread of the disease.

In military practice, the isolation of meningitis carriers is not only extravagant and against military efficiency, but also impractical and non-effective. The search for carriers and their care should not cause neglect of the more general measures, such as prevention of crowding, control of hand-to-mouth infection, the allowance of ample time for the "hardening and seasoning" of recruits, protection from weather, overwork, and fatigue, and the use of all measures that will promote the general health of the command. Sprays and douches have no protective value, and injudiciously used may be mischievous. They should be used only under skilled medical advice.

We must frankly admit that when cerebrospinal meningitis has once become epidemic it cannot be stamped out by any known means of practical application.

This does not mean that we should assume a supine attitude, for, even though the disease cannot be satisfactorily controlled, a certain number of secondary cases may be prevented. Vaccines have been tried, but their usefulness has not been established. The procedure is logical and deserves extended observation. Agglutinins develop in the blood of those vaccinated.

Antimeningitis serum is useful in the treatment of the disease; it is not practical as a preventive. It must be introduced into the subdural space

¹²⁷ *J. Am. M. Ass.*, 1918, 70: 153.

¹²⁸ *J. Am. M. Ass.*, 1917, 69: 359.

by lumbar puncture. Intravenous injections to counteract blood infection are also indicated. The serum should always be matched with the particular strain of meningococcus to obtain specific results. The serum should be provided free of cost or at a minimum price by health authorities. Further, boards of health should provide laboratory facilities for the bacteriological diagnosis of the disease, and the recognition of carriers.

CHAPTER IV

INSECT-BORNE DISEASES

GENERAL PRINCIPLES

The fact that disease may be transmitted through the bites of insects was suspected for years, but it was not until 1893 that it was demonstrated as a new principle by Theobald Smith in the case of Texas fever of cattle and the tick.¹ Since then many diseases have been added to the list, which is constantly growing. We now know that some diseases are always transmitted through insects and others occasionally. A thorough comprehension of the subject is necessary for sanitarians and others in the fight against disease in all climates and in all countries.

Ticks and mites belong to the lower class of the Arachnida and are not, strictly speaking, insects (Insecta), but are considered in the same group for practical convenience.

All the blood-sucking parasites must be regarded as dangerous. If they do not play the rôle of an intermediate host in the biological sense, they may occasionally transfer infections in a mechanical way, or the little wounds may allow the entrance of such infections as erysipelas, the pus cocci, anthrax, tetanus, and other microorganisms. Further, all blood-sucking parasites are potentially dangerous, in that new diseases may be established as the old ones must have been established at one time through the triple alliance of host, insect, and parasite.

Intermediate and Definitive Hosts.—The *intermediate* host in the zoological sense is that animal which harbors the asexual phase of the life cycle of the parasite; the *definitive* host is the animal which harbors the sexual phase. Thus, in malaria man is the intermediate host, the mosquito the definitive host. In popular parlance, the insects are spoken of as the intermediate hosts in all cases.

The Insects Not Harmed.—It may be stated as a general rule that the insect hosts are not harmed by the parasites which they harbor and which are pathogenic for man. Thus, the malarial protozoön is apparently harmless for the mosquito. The same is true of yellow fever and *Aedes aegypti*, Texas fever and the tick, sleeping-sickness and the tsetse fly, typhoid and the house fly; and to a certain extent of typhus fever and the louse, and plague and the flea. Rickettsia bodies may shorten the life of lice, and plague bacilli may prevent the feeding of fleas.

¹ The other names associated with the early work upon insects and their relation to disease are: Manson, Finlay, Ross, Grassi, and the United States Army Commission—Reed, Carroll, Lazear, and Agramonte.

Mechanical and Biological Transmission.—It may be stated as a general law that, if a period of incubation in the insect is necessary, it indicates that the parasite probably belongs to the animal kingdom and passes part of its life cycle within the insect. This constitutes the so-called *extrinsic period of incubation*. Malaria and yellow fever are examples of this class, which is spoken of as *biological* transmission. If, on the other hand, insects convey infection at once without a period of incubation in the insect, the transfer is a *mechanical* one; in this case the insect does not play the part of an intermediate host in the true biological sense, and there is no cycle of development of the parasite within the insect. Almost all of these cases are bacterial infections.

Methods of Mechanical Transmission.—Insects transfer infections mechanically in a variety of ways. The mouth parts, legs, or outer surfaces of the body may be smeared with the virus, which is then simply carried to the lips, fingers or food, and thus enters the susceptible individual; or the virus may remain attached to the proboscis of a biting insect, thus transferring the infection very much as a hypodermic needle would; or the virus may be passed in the dejecta of the insect and be scratched or rubbed into the wound made by the bite; or the virus may be contained in the digestive tube and be regurgitated when the insect bites; or the virus may be in the body cavity and be released when the insect is crushed.

Specificity.—As a rule, only one species, or at most a single genus, acts the part of vector for any particular infection, excepting in the mechanical transference of infection by insects. Malaria is confined to *Anopheles* and related genera, yellow fever to *Aedes*, Texas fever to *Margaropus annulatus*, sleeping-sickness to *Glossina*, etc. This is a question of specificity. The specific nature of some of these diseases may be due to the fact that the parasite is not pathogenic for other hosts. Thus, most of the insect-borne diseases do not occur naturally in the lower animals and cannot as a rule be transferred even though large amounts of the infected blood be inoculated. The disease may be specific, in the sense that it is confined to one species, because the insect conveying the infection refuses to bite other than its own host. Strict specificity is found in all the cases of biological transference, whereas mechanical transference of infection may take place through widely separated genera.

Protozoa, bacteria, and even parasitic worms may be transferred by insects. The character of the disease cannot be predicated from the nature of the insect host. Thus, ticks convey piroplasma and also spirochetes; flies convey trypanosomes, bacteria, the eggs of worms, and a variety of other infections; mosquitoes are concerned in the transmission of the plasmodium (a protozoön), filaria (a round worm), spirochetes and filtrable viruses.

Hereditary Transmission.—In some instances the virus is transmitted "hereditarily" through the insect from one molt to another, and even from one generation to the next. So far as known, however, hereditary transmission takes place only in those "insects" having an incomplete metamorphosis,

such as the ticks. Brues suggests that the hereditary transmission of a virus is practically impossible in insects passing through complete metamorphosis, owing to the active phagocytosis during the pupal stage.

Prevalence.—Insect-borne infections are types of true endemic diseases, and they are necessarily limited in geographical distribution to the habitat of the insect host. They prevail especially in tropical and warm, moist regions, where insect life abounds. The seasonal prevalence of insect-borne disease is therefore distinctive. The only exception is typhus fever, which is a cold weather disease because human lice find winter conditions most favorable.

Insect-borne diseases may occur in great epidemics, as yellow fever, malaria, dengue, typhus, relapsing fever, etc. When this happens it means that the particular insect involved prevails in enormous numbers in the epidemic area, with numerous susceptible victims.

The geographical distribution of the disease is always more limited than the geographic distribution of the insect host: *Anopheles* exist in many places where there is little or no malaria. *Aedes* mosquitoes are numerous in the Philippines and Hawaii, but yellow fever has not yet been carried there.

Many of the insect-borne diseases were formerly known as "place diseases." Thus, in yellow fever it was realized that the infection was not conveyed directly from man to man, but it was believed that the house or place became infected, and it was thought that the virus lived in the soil, upon the bedding, or on the clothing. This led to the notion that *fomites* or inanimate objects played an important rôle in the transference of disease. The early studies in bacteriology gave countenance to this view until our knowledge of the part played by insects and the importance of "contacts" and "carriers" has placed fomites in a subordinate and oftentimes negligible position in public health work.

Migration.—In the migration of insect-borne diseases it is usually the human host and not the insect that acts as the traveler. Insects, as a rule, do not go great distances of their own volition, and never overseas or from one country to another, unless taken in the conveyances of man or upon some animal host. When yellow fever or malaria goes from one country to another, the infection is translated in man. The infected mosquitoes are rarely transported, except occasionally upon old-fashioned wooden sailing vessels with water barrels that afford breeding places.

An apparent exception to this statement is the case of plague. It is the rat rather than man that spreads plague from land to land. In this case, however, the disease is primarily an infection of the rat, which carries the flea along and man is secondarily attacked. Flies, mosquitoes, and other insects are known to travel or be blown a mile or more upon the wing; salt marsh mosquitoes many miles.

Temporary and Permanent Parasites.—All the parasitic animals which live upon man and other animals may possibly act as go-betweens in the transportation of the microorganisms of disease. Parasites which live upon the skin are known as *ectoparasites*, in contradistinction to *endoparasites*,

which live within the body. The ectoparasites may be temporary parasites, as the mosquito; or permanent, as the tick, which spends all but its earliest and last days, and sometimes short periods at the time of molting, attached to the skin of its host. Between these extremes there are parasites spending more or less of their life attached to the host; thus, the bedbug and flea are temporary, whereas lice are more permanent parasites.

Principles of Prevention.—Insect-borne diseases may be controlled by attacking the insect, or the parasite in the host, or both. The object should be to attack the weakest link in the chain.

The prevention of the insect-borne diseases depends upon a knowledge and thorough comprehension of three factors: (1) the disease, (2) the parasite, and (3) the insect. The suppression or control of the insect depends upon a precise knowledge of its biology. Entomology, therefore, has become a vitally important subject so far as preventive medicine is concerned. Without an acquaintance of the life history and habits of the insect host there will be economic



FIG. 17.—A SOUTH AFRICAN BLOOD-SUCKING FLY (*Pangonia*), ILLUSTRATING LONG PROBOSCIS TO PIERCE HEAVY FUR OF CERTAIN ANIMALS, (Brues.)

loss, wasted energy, and disappointing results. Malarial mosquitoes are active at night and breed in the swamps and also in almost any still water; the yellow fever mosquito is active by day and breeds about houses. Other mosquitoes have their own particular breeding and hiding places. The suppression of lice depends largely upon bodily cleanliness, the suppression of the bedbug upon house cleanliness, the dangerous fleas come largely from association with other animals, the flies from manure and decomposing organic matter, the ticks from other animals and from infested ground and vegetation.

The prevention of malaria is a very good illustration of the importance of knowing the life history and habits of the insect involved. Great practical difficulties have been found in the control and prevention of malaria and there has been much waste of time and money and sometimes keen disappointments. We now know that it is not necessary to wage extensive and expensive campaigns against all anopheles, for only a few species are practically concerned; furthermore, that the incriminated species differ in different localities, and that their life habits vary with local environment. We are not sufficiently informed of the facts in some localities to make an impress upon the disease.

For the control of the insect-borne diseases it is not always necessary to

exterminate the particular insect host. In fact, the extermination of a particular species, much more a genus, is practically a biologic impossibility. A material reduction in the numbers of the insects in a particular area will control and often result in an elimination of the disease.

An effective campaign against mosquitoes, flies, or other insect pests requires the expenditure of time and money. Further, it requires the assistance of the entomologist, the engineer, and the practical administrator. When the campaign involves extensive drainage or filling-in operations, this calls for the services of an engineer who has specialized along these lines. To attack the problem without a complete knowledge obtained from a careful study of the habits and breeding places of the particular species of insect will probably result in economic waste. The habits and habitat of some species may vary in different localities, and a careful study of the local conditions is important to insure success. In the organization of a mosquito campaign the several branches of the work may be allotted to special divisions, each consisting of a foreman and crew. These men become skilled in their particular duties, and efficiency is thereby greatly promoted. One division should have charge of the oiling, another of the fumigation, another should seek to destroy the natural breeding places, another should attend to the screening, etc. In fly suppression one division should look after the storing and handling of horse manure, another to garbage and organic refuse, and so on. All the work must be centralized under the direction of one person with executive ability and a thorough understanding of the problem.

The suppression of insects and household vermin is essentially a question of cleanliness. The most effective measures are those which strike at the breeding places, and these will be considered separately under mosquitoes, flies, ticks, lice, fleas and bedbugs. Next to the suppression of their breeding places, the most important measure in a household is to starve out these pests. Food must be so protected that insects, mice, and rats cannot gain access to it. Floors and other surfaces must be kept clean, so that they do not have the least film of organic dirt upon which insects feed. There should be no cracks or crevices to collect dust and dirt, which offer comfort for insect life and actual breeding places for some types like the fleas. Cleanliness and incessant care must not only be exercised in the household itself, particularly in kitchen, pantry, dining room, cellar, attic, and toilets, but must also include the back yard and surroundings of the house. Old cans and broken bottles, rubbish, garbage, and general untidiness around the household afford breeding places, hiding places, or food for vermin.

Science has demonstrated the danger from insects. Experience long ago decided that a healthy home must be free of insects and vermin of all kinds—it remains for the future to extend this kind of cleanliness to municipal housekeeping and rural sanitation.

The principal insect-borne diseases, their causes, and the insect responsible in each case are stated in the following table:

THE PRINCIPAL INSECT-BORNE DISEASES

DISEASE	CAUSE	INSECT
MOSQUITOES		
MALARIA (Laveran, 1880, the parasite; Ronald Ross, 1895-1898, relation to the mosquito)	<i>Plasmodium malariae</i> (Laveran) <i>Plasmodium vivax</i> (Grassi and Feletti) <i>Plasmodium falciparum</i> (Welch)	<i>Anopheles</i>
YELLOW FEVER (Reed, Carroll, Lazear, and Agramonte, 1900-1902)	A filtrable virus <i>Leptospira icteroides</i> (Noguchi, 1919)	<i>Aedes aegypti</i>
FILARIASIS (Cobbold, 1877)	<i>Filaria bancrofti</i>	<i>Culex quinquefasciatus</i> , etc.
DENGUE (Graham, 1903; Ashburn and Craig, 1907)	A filtrable virus	<i>Aedes aegypti</i>
FLIES		
SLEEPING-SICKNESS (Gambian) (Dutton, 1902)	<i>Trypanosoma gambiense</i>	<i>Glossina palpalis</i> —a tsetse fly
SLEEPING-SICKNESS (Rhodesian) (Stephens & Fantham, 1910)	<i>Trypanosoma rhodesiense</i>	<i>Glossina morsitans</i> —a tsetse fly
NAGANA (of cattle, etc.) (Bruce, 1895)	<i>Trypanosoma brucei</i>	<i>Glossina morsitans</i> —a tsetse fly
SURRA (of horses, etc.) (Evans, 1880; Steel, 1885)	<i>Trypanosoma evansi</i>	<i>Stomoxys</i> and other biting flies; also fleas
PAPPATACI FEVER (3 day fever) (Doerr, 1909)	A filtrable virus (?)	<i>Phlebotomus papatassii</i> —a dipterous biting gnat
CALABAR SWELLING (Loa loa) (Cobbold, 1864; Manson, 1891)	<i>Filaria diurna</i>	<i>Chrysops</i> —a biting fly
TULAREMIA (Francis, 1921)	<i>Bacterium tularensis</i> (McCoy and Chapin, 1912)	<i>Chrysops discalis</i> —a blood-sucking fly
TYPHOID, CHOLERA, DYSENTERY, etc. Contagious ophthalmia, "pink eye," erysipelas, anthrax, glanders, skin infections. Smallpox and other exanthemata.		Flies and other insects, by mechanical transmission
TICKS		
TEXAS FEVER (of cattle) (Smith & Kilborne, 1893)	<i>Babesia bigemina</i>	<i>Margaropus annulatus</i>
ROCKY MOUNTAIN SPOTTED FEVER (Ricketts, 1906; Wolbach, 1916)	<i>Dermacentor variator</i> rickettsi	<i>Dermacentor andersoni</i>
RELAPSING FEVER (W. Africa) (African tick fever) (Dutton and Todd, 1904)	<i>Spirochaudinnia duttoni</i> ; now <i>Borrelia duttoni</i>	<i>Ornithodoros moubata</i> ; and also lice

THE PRINCIPAL INSECT-BORNE DISEASES—(Continued)

DISEASE	CAUSE	INSECT
BEDBUGS AND RELATED INSECTS		
TULAREMIA (Francis, 1921)	<i>Bacterium tularense</i> (McCoy and Chapin, 1912)	<i>Dermacentor andersoni</i>
EUROPEAN RELAPSING FEVER (Obermeier, 1873)	<i>Borrelia recurrentis</i>	Bedbugs and lice
INDIAN KALA-AZAR (Ross, 1903)	<i>Leishmania donovani</i>	<i>Clinocoris rotundatus</i> <i>Conorhinus</i> (?)
SOUTH AMERICAN TRYPANOSOMIASIS (Chagas disease) (Chagas, 1909)	<i>Trypanosoma cruzi</i>	<i>Conorhinus megistus</i> — a large hemipterous biting insect
LICE		
TYPHUS FEVER (Nicolle, 1909; Ricketts & Wilder, 1910; Anderson & Goldberger, 1910)	<i>Rickettsia prowazeki</i>	<i>Pediculus humanus</i> , var. <i>corporis</i> ; also <i>capitis</i>
TRENCH FEVER	<i>Rickettsia pediculi</i>	<i>Pediculus humanus</i> , var. <i>corporis</i>
ASIATIC RELAPSING FEVER (Carter, 1877)	<i>Borrelia carteri</i>	<i>Pediculus humanus</i>
EUROPEAN RELAPSING FEVER (Obermeier, 1873)	<i>Borrelia recurrentis</i>	Lice and bedbugs (?)
ALGERIAN RELAPSING FEVER (Sergent and Foley, 1910)	<i>Borrelia berbera</i>	<i>Pediculus humanus</i> , var. <i>corporis</i> and <i>capitis</i>
FLEAS		
PLAGUE (Kitasato, 1894; Yersin, 1894)	<i>Bacillus pestis</i>	<i>Xenopsylla cheopis</i> (<i>pallida</i>) and others

The following table gives a list of the principal diseases which are transmitted by other "intermediate" hosts:

DISEASE AND ADULT PARASITE	INTERMEDIATE OR USUAL HOST	INFECTING STAGE OF PARASITE	MODE OF INFECTION
Liver fluke <i>Fasciola hepatica</i>	Snails <i>Limnæus</i>	<i>Cercariæ</i>	Ingestion
Liver fluke <i>Clonorchis endemicus</i>	Fish	Encysted stage	Ingestion
Lung fluke <i>Paragonimus westermanii</i>	Fresh-water crab <i>Potamon dehaanii</i> and snails	Encysted larvæ	Ingestion
Schistosomiasis <i>S. japonicum</i> <i>S. mansoni</i> <i>S. hematobium</i>	Fresh-water snails <i>Oncomelania nosophora</i> <i>Oncomelania hupensis</i> <i>Bullinus planorbis</i>	<i>Cercariæ</i>	Through the skin
Fish tapeworm <i>Dibothriocephalus latus</i>	Pike, salmon, etc.	<i>Plerocercoids</i>	Ingestion
Dog tapeworm <i>Oypilidium caninum</i>	Dog fleas and dog lice	<i>Cysticeroids</i>	Contact with dogs
Rat tapeworm <i>Hymenolepis diminuta</i>	Rat fleas	<i>Cysticercoid</i>	Ingestion
Dwarf tapeworm <i>Hymenolepis nana</i>	(?)	<i>Cysticercoid</i>	Ingestion
Pork tapeworm <i>Tænia solium</i>	Swine	<i>Cysticercus</i>	Ingestion
Beef tapeworm <i>Tænia saginata</i>	Cattle	<i>Cysticercus</i>	Ingestion
Hydatid disease <i>Tænia echinococcus</i>	Dogs	<i>Onchosphere</i>	Ingestion
Guinea worm <i>Dracunculus medinensis</i>	A small crustacean <i>Cyclops coronatus</i>	<i>Larvæ</i>	Ingestion
<i>Lambliæ intestinalis</i>	Rats, mice	Encysted stage	Ingestion
Trichinosis <i>Trichinella spiralis</i>	Swine	Encysted stage	Ingestion
Infectious Jaundice, Weil's Disease <i>Spirochæta icterohæmorrhagiæ</i>	Rats*		

* Other diseases transmitted by rats and other rodents, see page 334.

INSECTICIDES ²

Practically all germicidal agents are also poisonous to insects. There are some exceptions to this statement, notably formaldehyd, which is a potent germicide, but has little or no effect in its gaseous state upon insect life.

The action of insecticides may be considered under three classes: (1) those

²All insecticides sold in interstate traffic in the United States must comply with the Insecticide and Fungicide Act of 1910, administered by the United States Dept. of Agriculture.

that act as general protoplasmic poisons, such as strong acids or alkalis; (2) those that suffocate the insects, such as oily substance; (3) those that act upon the nervous structures, such as chloroform, ether, and other general anesthetics.

Another classification considers insecticides under four groups: (1) those used by contact in liquid form or in solution; (2) those used by contact in dry or powdered form; (3) those used by contact in vapor form; (4) those used by mixing with food and which are poisonous when ingested.

Insects differ markedly in their power of resisting insecticides. Those with well-developed chitinous protection, such as bedbugs and roaches, are more difficult to kill than flies and mosquitoes. Many insecticides have marked specific action. Thus, iodoform kills lice within ten to fifteen minutes, but has practically no harmful action on bedbugs, and very little effect on fleas. Hydrocyanic acid is more destructive to mosquitoes than to fleas,

and still more is required for bedbugs and lice. Pyrethrum has a more powerful action on bedbugs than on lice, etc.

The most practical of the insecticides for the destruction of the winged insects in an enclosed space are those that may be used in the gaseous state. Of these, sulphur dioxid and hydrocyanic acid gas are most commonly employed and are most reliable. The method of killing insects by gases and fumes is called fumigation. See pages 539 and 1370.

Preparation of the Room for Fumigation.

—It is more important to seal tightly a room in which insects are to be destroyed than when only a germicidal action of the gas is looked for. Insects may escape through

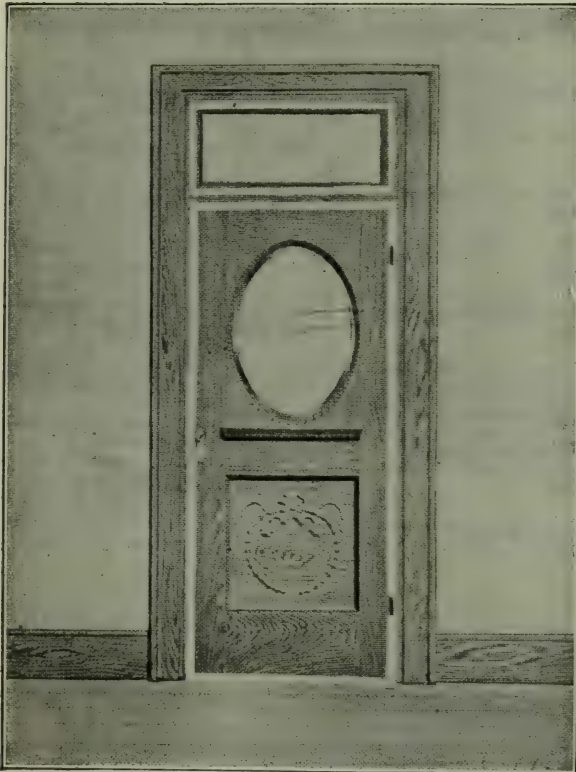


FIG. 18.—EXAMPLE OF SEALING DOORS FOR PURPOSE OF FUMIGATION.

minute openings, and they may hide in nooks and cracks where the gas permeates slowly and feebly, or may take cover under the folds of crumpled paper or

folded fabrics, and thus escape the insecticidal action of the gas. Self-preservation tempts mosquitoes and other insects, as well as rats and mice, to seek the light when in the presence of an irritating gas. It is, therefore, convenient to darken the place to be treated, leaving one source of light. The dead vermin may then be readily collected about this place.

Strips of paper should be pasted over doors and windows. Cracks and crevices may be calked with towels, waste, or other suitable substance. Ventilators, fireplaces, hot-air registers, and all openings into the room must be covered, otherwise both the gas and the insects will escape. Closets and small doors should be opened and all drawers, lockers, and similar places exposed in such a way that the gas may have fresh access to remote corners. Furniture should be moved away from the walls. Fabrics, paintings, instruments, bright metal work, or other objects liable to injury may be removed or covered, especially when sulphur is used (see also page 1375).

To insure success, a large volume of the gas should be liberated in a short time. If the gas is evolved slowly, much of it will be lost before the room can become charged with a sufficient amount to kill the insects. When large, leaky, or irregularly shaped spaces are to be fumigated, the amount of gas should be increased and the time of exposure prolonged. It is also advisable to generate the fumes in as many different places as practicable, as this favors rapid diffusion. The time of exposure varies with the gas, the insect (or rodent), and the conditions.

The best methods of generating gases for *fumigating* purposes are considered below. For further information concerning these substances, with special reference to their germicidal action, see Section XVI.

Sulphur.—Sulphur is a potent insecticide. It may be used either as a gas— SO_2 —or in its powdered form.

Sulphur dioxid is destructive to most forms of life. It will kill mosquitoes, flies, fleas, roaches, bedbugs, and all kinds of vermin, including rats and mice, but cannot be depended upon to kill the eggs of lice, fleas, mites, etc. While sulphur dioxid is a good insecticide, it is a rather feeble germicide. It diffuses poorly and has feeble penetrating power, requires expensive or cumbersome apparatus, and much labor.

Sulphur dioxid is a heavy, colorless, irrespirable gas with a peculiar suffocating odor and irritating properties. It has a density of 2.4. On account of the heavy specific gravity as compared with air it diffuses slowly and then settles toward the bottom of the compartment. It is also rather slow and has some risk of fire. It is further limited in practice on account of its destructive and corrosive action due to sulphurous acid and sulphuric acid produced in the presence of moisture. Fortunately the dry gas is quite as poisonous to mosquitoes, flies, rats, mice, etc., as the moist gas. Dry sulphur dioxid, however, has absolutely no germicidal value.

Sulphur dioxid possesses the advantage of being cheap and readily procurable. There is hardly a crossroad store in the country where a reasonable quantity of sulphur, either in the form of flowers or in rolls or sticks under

the name of brimstone, cannot be obtained. Sulphur dioxid is especially applicable to the holds of ships, freight cars, granaries, stables, outhouses, and similar rough structures—particularly if infested with vermin.

Sulphur dioxid tarnishes metals, rots fabrics, and bleaches pigments, especially when moist. Fumigation with SO_2 may, however, be done with little damage to property on dry days. Metal work, fabrics, and pigments that cannot be removed from the room may be protected from the sulphur fumes by coating with petroleum jelly or wrapping in paper.

Sulphur dioxid may be produced either by burning sulphur or by liberating liquefied sulphur dioxid. Two pounds of sulphur burned for each thousand cubic feet of air space and an exposure of one hour is sufficient to kill mosquitoes, flies, and other frail insects in a small tight space; three pounds and an exposure of six hours are ample for rats, mice, and fleas, and four pounds with an exposure of six hours for lice. If the space is large or leaky, the amount of gas should be increased and the time of exposure prolonged.

This gas is discussed in detail, with methods of production, under Maritime Quarantine, for it is chiefly used for the fumigation of ships (see page 540).

Flowers of Sulphur.—Sulphur in its dry, powdered state is useful against a number of parasites. In this form, however, it has little use as an insecticide in preventive medicine, not being efficacious against bedbugs, ants, roaches, or fleas.

It may be applied in several ways, the simplest of which is to sprinkle the dry sulphur about the places where insects are found. Flowers of sulphur may also be combined advantageously with other insecticides, such as kerosene emulsion, resin wash, or soap wash. It should first be mixed into a paste and then added to the spray tank in the proportion of about one or two pounds to fifty gallons. It is effective for the destruction of the mites and rust of plants and fruits.

Sulphur Ointment.—Sulphur in the form of an ointment is particularly obnoxious to ticks and other ectoparasites. The itch-mite (*Sarcoptes scabiei*) is very susceptible to the flowers of sulphur, which is, therefore, one of the ingredients of almost all ointments used in scabies.

Sulphur dips are used to destroy the mites on domestic animals. The formula now recommended for the treatment of scabies of cattle is as follows: Flowers of sulphur, twenty-four pounds; unslaked lime, twelve pounds; water, one hundred gallons. It is common experience that, while sulphur dips may be depended upon to destroy the mites, they do not destroy the eggs, hence the treatment should be repeated in about ten days, which permits time for the eggs to hatch and develop into adults. The lime and sulphur dips are widely used for both cattle and sheep affected with scabies. The advantages of this class of dips over arsenical dips are that they are effective, but not poisonous for cattle or man. Lime and sulphur dips do not kill the Texas fever tick, and arsenic should therefore be used.

Pyrethrum.—Pyrethrum is a popular and much used insecticide because it is comparatively cheap and is non-poisonous to man and the higher animals. It is also non-corrosive and does no harm to fabrics and pigments, and has therefore been used in those cases where sulphur is prohibitive on account of damage to paintings, fabrics, tapestries, metal work, musical instruments, and the like. Unfortunately, it has but feeble action against roaches, ants, bedbugs, flies, fleas and mosquitoes. It has no germicidal action.

Pyrethrum, also sold under the names of Buhach or Persian insect powder, or simply "insect powder," is the flowers of the *Chrysanthemum roseum* and the *Chrysanthemum carneum*, both hardy perennials and resembling camomile in appearance. According to Kalbrunner, four grains of the pure powder sprinkled on a fly in a vial should stupefy it in one minute, and kill it in two or three minutes. It acts on insects externally through their breathing pores. It may be used as a dry powder, either pure or mixed with flour, in which form it is puffed about the room, especially into cracks; or by its burning fumes. Pyrethrum powder is used in the proportion of two pounds per thousand cubic feet of air space, the exposure being for not less than four hours. It should be distributed in pots or pans and set on fire with a little alcohol, which should first be sprinkled over it. The quantity apportioned to any one pot or pan should not exceed one and one-half inches in depth, if the exposure is to be for four hours. The pots and pans should be set on bricks to prevent scorching the floor.

When pyrethrum powder is ignited it smolders, giving off fumes which stun, but do not always kill, mosquitoes.³ Because of this uncertain effect, therefore, these insects should always be carefully swept up and burned after fumigation.

Much of the pyrethrum upon the market is impure, which further weakens what is a feeble insecticide at best.⁴

Phenol Camphor (*Mim's Culicide*).—Camphophenique or phenol camphor is prepared by rubbing up equal weights of phenol crystals and camphor. It may be more conveniently prepared by first liquefying the phenol by gentle heat and then pouring it over the camphor. The camphor and phenol combine to form a new chemical compound, which remains fluid at ordinary temperatures. When phenol camphor is moderately heated it gives off dense fumes, which rise rapidly and diffuse slowly, and after thirty to sixty minutes, depending upon the amount employed and the temperature of the air, the fumes condense and are deposited as a moist film on all exposed surfaces. As a culicide phenol camphor is about equal to pyrethrum; the fumes stun the mosquitoes, but do not always kill them. The fumes are somewhat irritating to the mucous membranes, especially the eyes; they may cause dizziness, headache, cloudy urine, and other mild symptoms of phenol poisoning in susceptible individuals much exposed to their inhalation. The fumes of

³ Tobacco smoke and other substances which produce dense fumes, particularly those containing pyroligneous products, will kill mosquitoes.

⁴ *Insect Powder*, Bull. 824, U. S. Dept. of Agriculture, 1920.

phenol camphor do not tarnish metals, rot fabrics, or bleach pigments. They have, however, the disagreeable property of softening the varnish of surfaces on which they condense. On account of its slight power of diffusion, relatively high cost, and uncertainty of action, phenol camphor cannot take the place of sulphur except in the parlor, pilot house, and other compartments where sulphur is prohibited on account of the damage it produces.

Phenol camphor is used in the proportion of four ounces to every thousand cubic feet of air space, and with an exposure of two hours. It is heated in agate basins over an alcohol flame, care being taken to use just enough heat to cause evaporation of the fumes; too much heat will set the liquid on fire.

Hydrocyanic Acid Gas.—Hydrocyanic acid gas is extremely poisonous to all forms of life. It kills roaches, bedbugs, mosquitoes, fleas, flies, rats, mice, and other vermin with great certainty and very quickly. HCN also kills insect eggs, which sulphur dioxid and other gases may fail to do. Hydrocyanic acid gas is not a germicide. It is not very poisonous to the higher forms of plant life and therefore is much used in greenhouses for the destruction of insect pests and for scale and other parasites of fruit trees. The gas has a distinct place in the fumigation of granaries, stables, ships, barns, outhouses, railroad cars, and other uninhabited structures infested with vermin. It is also extensively used in flouring mills against weevils, in sleeping cars against bedbugs, and in tobacco warehouses against insects in general. It should be used in the household only with the greatest precaution, as the least carelessness with it would probably mean the loss of human life. A marked advantage is that it does not harm metals, fabrics, or pigments, and may be used in the most expensive drawing-rooms.

Hydrocyanic acid gas is lighter than air and has an agreeable aromatic odor, quite familiar in the flavoring essence of bitter almonds. The methods of producing this gas and its details are given on page 540.

Creel and Faget⁵ found that four ounces of potassium cyanid per thousand cubic feet are sufficient to kill mosquitoes in fifteen minutes; five ounces per thousand cubic feet kill bedbugs and roaches in one hour, lice in two hours; two and one-half ounces per thousand cubic feet kill fleas in fifteen minutes.

Hydrocyanic acid gas is more effective than sulphur dioxid, is not destructive, is reasonably cheap, and is certain in its action, but its poisonous nature is such a serious drawback that it has a limited place as an insecticide in public health work. It is finding favor in maritime quarantine practice, where it is largely replacing SO₂ for the reasons that it is less destructive, has better powers of penetration and diffusion and is more certain.

Disulphid of Carbon.—Disulphid of carbon (CS₂) is a very efficient insecticide, but a dangerous one, on account of its inflammable and explosive nature. It quickly kills mosquitoes, roaches, flies, ants, and insects of all

⁵ *U. S. Pub. Health Rep.*, 1915, 30: 3537.

kinds, as well as rats, mice, and squirrels. When pure it is a mobile, colorless liquid with an agreeable ethereal odor, but often it has a more or less fetid odor from the presence of other volatile compounds. The liquid must be kept in well-stoppered bottles in a cool place, and away from the light and fire. It evaporates rapidly at ordinary temperatures, so that in using this substance in a confined space it is sufficient to pour it into open pans. Carbon disulphid is very inflammable—more so than ether—and burns with a pale blue flame yielding sulphur dioxid and carbon dioxid or monoxid. In its use every precaution must be taken to see that there is no fire, lighted cigar, etc., in or about the field of operation. On account of its poisonous nature, if used in a house or other inhabited structure, the rooms must be thoroughly aired after its use.

According to Hinds, shallow tin pans or plates make good evaporating dishes for carbon disulphid. The larger the evaporating area the better. About one square foot of evaporating surface is used to every twenty-five square feet of floor area, and one-half to one pound of the liquid carbon disulphid is used for each square foot of evaporating surface. These figures, of course, are only suggestive and approximate. The pans should be placed as high in the room as possible, since the vapor is so heavy that it settles rapidly. Care should be taken when placing the pans to see that they are nearly level so as to hold the liquid, though ordinarily no particular harm will be done if some of it is spilled. It should not be found necessary to lose time in adjusting such things after the operation has begun.

Carbon disulphid was extensively used in California in the plague campaign. A piece of waste the size of an orange is saturated with the liquid and the wet ball placed in the mouth of the squirrel hole. Wet clay is then stamped into the warren so that the gas which is generating may have no opportunity to escape. All of the holes of the burrows are treated in this way. In some instances the ball is placed deeply in the hole and then ignited. This is more or less uncertain, as an explosion occurs, and, while the gas is thus disseminated, its action only covers a limited period of time, and is, therefore, not as certain as simply allowing the carbon disulphid to evaporate. It kills not only the squirrels, but also the fleas on them.

Carbon tetrachlorid (CCl_4) may be used in place of carbon disulphid. It is neither inflammable nor explosive, but somewhat more poisonous than chloroform. The sale of carbon tetrachlorid is forbidden in Paris on account of deaths from its use as a shampoo. It is sold in this country as a cleaning agent.

Petroleum.—Petroleum, kerosene, or coal oil is a very valuable insecticide. Petroleum and its products, such as kerosene, gasoline, naphthalene and lubricating oils, are the most dependable insecticides we have, and are used more and more in one form or another.

As a remedy for mosquitoes petroleum oil is applied in the proportion of about one ounce to fifteen square feet of water surface. It should form a uniform film over the surface, and will then destroy the larvæ and pupæ of the

mosquito and the adult females coming to the water to lay their eggs. The oil must be renewed every week or two, depending upon the temperature and other conditions. A light grade of fuel oil is best for this purpose (see page 281).

Petroleum is also useful against roaches, bedbugs, fleas, lice and other insect vermin when used by direct application or by spraying, either in the form of the pure oil or as an emulsion. Petroleum is very effective against fleas. Frequent application to the floor or other places will keep away ants, and by direct application to the breeding, feeding, and traveling places, it is a useful remedy against household vermin in general. Coal oil is the cheapest and most effective remedy for lice, and is applied directly to the head or other parts affected.

Emulsion of crude petroleum for application to the skin of animals, to trees or other plants, or for general insecticidal purposes is made from the formula of T. M. Price:

Crude petroleum	2 gallons
Water	$\frac{1}{2}$ gallon
Hard soap	$\frac{1}{2}$ pound

Dissolve the soap in the water with the aid of heat. To this add the crude petroleum; mix with a spray pump or shake vigorously and dilute with the desired amount of water. The emulsion of crude petroleum made according to this modified formula remains fluid, and can be easily poured. It will stand indefinitely without any tendency toward separation of the oil and water, and can be diluted in any proportion with cold soft water.

Gasoline, naphtha, and benzin, also naphthalene are among the best pulicides, and as such are extensively used to kill lice in typhus fever campaigns (see page 364).

Arsenic.—The two arsenicals in most common use obtainable everywhere are arsenate of lead and Paris green. Scheele's green, or arsenite of copper, is less known and less easily obtainable, but in some respects is better than Paris green. The use of powdered white arsenic is not recommended on account of its corrosive action, as well as the fact that it is apt to be mistaken for harmless substances.

The arsenical poisons may be applied in one of three ways: (1) in suspension, as poisoned waters, mainly in the form of sprays; (2) as a dry powder blown or dusted about the infested areas; or (3) as poisoned bait.

It must be remembered that the arsenicals are very poisonous; they should be so labeled, and care taken to prevent accidents.

Paris green is a definite chemical compound of arsenic, copper, and acetic acid (acetoarsenite of copper), and should have a nearly uniform composition. It is rather a coarse powder, or, more properly speaking, crystal, and settles rapidly in water, which is its greatest fault so far as the making of suspensions of this substance is concerned.

Scheele's green is similar to Paris green in color and differs from it only

in lacking acetic acid; in other words, it is simply arsenite of copper (CuHAsO_3). It is a finer powder than Paris green, and, therefore, is more easily kept in suspension.

Arsenious oxid (As_2O_3), or white arsenic, commonly known as arsenic, is used in dips to destroy ticks (see page 351).

Arsenate of lead is prepared by combining, approximately, three parts of the arsenate of soda with seven parts of the acetate of lead (white sugar of lead) in water. These substances, when pulverized, unite readily and form a white precipitate, which is more easily kept suspended in water than any of the other arsenical poisons. Its use is advised where excessive strengths are not desirable, and upon delicate plants, where otherwise scalding is likely to result.

An average of one pound of either Paris green or Scheele's green, or London purple to 150 gallons of water is a good strength for general purposes in using the wet method. The powder should first be made up into a thin paste in a small quantity of water, and, if the suspension is to be used upon plants or vegetables, or about foliage, an equal amount of quicklime should be added to take up the free arsenic and thus remove or lessen the danger of scalding.

For the distribution of dry poison the arsenicals are diluted with ten parts of flour, lime, or dry gypsum.

The following mixtures are used in the form of sprays, to destroy insects and fungi upon plants.⁶ They are equally useful as insecticides wherever sprays or local applications are practicable. The arsenate of lead mixture has been much used in Massachusetts with success against the gipsy moth and other destructive insects upon trees and plants. Paris green is an efficient larvicide in solution or dusted on the surface of water.

ARSENATE OF LEAD

Arsenate of soda (65 per cent strength), 4 ounces
Acetate of lead, 11 ounces
Water, 100 gallons

Put the arsenate of soda in two quarts of water in a wooden pail, and the acetate of lead in four quarts of water in another wooden pail. When both are dissolved, mix with the rest of the water. Warm water in the pails will hasten the process. For the elm-leaf beetle use ten instead of one hundred gallons of water.

A number of ready-made arsenates of lead are now on the market, and even when very large amounts are needed, it will probably prove cheaper to buy the prepared material than to make it. With this ready-made material use three pounds to fifty gallons of water for codling moth, and five pounds to fifty gallons for the elm-leaf beetle and on potatoes.

⁶ From Bulletin No. 123, April, 1908, of the Massachusetts Agricultural Experiment Station by Stone and Fernald.

ARSENATE OF LIME

White arsenic, 2 pounds

Sal-soda, 8 pounds

Water, 2 gallons

Boil till the arsenic all dissolves—about forty-five minutes. Make up the water lost by boiling and place in an earthen dish. For use take one pint of this stock, two pounds freshly slaked lime, and forty-five gallons water, and spray.

KEROSENE EMULSION

Hard soap, shaved fine, $1\frac{1}{2}$ pound

Water, 1 gallon

Kerosene, 2 gallons

Dissolve the soap in boiling water; remove from the fire and pour it into the kerosene while hot. Churn this with a spray pump till it changes to a creamy, then to a soft, butter-like mass. Keep this as a stock, using one part in nine of water for soft-bodied insects, such as plant lice, or stronger in certain cases.

RESIN-LIME MIXTURE

Pulverized resin, 5 pounds

Concentrated lye, 1 pound

Fish or other animal oil, 1 pint

Water, 5 gallons

Place the oil, resin and one gallon of hot water in an iron kettle and heat till the resin softens; then add the lye and stir thoroughly; now add four gallons of hot water and boil till a little will mix with cold water and give a clear, amber-colored liquid; add water to make up five gallons. Keep this as a stock solution. For use take:

Stock solution, 1 gallon

Water, 16 gallons

Milk of lime, 3 gallons

Paris green, $\frac{1}{4}$ pound

BORDEAUX MIXTURE

Copper sulphate (blue vitriol), 4 pounds

Lime (unslaked), 4 pounds

Water, 25 to 50 gallons

Dissolve the copper in hot or cold water, using a wooden or earthen vessel. Slake the lime in a tub, adding the water cautiously and only in sufficient amount to insure thorough slaking. After thoroughly slaking, more water

can be added and stirred in until it has the consistency of thick cream. When both are cold, dilute each to the required strength and pour both together in a separate receptacle and thoroughly mix. Before using, strain through a fine mesh sieve or a gunny cloth; the mixture is then ready for use.

If the amount of lime in the Bordeaux mixture is insufficient there is danger of burning tender foliage. In order to obviate this, the mixture can be tested with a knife blade or with ferrocyanid of potassium (1 oz. to 5 or 6 oz. of water). If the amount of the lime is insufficient, copper will be deposited on the knife blade, while a deep brownish-red color will be imparted to the mixture when ferrocyanid of potassium is added. Lime should be added until neither reaction occurs. A slight excess of lime, however, is desirable, and it is seldom one has to apply these tests. The Bordeaux mixture is a good fungicide, but is less useful as an insecticide.

Danger from Spraying.—The abundant and increasing use of the salts of lead, arsenic and copper to spray fruits, berries, and vegetables of all sorts has opened a new question, as these substances are poisonous for man. Ordinarily, an apple will carry about 0.5 mgm. of arsenate of lead. However, as much as 5 mgms. has been found on the surface of an apple directly after spraying. A quart of strawberries may carry as much as thirty-two mgm. of oxid of arsenic. A head of cabbage may carry a relatively large amount, especially on the outer leaves. The importance of thoroughly washing all fruits, vegetables, and berries is again emphasized.

MOSQUITOES

Mosquitoes differ markedly in their habits. Some may be classed as domestic species because they are commonly or almost exclusively found in or close to human habitations. This is notably the case with *Aedes aegypti*, the yellow fever and dengue mosquito; *Culex quinquefasciatus*, the intermediary for *Filaria bancrofti* (filariasis). The sylvan or wild mosquitoes, of which the *Aedes sollicitans*, the common salt marsh mosquito of our Atlantic coast, is a well-known example, are seldom met with in human habitations. A third or semidomestic class may be encountered either in or near houses, or in fields or swamps. Some species of anopheles are wild (*A. walkeri*), some are semidomestic, and one, *A. quadrimaculatus*, is generally considered a domestic species. "Domesticity" here refers to their feeding habits rather than their breeding places.

The flight range of mosquitoes varies with the species. *Aedes aegypti* flies but a few hundred yards from its breeding place; *Anopheles quadrimaculatus* flies one to one and one-half miles; while the salt marsh mosquito, *Aedes sollicitans*, may migrate inland forty miles aided by on-shore winds. Wind, however, does not have much to do with the distance and direction of mosquito flight. The radius of freedom from mosquitoes following destruction of their breeding places varies with the species.

Life History and Habits.—Mosquitoes pass through four stages: (1) the

egg or embryo, (2) the larva, (3) the pupa, and (4) the imago or adult winged insect. The egg, larval, and pupal stages are aquatic. Mosquitoes never breed in damp grass, weeds, or bushes, as is popularly supposed, but the winged insects frequently rest and hide in vegetation. The different species of mosquitoes differ markedly in their habits, and in the character of their breeding places. Each species has *selective* or *preferential* breeding places and *compulsory* breeding places; that is, if, when the urge comes to the female to lay her eggs, she cannot find the place of her choice, she is compelled to oviposit in some place not common to the species. *Aedes aegypti* is a container breeder, preferring to lay her eggs on the water in buckets, tubs, barrels, cisterns, cans, bottles, and the like; *Anopheles quadrimaculatus* is a pond breeder; while *Aedes sollicitans* prefers the salt marshes. From the standpoint of control, it is important to know that, if the breeding places of election are destroyed, the mosquito may seek others by compulsion.

Male mosquitoes are vegetarians. The females of many species have developed a taste for blood, and, indeed, blood has become indispensable to nearly all for the full development of their eggs. This is the case with *Aedes aegypti*. Remembering how all-important the generative instinct is, we can now well understand why the yellow fever mosquito, for example, will, when disturbed, return again and again in an endeavor to obtain her fill of this life-perpetuating fluid.

The mosquito usually lays her eggs upon the surface of the water, and these, depending upon the species, either float separately on their sides (*Anopheles*), or adhere together in irregular, raft-like masses (*Culex*). In a day or two, under ordinary conditions, the eggs hatch out into larvæ or "wiggle-tails." Although the larva is an aquatic animal, it is a true air-breather.

The larvæ move about more or less, actively searching for food, but at intervals of a minute or two they may be seen to come to the surface for air, where they hang, head down, attached by their more or less prominent conical breathing tubes to the surface film. The larva of *Anopheles* ordinarily rests and feeds at the surface, where it lies in an almost horizontal position, its tail and dorsal bristles touching the surface film, while it breathes through a breathing siphon, which is very short and insignificant in appearance. The mosquito remains in the larval stage about a week—the length of time varying with the species, and for any species with the temperature, amount of food and other factors; and is then transformed into a curiously shaped creature known as the pupa.

The pupa has no mouth and does not feed. It remains quietly at the surface except when disturbed. It breathes through a pair of projecting trumpet-shaped tubes. The pupal stage usually lasts two or three days or more, and is terminated by the emergence of the adult winged insect (imago) from its pupal case through a rent in the region of the breathing tubes.

The time from the laying of the egg to the winged insect may, therefore, be as short as nine days. The time depends upon the temperature and the

abundance of the food supply. Warmth favors and cold retards; therefore, mosquitoes are most abundant during the summer, late spring, and early fall months in our climate. In the tropics the wild species become more abundant during the wet season.

The way in which mosquitoes manage to pass through the rigors of the winter probably varies with the different species. Some, like the malarial anopheles, hide in sheltered cellars or dark nooks, or hibernate in other out-of-the-way places. Other species survive through the power of the larva and particularly of the egg to resist cold, for the larvæ of some species will develop even after they have been frozen, and some regularly outlive the winter as eggs.

THE DESTRUCTION OF MOSQUITOES¹

The measures aimed at the destruction of the mosquito naturally fall into two classes: (a) those directed against the egg, larva and pupa—the aquatic stages—and (b) those directed against the winged insect.

The most effective measures are those which destroy breeding places of the mosquitoes and thus prevent their multiplication. For the best results, both individual and communal effort are necessary, but the importance of individual effort alone cannot be too much emphasized. The individual, by attacking the problem on his own premises, grounds, or estate, can do much not only to rid his own immediate neighborhood of mosquitoes, and thereby increase his own comfort and guard against disease, but the example thus set will perhaps stimulate his less enterprising neighbors. To a large extent, the control of the mosquito-borne diseases are essentially local problems.

We must be satisfied with suppression, for extermination is not possible and may even be biologically undesirable. Largely reducing the numbers of mosquitoes will control and even eliminate the diseases which they carry. It is not necessary to render the anopheles extinct in order to control malaria; yellow fever may be vanquished with the local suppression of *aedes*.

Natural Breeding Places.—Natural collections of water which may serve as breeding places are best dealt with by filling in or by draining. In this way they are disposed of once for all.

Filling and Draining.—For filling, inorganic refuse, such as cinders and ashes, may be employed, or sufficient earth may be dug from a nearby knoll or hill, care being observed that in so doing a depression capable of holding water is not made. Low marshy lands adjacent to rivers, lakes, or the sea may be filled by pumping silt or sand. Many cities in our southern states have used city refuse to fill up anopheles breeding places. One of the very best means of clearing the land of the numerous small natural collections of water is to place it under cultivation.

When filling is not practicable, good and permanent results may be ob-

¹ Le Prince and Orenstein, *Mosquito Control in Panama*. G. P. Putnam's Sons, New York, 1916.

tained by drainage. As a rule, the draining of ponds, pools, or marshes is the simpler and cheaper method. By the draining of marshes is meant the draining of the pools of stagnant water, or in the case of coastal marshes the draining of the stagnant fishless pools that are beyond the reach of the ordinary tides; it does not necessarily include the draining of the water-soaked soil itself. The underdraining of wide acreages of our arable land in the Middle West has been very effective in suppressing the malarial mosquito. Mosquito breeding places in the pools in coastal marshes may be suppressed by connecting them with tidewater, so that they may be freely scoured by the daily tides. Marshy lands may be drained simply by means of ditches. These must be of sufficient depth completely to empty the pools under treatment and have sufficient fall to prevent stagnation in the course of the ditch itself. Ditches should have clean sides and must be inspected at frequent intervals. Care must be taken to see that they do not become choked and that they remain accessible to fish.

Artificial Breeding Places.—There are many artificial or man-made breeding places. No body of water is too small for a mosquito nursery. They breed in puddles by the roadside; in water that accumulates in furrows in gardens or fields, especially in clay; in street gutters and sagging house gutters; in holes in rocks; in hollows of trees, in pitcher plants; in discarded tin cans and broken pottery; in flower pots, bottles and fire buckets; in chicken pens in poultry yards; in water cups on the frames of grindstones; in baptismal fonts; in catch basins, or anywhere that a gill of water is allowed to stand.

In a city, the permanent elimination of artificial breeding places for mosquitoes depends upon a good water supply in a modern, closed system. This will permanently do away with the necessity of cisterns, barrels and tubs for the storage of water about the premises. When domestic storage is a necessity, care must be taken to keep the containers free of mosquitoes by (1) mechanical protection, such as tight covers and screens, and (2) fish.

Water barrels, cisterns and other containers should have tightly fitted covers, which should be fortified with screens. For emergency work, burlap, sheeting or several thicknesses of cheesecloth, or better still wire screening held in place by a well fitting hoop, serve this purpose very well. Whatever the form of cover used, it should not be removed except for cleaning or refilling. The water should be drawn from a spigot. Keeping fish in such containers is quite feasible and does not harm potable water.

One of the chief sources of malaria in the South at present is the artificial breeding places developed by impounding rivers for water power. The sides of the reservoir must be kept clean of vegetation and floatage and well stocked with minnows (see the admirable Alabama State Regulations in this regard). Railroad and highway improvements often involve the building of small dams, culverts, ditches and gradings; these create small impounded bodies of water and make ideal breeding places for *Anopheles* mosquitoes.

Cesspools and privy vaults should be done away with and be replaced

with dry earth closets or a water carriage system. Where this has not been done, they should be frequently and copiously oiled.

Coal Oil and Larvicides.—When radical measures, such as filling in or draining, are not practicable, resort may be had to coal oil. Coal oil upon the surface of the water poisons and also suffocates the larvæ and pupæ. A light quality of oil should be used,⁸ but all the lighter distillates from crude oil are useful as larvicides. Usually, two parts of crude oil are thinned with one part of kerosene⁹ and this is applied either from a knapsack or from a pressure boiler. Sufficient oil should be used to cover the entire surface with a thin film. As the oil is volatile, it may disappear within a few days. Furthermore, the film, which should be intact to be effective, may be broken by winds. A strong wind will blow all of the oil to one side, thereby largely defeating the object desired. It is, therefore, important to repeat the oiling regularly at intervals of about one week or twelve to fourteen days for anopheles. Oiling, though fairly effective when properly carried out, is only a temporary expedient, and in the end is rather expensive (see also page 273).

Griffiths and Hazlehurst have developed a method of distributing oil under pressure which is very effective. The oil (crude oil $\frac{2}{3}$, kerosene $\frac{1}{3}$) is placed on a metal tank under sixty pounds air pressure and is sprayed over the surface of the water by means of a Bordeaux spray. The oil is distributed in a fine mist and is effective even in places where there is heavy vegetation. The tank is carried on a small motor boat or rowboat and the radius of the spray is about fifty feet.

Paris green causes destruction of anopheles larvæ, regardless of vegetation and of whether the water is standing or flowing. It does not kill the eggs or pupæ. The cost is relatively cheap and the results good. Barber¹⁰ has devised an excellent method for the destruction of anopheles larvæ. Paris green is mixed with ashes in the proportion of one part to one hundred, and this is thrown into the air on the windward side of the pond. Surface tension holds the fine particles on the surface of the water, and the larvæ eat the Paris green and are destroyed. The method will not kill culex larvæ. It does not injure the potability of the water.

Many other substances will kill larvæ, but few are practical. "Niter cake," a by-product of powder and dynamite factories, is effective in water barrels. Carbide may be used in non-potable waters. A lump is dropped in a pool and a tenacious film forms on the surface of the water. It is especially recommended for sewer outlets, privy vaults, pot-holes, old cisterns, and the like.

Fish Control.¹¹—Fish are the most effective of the natural enemies of the

⁸ The best oil for the purpose is known to dealers as "Fuel oil 29-31."

⁹ Soaps and alkalies must not be added to the mixture, for they prevent spreading and film formation. The oil sprays and spreads better if warm.

¹⁰ *U. S. Pub. Health Rep.*, Reprint No. 714, 1921.

¹¹ "The Use of Fish in Mosquito Control"; "The Use of an Indigenous Fish in Combating Malaria," by H. H. Howard; "Fish as Mosquito Destroyers," by M. E. Connor; "Some Personal Experiences with Fish as Antimosquito Agencies in the Tropics," by

mosquito. Fish control now takes first rank in anti-mosquito work as a practical measure. Many independent investigations have shown that within certain definite limits excellent results can be obtained by this biological method.

The best way is to stock lakes, ponds, reservoirs and streams, as well as small pools, ornamental fountains, cisterns and barrels, with minnows or other larvivorous fish. In some cases the fish may be admitted to the ponds, pools and ditches by connecting them with larger bodies of water. *Gambusia affinis*, or the common top minnow, is the most valuable fish for this purpose in the United States. It is a hardy fish, readily adaptable to many different conditions; it even thrives in brackish water. A medium sized female gambusia will eat 165 large mosquito larvæ in a single day. An indigenous species is usually best, for imported varieties must sometimes be acclimated. The margins of pools, rivers and other bodies of water must be kept free of reeds and water plants, so as to permit the fish to reach the edges—a favorite breeding place for mosquitoes. Furthermore, the fish must not have too much vegetation and other food to divert the appetite. In still waters, fish are much more effective than in free-running streams. Wave action at the margin will kill larvæ.

In general, small fish of the families of *Cyprinodontidæ* or *Poecilidæ*, found in low altitudes in all parts of the world, are most useful for malaria control. One variety, known as "millions" (*Lebistes reticulatus*) has earned a reputation, perhaps not entirely deserved, as a mosquito destroyer in the Barbados and elsewhere. It is closely related to the *Gambusia affinis*, and like that species perhaps does its best work in its native habitat. In India, where fish have been experimented with extensively, the genus *Haplochilus* has been found to meet all requirements. Under different conditions different fish may prove satisfactory.

For yellow fever control, where the fish must live in containers, the *chalaco* (*Dormitator latifrons*) was found by Connor, in Ecuador, to be the most satisfactory. The *life* (*Pygidium piura* E.) has been used in Peru. Fish for *aedes* control must be frequently renewed, for only a few are allowed in each container.

Screening.—Mosquito screens are the obvious and most effective single measure for personal prophylaxis where disease-carrying mosquitoes exist. In order to be effective, the screening must be intelligently carried out with careful attention to details. The screen itself must be sufficiently close to keep out the mosquitoes. Some of them are able to squeeze through surprisingly narrow chinks. I was able to demonstrate, in the experimental work at Vera Cruz, that the *aedes* mosquito can pass a metal wire screen containing

Daniel M. Molloy: "Suggestions for Developing a Campaign to Control Yellow Fever," by M. E. Connor: "The Use of Fish in the Control of Yellow Fever in Peru," by Henry Hanson and L. H. Dunn. These pamphlets may be obtained by applying to the International Health Board of the Rockefeller Foundation, New York City. See also "The Use of Fishes for the Control of Mosquitoes in Northern Fresh Waters of the United States," by J. Percy Moore, Bureau of Fisheries, Document No. 923, 1922.

sixteen strands or fifteen meshes to the inch, but cannot pass one containing nineteen strands or eighteen meshes to the inch. When the screen consists of a fabric which is apt to pull out of shape so that some of the meshes are larger than others, it is advisable to use a net woven closer than twenty strands to the inch. Experience in malarial and yellow fever districts has taught this lesson, so that it is customary in those countries to use a rather closely woven material resembling nainsook. The screens should have a mesh of not less than sixteen to eighteen meshes to the inch, depending upon the gauge of the wire used in their construction. For the former size, the wire gauge should have a diameter of .016 of an inch; for the latter, .0125 (English standard). Metal screens made of iron wire are cheapest only when first cost is considered. They hardly last a season unless painted, in which case the size of the mesh is considerably reduced and interferes with ventilation, a serious objection in hot weather or a tropical climate. The wire should be of bronze, galvanized iron, copper, or monel metal. When exposed to sea air, only pure copper or monel metal wire is durable. The copper should not contain more than one-half of one per cent of iron.

The screening should include the entire house, or at least those parts that are occupied. In the tropics it is better to screen the "galleries" than each individual window. In any case, frequent and repeated inspection should be made to discover breaks in the screen or openings due to warping of the woodwork. Care must be exercised not to overlook fireplaces, ventilators, and other openings. The door should be guarded by a screened vestibule of such a depth as to make it difficult for a person to hold both doors open at the same time. The screen door should open outward and, if possible, should be exposed to the direct sunlight during the day, without vines or nearby vegetation of any kind to protect and lodge the mosquitoes. An electric fan directed outward is a very good device to prevent mosquitoes flying through the doorway. In addition, a whiskbroom or feather duster should be at hand to brush off the insects that may rest upon the clothing. A screened house is safe only to careful and intelligent people.

In addition to screening the house, mosquito bars over the bed will be found necessary in mosquito-infested places. It is best to suspend the mosquito bar from the ceiling and carefully gather the bottom together so as to keep the insects out during the day time. At night the bar is usually tucked in around the bed so as to leave no openings. Mosquitoes have no trouble in biting through the meshes of the bar, provided a restless sleeper comes close enough to it. To avoid this the bar should be spread and weighted to the floor.

Screening is useful but only a mechanical expedient; it is inconvenient, expensive, often imperfect, and does not strike at the root of the problem.

Persons who are required to go out at night in a malarious district, or who must expose themselves during yellow fever times, may screen themselves effectively with a veil of mosquito netting hanging from a broad-brimmed hat to the shoulders and chest. The hands and wrists may be protected with

gloves, and the ankles with leather leggings or other suitable mechanical device.

Mosquito Repellents.—A number of volatile and odorous substances, such as camphor, oil of pennyroyal, peppermint, citronella, creosote, etc., are used for this purpose. None of them is dependable. At most, their action is transient and they give a false sense of security.

The diseases known to be conveyed by mosquitoes are: malaria (*Anopheles* spp.), yellow fever (*Aedes aegypti*), filariasis (*Culex*, *Aedes* and *Anopheles*), dengue (*Aedes aegypti*); and doubtless others.

MALARIA

Malaria is a group of closely related infections due to protozoan parasites that live chiefly in the blood, and which are transmitted by anopheles mosquitoes. The chills and fever are usually paroxysmal, coming on every day (quotidian), every other day (tertian), or every third day (quartan). Chronic cases are associated with anemia and an enlarged spleen. Mixed infections occur; there is some tolerance to the parasite, but no immunity; relapses are common; carriers are frequent. The incubation period in both the mosquito and in man is twelve to fourteen days, or longer. In the mosquito, the period of incubation is largely dependent on the temperature; in man, upon the amount of initial infection and resistance of the host.

At least three separate malarial parasites of man are known, namely: (1) *Plasmodium malariae* (Laveran), quartan fever; (2) *Plasmodium vivax* (Grassi and Feletti), tertian fever; and (3) *Plasmodium falciparum* (Welch), estivo-autumnal or tropical malaria. These are closely allied hemosporidia or blood parasites. They produce diseases with well-defined clinical differences, but having the same etiology and mode of transference, so that, as far as prevention is concerned, they may be regarded as one infection. Quartan is comparatively mild and rare; tertian, common and apt to relapse; estivo-autumnal, most malignant and pernicious in tropical places.

Malaria is one of the most prevalent of all preventable diseases; it is the scourge of the tropics. The cause of this infection was one of the first to be revealed (Laveran, 1880), and its mode of transmission was one of the brilliant discoveries in sanitary science (Ross, 1895). Despite the fact that we have more exact knowledge of malaria, considering the difficulties of the subject, than perhaps any other disease, despite the fact that we have accurate means of diagnosis and a ready cure, and despite the fact that we have assured measures of prevention, malaria counts its victims by the hundreds of thousands annually. It has a wide geographic distribution, occurring from the Arctic Circle to the equator, but is more prevalent and more virulent in warm moist climates.

A gradual improvement in our southern states is noticeable; nevertheless, there are still about 250,000 cases annually with half as many carriers. The distribution is very unequal, but chiefly rural, and now distinctly focal. Cities

are practically free. Wherever malaria prevails, and almost in direct proportion to its prevalence, the population is generally subnormal physically, mentally and economically. As malaria lessens, prosperity increases due to improvement in strength and energy of the people, and with increased prosperity comes land better cleared, better drained and of higher value, houses screened, and better hygiene generally—an endless chain of betterment. It is only in this way—through mosquito control—that the results are satisfactory and abiding.

Malaria has all the earmarks of a communicable disease. The infection was probably brought to America by slaves from Africa. The decline of the glory that was Greece was doubtless hastened by malaria, likewise brought to that country by slaves and captives. Epidemics are common. In recent years an epidemic wave spread over southern Russia: in 1922, 2,490,982 cases were reported, and in 1923 the number increased to 5,566,856. The story of the introduction and spread of malaria in Mauritius is typical. Mauritius was free of malaria until 1865, when the chance call of a ship at Petite Rivière Bay in the Black River district introduced *Anopheles costalis* from the malarious African coast. The disease nearly decimated the island in 1867 and 1868: 31,800 of the population succumbed in this epidemic. From then on, malaria continued endemic, claiming 3,000 to 4,000 victims each year.

Malaria is peculiar to man; there is no animal reservoir. Many species of animals have a malarial-like infection closely resembling malaria in man; for example, Texas fever of cattle, piroplasmosis of dogs and sheep, proteosoma of birds, etc. *Anopheles* are the only mosquitoes capable of transmitting the malarial parasites of man. Both man and the mosquito are necessary to complete the life cycle of the plasmodium. Man is the intermediate host harboring the asexual phase, and the mosquito is the definitive host harboring the sexual phase of the life cycle of the plasmodium.

Mosquito Transmission.—In nature, malaria is transmitted only by the bite of the female *anopheles* mosquito. Experimentally, the infection may be transferred by injecting blood (containing the parasites) of one person into the blood of another. The infection is now purposely produced for the treatment of the parietic dementia, with good results. Nearly 2,000 years ago Varro and Columbella mentioned the possibility that the disease was transmitted by mosquitoes. In Africa some savage tribes called malaria the "mosquito disease." In 1848 Nott, of New Orleans, considered the matter proved from biological analogies. In 1854 John Snow suggested mosquito transmission from analogy of flukes and the snail. In 1882 King, of Washington, vigorously advocated the mosquito theory, based upon philosophical deductions but no proof. In 1884 Laveran suggested mosquito transmission as probable. In 1894 Manson elaborated the mosquito theory and inspired Ross, of the Indian Army Medical Service, who in 1895 demonstrated that the crescents of estivo-autumnal malaria undergo changes in the mosquito. In 1896 Bignami advocated the theory and compared it to the transmission of Texas

fever by the tick. In 1897 Ross¹² published further convincing observations upon the development of the estivo-autumnal parasite in the mosquito. In 1897 MacCallum discovered an important missing link in the life cycle by observing the flagellum of the microgametocyte (male) fertilize the macrogametocyte (female) with the formation of the vermicle. These observations were made upon *Halteridium* or malaria of birds; later he saw the same phenomenon in estivo-autumnal malaria. The life cycle of the malarial parasite has been confirmed by Daniels, Koch, Grassi, Bignami, Celli, Manneberg, Schaudinn, and many others.

Dramatic evidence that malaria is transmitted by the mosquito was furnished by Sambon and Low, of the London School of Tropical Medicine, and Terzi, who lived during the three most malarial months of 1900 in Ostia, a very malarial locality of the Roman Campagna. These observers escaped infection simply by keeping within their well-screened huts from before sundown until after sunrise. Convincing proof was furnished in 1900 by P. Thurber Manson and George Warren, who were bitten by infected mosquitoes forwarded in cages from Italy to London.

The Malarial Mosquitoes.¹³—At least twenty-five species, in six genera of the subfamily *Anophelinae* are more or less definitely known to carry malaria.¹⁴ The chief culprits are: in Europe, *Anopheles maculipennis*; in tropical America, *A. argyrotarsus* or *albipes*; in temperate America, *A. quadrimaculatus*, which is very similar to *A. maculipennis*, also *A. punctipennis* and *A. crucians*; in India, *A. sinensis*; in Africa, *A. costalis*. Darling¹⁵ found that 70.8 per cent of *Anopheles albimanus* induced to bite malarial patients became infective, while with *Anopheles pseudopunctipennis* only 12.9 per cent could be infected.

Though *A. punctipennis* and *A. crucians* have been infected with malaria under laboratory conditions, extensive field work indicates that *A. quadrimaculatus* is the important vector of malaria in the United States. It transmits all three types of the disease.

The anopheles mosquitoes are brownish and rather large. They may be distinguished by the fact that the palpi in both the male and the female are at least as long as the proboscis. Only the female transmits the infection. It sits more or less at right angles upon the wall, the head, thorax, and abdomen being in a straight line. Contrary to the yellow fever mosquito, the malarial mosquito is nocturnal in its habits and breeds chiefly in open ponds, puddles, and natural collections of water in the woods, fields and swamps. Each species has its own particular habits and habitat.

¹² A graphic history of malaria and its ravages and a vivid story of the discovery of its mode of transmission is given in Ronald Ross's "*Memoirs: With a Full Account of the Great Malaria Problem and Its Solution*," Dutton, N. Y., 1923.

¹³ See pp. 277-284 for a discussion of mosquitoes in general.

¹⁴ See Craig, *The Malarial Fevers*, New York, 1909, and Knab, *Am. J. Trop. Dis.*, 1913, I, 1, 37, also Castellani and Chalmers, *Manual of Tropical Medicine*, 1919, for full list, classification and geographical distribution.

¹⁵ *J. Am. M. Ass.*, 1909, 53: 2051.

The mosquito becomes infected upon drinking the blood containing the micro- and macro-gametocytes. It requires about twelve days before the sporozoites appear in the salivary glands of the insect. It cannot, therefore, transmit the infection to another person until the lapse of this extrinsic period of incubation. The infected mosquito may live a long time and infect more than one person successively. The malarial parasite does not seem to harm the mosquito.

The parasite will not develop in the mosquito when the mean temperature is below 60° F. It can, however, survive at much lower temperatures for a short time. King¹⁶ has shown that the tertian parasite is able to survive exposure to a temperature of 30° F. for two days; 31° F. for four days; and a mean temperature of 46° F. for seventeen days. The sporonts of the estivo-autumnal parasite have resisted 35° F. for twenty-four hours. The anopheles mosquitoes are not active at temperatures below 40° F. nor above 104° F.

A. quadrimaculatus, our chief malarial mosquito, is essentially a pond breeder, and flies about a mile in all directions irrespective of the wind. It hibernates during the winter, and the insects which hatch out in the spring are not infective. These early mosquitoes are weaklings and die soon after oviposition. Very few adult insects are therefore found in the early spring. They become more plentiful as the summer advances and increase into the fall, corresponding to the prevalence of the disease. While *A. quadrimaculatus* breeds in the open, the adult mosquito is domestic in its habits. In making anopheline surveys, the number of adult anopheles mosquitoes in and under the houses are used as an index of prevalence. *A. punctipennis* is most prevalent in winter and early spring; *A. crucians* is most prevalent in the late spring, April to June, when malaria is not prevalent. They are wild and are not found with great frequency in the houses. They are not efficient carriers of malaria in the United States. Effective and economic malaria control depends upon a knowledge of these facts.

Immunity.—A person who once has had malaria seems more prone to have subsequent attacks, which, in fact, may be relapses. However, repeated infections leave a pronounced resistance. Tolerance to the infection is also indicated by the fact that the symptoms often subside without treatment. In malarious regions many natives carry the parasites in their circulating blood

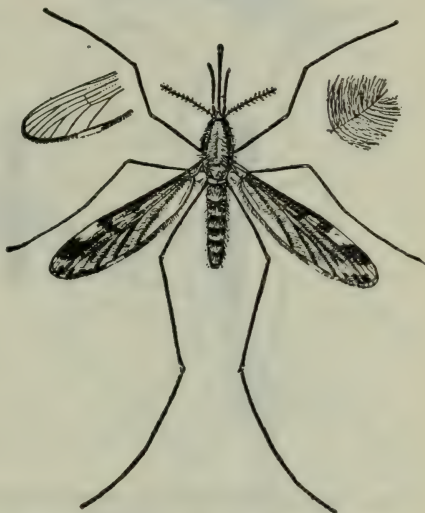


FIG. 19.—ANOPHELES PUNCTIPENNIS.

¹⁶ *J. Exper. M.*, 1917, 25: 495.

without manifestations of the disease. These carriers are important factors in spreading the infection in endemic areas, and must be taken into account in preventive measures. In endemic regions carriers far outnumber the cases at any one time. It requires much more quinin to sterilize a carrier than to cure a clinical case.

There is no true racial immunity in this disease. Practically all persons who receive the infection for the first time are susceptible. The freedom from malaria which some persons seem to enjoy may be accounted for partly by the fact that mosquitoes seldom bite such persons. It is well known that on account of body odors, or what not, mosquitoes do not bother certain individuals.



FIG. 20.—*ANOPHELES QUADRIMACULATUS*—MALE AND FEMALE.

(From Bureau of Entomology, U. S. Department of Agriculture, Washington, D. C.)

Where Negroes do not suffer as severely from malaria as whites, although they have no more immunity to infection, but they develop "tolerance" and complain less about their illnesses. Where Negroes and whites are living under the same conditions, the parasite rate and the spleen rate among Negroes is usually as high or higher than in the whites.

Individual resistance varies in different individuals and in the same individual at different times. The parasite may remain latent in the spleen or other organs for years. Exposure, overeating, fasting, overwork, worry, or anything that lowers the vitality of such individuals predisposes to an attack of malaria. The disease often breaks out in persons in good health leaving a malarial region for a health resort, whether mountain or seashore. I was enabled to confirm this observation upon the returning transports from Cuba following the Spanish-American War, when many cases of malaria broke out among the troops previously in good health upon exposure to the cold winds about Cape Hatteras. Personal prophylaxis, therefore, includes careful attention to personal hygiene.

Carriers.—About 20 per cent of apparently healthy natives in most malarial regions harbor plasmodia, and about half of these have the parasites in their blood. In badly infected regions, as high as 60 to 70 per cent are carriers. In localities less severely involved, the number of latent infections and carriers is very much less.

Unrecognized malarial infection and incomplete treatment lead to the carrier state. The recognition of carriers depends upon microscopic blood examination. The thick film method is useful. The prevention of carriers depends upon: (1) the control of malaria in the district, (2) proper treatment of cases with quinin (page 291), and (3) recognition and treatment of carriers until free of infection.

The malarial index of a community can be determined by the spleen rate, which consists in palpating the spleens of children between the ages of two and twelve. The enlarged spleens tell the story of malarial prevalence. This method is much easier than any other and sufficiently reliable.

Prevention.—The successful suppression of malaria requires a combined attack upon the mosquito and the parasite in the human host. *Ultimate success rests upon the suppression of the mosquito.* Immediate relief is most quickly gained by measures directed against the infection in man. Screening and quinin prophylaxis, while practical, are only temporary measures. Each of these needs consideration.

Measures Directed against the Mosquito.—Celli¹⁷ thought that although the destruction of mosquitoes is possible in the laboratory and in small areas, the difficulties in extensive areas are generally in-

superable. So Gorgas thought of aëdes, when he began work in Havana, but such is not the case. All anopheles mosquitoes do not need to be destroyed; in fact, extermination is impossible. It is entirely a question of relative density of human population and anopheline population. Where human population is dense, as in cities, a low anopheline index may produce malaria and suppressive measures must be complete. When human population is sparse, as in rural districts, a comparatively high anopheline index may exist without danger of an outbreak of malaria. In some places control fails for want of knowledge.

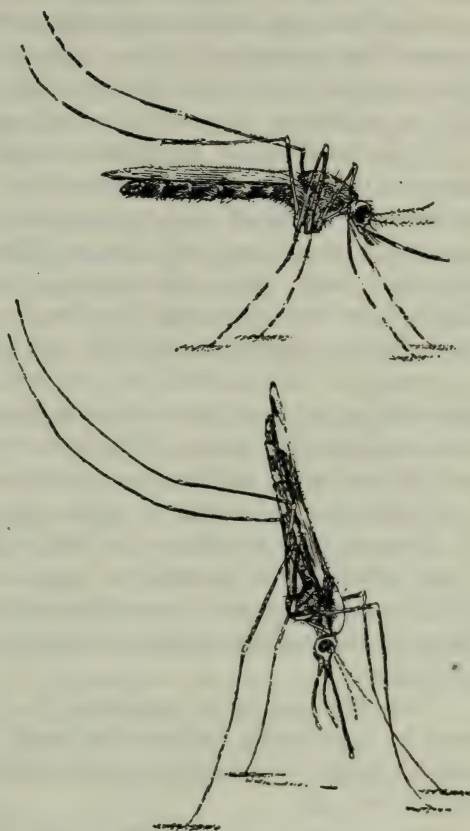


FIG. 21.—RESTING POSITION OF CULEX (ABOVE) AND ANOPHELES (BELOW)

(From Bureau of Entomology, U. S. Department of Agriculture, Washington, D. C.)

¹⁷ Cited by Leslie, *Proc. Imp. Malar. Conf., Simla, 1910, 8.*

Le Prince¹⁸ demonstrated that at Gatun anopheles will fly great distances. Le Prince and Griffith registered flights of a mile in South Carolina by *A. quadrimaculatus* from breeding places producing profusely. This was confirmed by Geiger in Arkansas and by Metz for *A. crucians* in Alabama. There is, however, very little doubt that in the majority of places *malaria is a local infection*; that is, the mosquitoes usually acquire the gametes and transmit the sporozoites within a restricted area. Each individual house or farm must be considered, for one farm may be practically uninhabitable, while an adjoining one may be free of malaria.

If the breeding of Anophelinae could be stopped malaria would cease. Mosquito suppression is fundamental and radical. The best way to abolish the breeding places of malaria mosquitoes is to fill up low places or to dry the surface of the land with drains. These two measures hold first place as permanent work. The underdraining of large areas of our arable land of the Middle West with tiled drain has been very effective in suppressing malaria. Open ditches properly constructed and cared for are likewise effective. In the tropics the ditches should be lined with cement, on account of the luxuriant vegetation which soon interferes with their efficiency or may actually convert them into breeding places. The open ditches are much the cheapest in first cost, but not when maintenance is reckoned. The draining of swamp lands is an engineering problem in which the economic factor looms large. The tile drainage costs a minimum of \$20 to \$30 an acre. Dynamite has proved a very effective and inexpensive means of constructing drainage ditches in thickly wooded swamps. One of the very best means of destroying the breeding places of the malaria mosquito is to clear the land and to keep it under cultivation.

When drainage is not practical, the number of mosquitoes may be kept down by introducing fish into the pools, streams, ditches, and other collections of water. Upon limited water surfaces the larvæ may be killed with a film of coal oil.

Large open spaces cause the destruction of a number of mosquitoes, as they cannot live long in the hot sun. Therefore, clearing the brush and high grass, which furnish shelter to the insects, aids in preventing wild mosquitoes approaching dwelling houses. Keeping domestic animals between the swamps and the residence acts as a buffer, for many mosquitoes prefer domestic animals to man. In this country, however, anopheles have not become zoöphilic, as described by Roubaud for France. The use of screens and culicides has already been referred to (pages 281, 282). The best time to begin antimosquito measures is as early in the spring as possible, so as to prevent infection of the new summer brood of mosquitoes. For details of mosquito control, screening, oiling, ditching, etc., see pages 279 to 283.

Wickliffe Rose states that for the average town in our southern states having a thousand or more inhabitants and a reasonably high infection rate,

¹⁸ Tr. Fifteenth Internat. Cong. Hyg. & Demog., V, Part II, 544, U. S. Pub. Health Rep., 1917.

malarial control by antimosquito measures is economically feasible and a sound business investment.

Personal Prophylaxis.—Persons visiting or residing in a malarious region should be particularly careful not to expose themselves at night time. The experience of Sambon and Low on the Roman Campagna (page 286) is instructive and should be imitated. The location of the residence is important. The safest place is in the city, and distant from the native quarter, because infections of various sorts are there most concentrated. The dwelling should if possible face the trade winds. The house should be on high land if practicable, as it is an old observation that the malarial mosquito does not like to fly high. Physical vigor and the avoidance of fatigue, exposure and excesses are helpful. If it is necessary to go out in the night time, one may protect himself by the use of gloves and mosquito netting hanging from the helmet to the shoulders. Care must be taken to guard the ankles against mosquito bites. As all these measures require much time and attention to details, they are usually not efficient in actual practice. Therefore, quinin prophylaxis is much used.

Quinin Prophylaxis.—Quinin does not prevent infection, although it does usually prevent clinical symptoms. The prophylactic use of quinin lowers both the malarial morbidity and mortality rates. The administration of quinin to healthy individuals is neither ideal in theory nor satisfactory in practice. The method has been tried extensively, but in the main has proved disappointing. It is expensive in the long run, difficult to administer to an entire community, and the effects are only transient. As a community measure, it needs willing coöperation and intelligence of the people. The amount of quinin administered is not sufficient to prevent the carrier state. Symptoms have been controlled, but it seems impracticable to eradicate the infection with quinin; at best it is a temporary expedient. It cannot take the place of radical and permanent measures aimed at mosquito breeding places, such as drainage, filling, ditching and oiling.

Quinin prophylaxis is particularly useful where screens and mosquito bars are not available, as in camping, marching, traveling, or where the occupation takes one out at night. Persons visiting malarial districts for a short time may protect themselves with quinin, but it is not advisable to stop the use of quinin on the advent of cold weather or on leaving the malarial district, even if one has not had symptoms. The quinin should be continued for one or two months, until the carrier stage is overcome.

The dose and time of administration are important factors in quinin prophylaxis. From two to five grains a day have proved effective, but no reliance should be placed on less than ten grains daily. Even this amount at times fails to prevent the development of symptoms. The amount depends upon the kind and intensity of the infection and the experience of the region.

Quinin Treatment.—Quinin treatment should be aimed not only to relieve symptoms and prevent relapses, but to prevent the carrier state and the danger of infection to others. Successful treatment means killing all the plasmodia

in the body. This is accomplished in the majority of cases by the standard method of treatment,¹⁹ which is as follows:—

“For the acute attack, 10 grains of quinin sulphate by mouth three times a day for a period of at least three or four days, to be followed by 10 grains every night before retiring for a period of eight weeks. For infected persons not having acute symptoms at the time only the eight weeks’ treatment is required.

“The proportionate doses for children are: Under 1 year, one-half grain; 1 year, 1 grain; 2 years, 2 grains; 3 and 4 years, 3 grains; 5, 6 and 7 years, 4 grains; 8, 9 and 10 years, 6 grains; 11, 12, 13 and 14 years, 8 grains; 15 years or older, 10 grains.

“It is not claimed that this is a perfect or even the best treatment in all cases, but it is our belief that it is a good and satisfactory method for practical use to prevent relapse and transmission to other people.”

One disadvantage of the standard method is that it is not practicable to get the entire treatment taken as recommended. Another drawback is that parasites remain in the blood in 25.4 per cent.²⁰ In a heavily infected, poorly nourished population, the standard treatment failed to free a large percentage of cases of either asexual or sexual forms of the parasites. A drug for malaria superior to quinin is much needed.

Quinin given intravenously saves lives, especially in the pernicious, estivo-autumnal type. Intramuscular injections are painful and apt to cause abscesses.

Blackwater fever occurs where malaria is most intense. The prevention of blackwater fever depends upon the prevention of malaria.

REFERENCES

- DEADRICK. *A Practical Study of Malaria*. Philadelphia, 1909.
 CRAIG. *The Malarial Fevers, Hemoglobinuric Fever and the Blood Protozoa of Man*. New York, 1909.
 HENSON. *Malaria Etiology, Pathology, Diagnosis, Prophylaxis and Treatment*. St. Louis, 1913.
 ROSS. *The Prevention of Malaria*. London, 1910; also his *Memoirs*, 1923.
 The original discovery of the malarial parasite was announced by Laveran in the Académie de Médecine, Paris, Nov. 23, 1880, and Dec. 28, 1880.
 Publications of the United States Public Health Service, Washington, D. C., and the International Health Board of the Rockefeller Foundation, 61 Broadway, New York City.

YELLOW FEVER

Yellow fever is an acute febrile disease characterized by jaundice, albuminuria and a tendency to hemorrhages, especially from the stomach (black vomit). It is due to a filtrable virus, and is spread only by a mosquito, *Aedes aegypti*. One attack produces a lifelong immunity. All ages and both

¹⁹ U. S. Pub. Health Rep., 1919, 34: 2959. ²⁰ U. S. Pub. Health Rep., 1925, 40: 539.

sexes are susceptible. Negroes are as susceptible as whites, although the disease in them is milder and rarely fatal. There are few sequelæ. The case fatality is ordinarily from 25 to 37 per cent, but occasionally as low as 5 per cent. The incubation period is usually two to five days, maximum six days.

That yellow fever is a vanishing disease is clearly shown in Figure 22.²¹

This splendid result has been achieved by aggressive and well-considered measures under the guidance of the International Health Board of the Rockefeller Foundation. Yellow fever has been suppressed in the Western Hemisphere, and the only known foci left in the world are the original endemic area on the west coast of Africa and a few residual foci in tropical America. The disease is probably not indige-

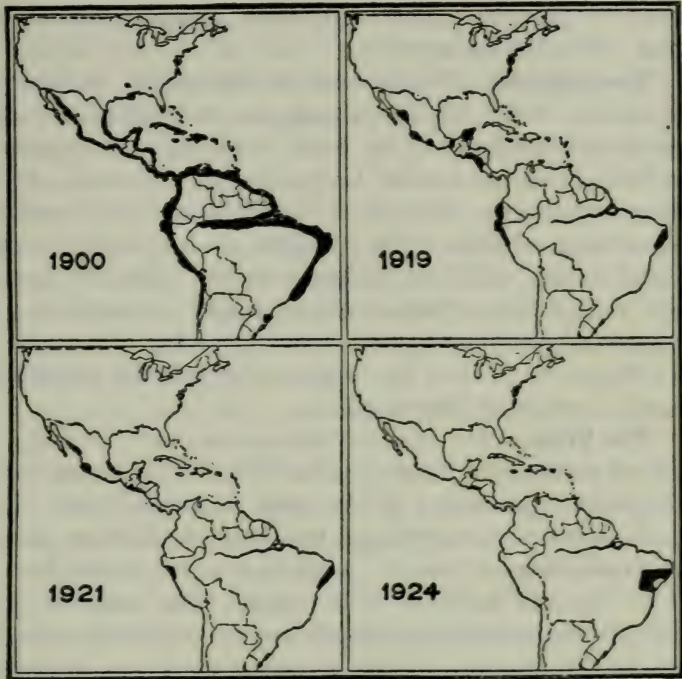


FIG. 22.—YELLOW FEVER IN RETREAT FROM THE WESTERN HEMISPHERE.

(From the Annual Rep., Int. Health Bd., 1924.)

nous to America, but was brought to our shores with the slave trade.

The Yellow Fever Mosquito.—The yellow fever mosquito has a wide distribution, ranging from 38° south to 38° north latitude. Although it is prevalent in the Philippines and other parts of the world, where it is presumably infectable, these regions have been spared on account of their isolation. There are nineteen species of *aedes* in Africa, but only one of them, *Aedes aegypti*, has flourished in America.

This mosquito was first called *Culex fasciatus*, which was changed to *Stegomyia fasciata* as soon as its disease-bearing importance was discovered. Since then, it has been divorced and remarried in the nomenclature family many times. It soon became *Stegomyia calopus*, which was changed to *Aedes calopus* by Coquippett; later, it was *Stegomyia argenteus*, and it is now *Aedes*

²¹ Ann. Rep. Int. Health Bd., 1924.

egypti. All these names are found in the literature. It is still commonly called stegomyia.

The yellow fever mosquito is a domestic insect. It breeds by preference in any standing water about the household, such as cisterns, rain barrels, buckets, bottles, old cans, etc. It does not breed in the fields, woods or swamps, which are favorite resorts of the malarial mosquito. *Aedes egypti* does not fly far of its own volition, but shows a cat-like tendency to remain about its place of birth or adoption. These facts have an evident bearing upon preventive measures.

Transmission.—Yellow fever is transmitted in nature only by the bite of infected *Aedes egypti*. A mosquito after drinking infected blood during the first three days of the fever is not able to transfer the infection to another person until about twelve days have elapsed. During this time the parasite undergoes its cycle of development. This constitutes the extrinsic period of incubation. The mosquito, once infected, remains so during the rest of its life, which may be some months. Only the female mosquito transmits the infection. The male *Aedes egypti* is a vegetarian, its proboscis being too soft to penetrate the skin. A single sting of a single infected mosquito is sufficient to produce the disease. An infected mosquito may infect more than one person at different times.

The Virus.—The virus is “ultramicroscopic”; in fact, it passes the closest grained pores of the finest porcelain filter. Noguchi has described a spirochete (*Leptospira icteroides*) as the cause of yellow fever. It closely resembles *Leptospira icterohamorrhagiae*, the cause of infectious jaundice.

Prevention.—There is a weak link in the yellow fever chain on account of the domestic habits of *Aedes egypti*. This mosquito is readily suppressed and it is therefore comparatively easy to control the disease and even exterminate the infection. So far as known, there is no animal reservoir. Yellow fever in children is frequently mild and usually unrecognized, and thus the disease is kept alive in large endemic foci. It dies out spontaneously in a community with a stationary population, burning itself out for want of susceptible material to feed the flame. Yellow fever prevention by control of the human host is not practical because of missed cases, and especially because the disease in children is mild and difficult to recognize.

Aedes egypti is best controlled by substituting a closed modern water system for cisterns, barrels and household containers. By this one measure, yellow fever disappeared from Philadelphia, Baltimore and many other cities long before its mode of transmission was suspected. Where this is not practical, the cisterns, tanks, etc., must be tightly closed and screened against mosquitoes, or each container may be stocked with fish. This has proved effective, practical and economical in several recent yellow fever campaigns in Central and South America (see page 282). Another measure is to oil or periodically empty water containers. Broken bottles and all other breeding places must be destroyed. One preferential breeding place, such as a water bucket, may be left to prevent the mosquitoes seeking compulsory and unusual

breeding places to deposit their eggs. If this be done, the container must be closely watched and frequently emptied to prevent hatching. Screening as an accessory measure is helpful, but cannot control yellow fever, on account of the delays in discovering cases and because of the many missed cases, especially in children. In any yellow fever campaign, communities should be urged to screen all cases of fever until diagnosed. Quarantine for yellow fever is discussed on page 534.

Historical Note and References.—Charles J. Finlay studied the relation of the mosquito to yellow fever as far back as 1882 and 1883. The first insects used by the United States Army Commission to bring about the demonstration of the new doctrine were received from the hands of Finlay. Finlay believed that the cause of the disease was a micrococcus and considered that the insects were capable of transmitting the infection a few days after they had stung a yellow fever patient. Sternberg's studies upon yellow fever are published by the Government as a report of the United States Marine Hospital Service on the "Etiology and Prevention of Yellow Fever," 1890. Carter's observations at Orwood, Mississippi, upon the extrinsic period of incubation were published in *New Orleans Medical and Surgical Journal*, May, 1900, and the *Medical Record*, June 15, 1901.

The work of the United States Army Commission appeared in the following publications:

"The Etiology of Yellow Fever—a Preliminary Note," *Proceedings of the 28th Annual Meeting of the American Public Health Association*, Oct. 22-26, 1900; also *Philadelphia Medical Journal*, Oct. 27, 1900.

"The Etiology of Yellow Fever—An Additional Note," *Journal American Medical Association*, Feb. 16, 1901.

"Experimental Yellow Fever," *American Medical Journal*, July 6, 1901.

"Etiology of Yellow Fever—Supplemental Note," *American Medical Journal*, Feb. 22, 1902.

A series of articles by Noguchi, describing *Leptospira icteroides*, appeared in the *Journal of Experimental Medicine*, June 1, 1919, Vol. 30, No. 5, and succeeding numbers.

DENGUE

Dengue is an acute insect-borne fever due to a filtrable virus. The disease is characterized by sudden onset, initial erythema, headache, and pains in the trunk and limbs, especially the joints. The pain and stiffness of the joints causes a dandified gait; hence, the name dengue. The fever is of short duration and shows a saddleback curve. The pulse is slow, there is a marked leukopenia, a terminal rash and slow convalescence. The primary rash comes during the first day or two, and may be a congestion or an erythema. The secondary rash appears about the fifth day and may be measles-like. The chief characteristics of the rashes are their lack of characteristics. In some epidemics the lymph-nodes are involved. Dengue is practically never fatal; the pathological anatomy has therefore not been disclosed.

The cause of dengue is not known. The virus is filtrable and is in the blood during the febrile stage. This has been demonstrated by injecting volunteers. Several blood parasites have been described, but none confirmed. The incubation period is usually three to six days, but quite variable. In experimental cases produced by infected mosquitoes, the incubation period ranged from four to ten days, and when produced by injection of blood, four to fifteen days.

Dengue is spread by the same mosquito that conveys yellow fever, *Aedes aegypti*. The two diseases therefore have a similar epidemiology. Dengue attacks both sexes and all ages. It is practically confined to cities, showing little tendency to spread to rural areas and villages. It is endemic in the tropics, but extends into the warm subtropical belt and occasionally into temperate regions. All who visit the tropics or subtropical countries where dengue prevails are very apt sooner or later to contract the disease.

Outbreaks of dengue occur with explosive violence. A large number are attacked in a short time. The incidence is even higher than influenza when epidemic, disabling community life. At Austin, Texas, in 1885, it is estimated that 16,000 out of 22,000 population were attacked; at Cairo, Egypt, in 1880, four-fifths of the people are said to have suffered with the disease; at Lima, Peru, in 1818, only a few persons are said to have escaped; in Galveston, Texas, in 1917, it is estimated that one-half of the population suffered, and in 1922, 60 per cent. Whole army units have been disqualified by the disease.

Dengue has the same seasonal prevalence as yellow fever. Frost brings it under control.

One attack is followed by a definite, although not absolute immunity, which as a rule lasts but a few years.

Mosquito Transmission.—Graham studied dengue in Beirut, Syria, and described a protozoön inhabiting the red blood-corpuscles and closely resembling the plasmodium of malaria except for the absence of pigment.²² Graham believed that this organism undergoes a development stage within the mosquito (*Culex fatigans*).²³ He claimed to have obtained the spores of this organism "in among the cells of the salivary glands" after forty-eight hours in mosquitoes which had bitten a dengue patient upon the fourth day of the disease. Graham produced a very severe case of fever resembling dengue by inoculating a man subcutaneously with peptonized normal salt solution containing the salivary glands of a mosquito which had bitten a dengue patient twenty-four hours before. Graham's observations concerning the parasite in the blood and in the mosquito have not been confirmed, although the subject has been studied by several experienced microscopists. Carpenter and Sutton,²⁴ however, obtained two positive results out of four experimental cases of mosquito inoculation. The period of incubation in one of these, however, was two

²² *J. Trop. M.*, 1903, 6: 209.

²³ Now *Culex quinquefasciatus*.

²⁴ *J. Am. M. Ass.*, 1905, 44: 214.

weeks, and the subjects were not sufficiently controlled to exclude the bites of other mosquitoes. Agramonte²⁵ studied an epidemic in Havana which was accompanied by a plague of *Culex fatigans*. He attempted to transmit the disease by mosquitoes, trying various species at various intervals after the insects had fed upon dengue patients, but did not succeed in producing the disease in this way. Guiteras and Finlay²⁶ endeavored to transmit the disease with *Culex pipiens*, but with negative results. Guiteras, Finlay, Agramonte, and others who have worked upon this subject state that their faith remains unshaken that the mosquito acts as the vector of dengue, despite the negative results of their experiments.

Graham,²⁷ in 1903, tried a few experiments which seemed to show that *Culex fatigans* is able to convey the infection of dengue fever. He admits, however, that in many, perhaps in all, of his experiments, *Stegomyia fasciata*²⁸ were present amongst his mosquitoes. While he demonstrated that mosquitoes can carry the disease, the variety remained in doubt. Bancroft,²⁹ in 1905, transmitted dengue in two apparently successful cases which were 'bitten by *Stegomyia fasciata*, twelve and ten days after they had bitten dengue patients, but failed when the period was longer. Bancroft worked in an infected district, and his results are not convincing. Ashburn and Craig,³⁰ in 1907, reported one doubtful case in nine persons bitten by *Culex fatigans*, suggesting the possibility of that species being a vector of dengue. Cleland, Bradley and McDonald,³¹ in 1916, reproduced the disease in four out of seven persons on whom biting experiments were conducted with *Stegomyia fasciata* mosquitoes caught in a dengue-infected district in the surroundings of cases of the disease, and some of them known to have fed on a dengue patient on the first and second days of his illness, and then transported to a non-dengue district. The incubation period of the four cases was found to be between five and nine and one-half days. The disease did not spread from any of the above cases. Experiments with *Culex fatigans* were negative. The blood taken from the experimental cases when injected into further persons reproduced the disease. This admirable piece of work in Australia clearly demonstrated the main features of the disease and its mode of transmission, and made preventive measures precise and profitable.

Siler, Hall and Hitchens,³² in a series of careful experiments on sixty-four volunteers in the Philippines, confirmed the fact that *Aedes aegypti* transmits the infection, but failed to get positive results with *Culex quinquefasciatus*. They found the extrinsic period of incubation in the mosquito to be from eleven to fourteen days. The period of incubation of the disease in

²⁵ N. York M. J., 1906, 84.

²⁶ Rev. de med. trop., 1906, 7: 53.

²⁷ J. Trop. M., 1903, 6: 209.

²⁸ Now *Aedes aegypti*.

²⁹ Austral. Med. Gaz., Jan., 1906, p. 17.

³⁰ Philippine J. Sc., 1907, 2: 93; also Craig, J. Am. M. Ass., 1920, 75: 1171.

³¹ Med. J. Australia, Sept. 2, 1916, 179; Sept. 9, 1916, 200.

³² Proc. Soc. Exper. Biol. & Med., 1925, 23: 197.

man was usually four to six days, though sometimes it stretched to ten days. The blood was infective during the first three days of the disease, and one of the mosquitoes maintained its infectivity for seventy-five days after drinking the dengue blood. The virus was not transmitted hereditarily through the eggs of the mosquito.

Prevention.—All preventive measures rest upon the demonstration that dengue is a mosquito-borne infection. Experiments with volunteers and also observations of the disease on board ships and in screened hospitals make it clear that it is not transmitted by direct contact. An instance showing the non-contagiousness of dengue is given by Persons, U. S. N.: A squad of marines from the U. S. S. *Baltimore* were given shore leave at Cavite. Twenty of the twenty-four marines who had been ashore came down with the disease after returning to the ship, while there was a total absence of infection among those who had remained aboard. Observations made at the Naval Hospital at Canacao demonstrated that in the mosquito-free wards the disease did not spread, whereas when the hospital was located at Cavite it was noted that practically every case admitted became infected with dengue while under treatment for the original complaint (Stitt).

The prevention of dengue is a counterpart of that of yellow fever. It depends upon the control of the *aedes* mosquito. Other mosquitoes, especially *Culex quinquefasciatus*, have been accused of transmitting the disease, but this lacks confirmation. Communities can make themselves fireproof against both dengue and yellow fever by substituting a closed water system for household containers. Where this is not practical, these mosquitoes can be suppressed in any community by covering and screening containers, placing fish in them, periodically emptying, or oiling them (see page 281). Screening as an accessory measure is quite imperfect in the case of dengue on account of the obvious difficulty of locating the cases.

A complete bibliography, as well as an admirable summary of our knowledge of the disease, is given by Armstrong in his article "Dengue Fever," United States Public Health Reports, 1923, 38:1750.

FILARIASIS

The filaria is a long, slender filiform threadworm with a curved or spiral tail. The adult worms live in the lymphatics, connective tissue, and body cavities. The embryos or larvæ are found in great numbers in the blood. In several species of which the life history is known mosquitoes act as the intermediate host. The most important filariæ of man are: (1) *Filaria bancrofti*, the larva of which is known as *Filaria nocturna*, appearing in the blood at night and occurring in all tropical lands, including America; (2) *Filaria loa*, the larva of which is known as *Microfilaria diurna*, occurring in the blood by day and prevalent in West Africa and India; (3) *Filaria perstans*, the larva of which is known as *Microfilaria perstans*, which persists in the blood both day and night, and occurs especially in West Africa and also in a number

of other places. None of these larvæ in the blood do any appreciable injury; only one of the adult worms, namely *Filaria bancrofti*, can be viewed as serious, causing elephantiasis, chyluria, etc., while the second species, *Filaria loa*, is more or less troublesome. According to Manson, we are hardly justified at present in assuming that all the other species are entirely without effect upon their hosts. These parasites infest man throughout the tropical and sub-tropical belt.

Filaria bancrofti (Cobbold, 1877), known in the medical books as *Filaria sanguinis hominis*, is the most harmful of the filarial worms. It is a common infection in South China, the West Indies and in the Pacific Islands, especially Samoa. In some of the South Sea Islands 50 per cent, and in the Congo-Cameroon about 75 per cent are infested. In the United States the infection is not very prevalent except in Charleston, South Carolina,³³ which is an endemic center, the infection having been brought in by a boatload of slaves many years ago.

The sexes are usually found together and lodge in the lymph vessels and lymph glands, forming cyst-like dilatations. The adult worms and their eggs block the flow of lymph, causing elephantiasis, varicose groin glands, chyluria, and lymph scrotum, and occasionally chylocele or lymph abscesses. The worms and their larvæ are often present without causing symptoms. The disease is puzzling.

This parasite is transferred by several mosquitoes,—*Culex quinquefasciatus*, *Aedes variegatus* and *Aedes albopictus* are the most important vectors. Complete development of the worm has also been observed in *Anopheles rossii*, *A. ludlowi* and *A. costalis*. Man is the intermediate host harboring the sexual phase; the mosquito is the definitive host. It takes about three weeks for the larva to complete its circle of development in the mosquito, and it comes out through the labium of the proboscis when the mosquito bites. It was formerly supposed that the larvæ enter man through the puncture by the biting parts of the mosquito, but Bahr has shown by experiments that they can pierce the intact skin just as do hookworm larvæ.

Filaria loa (Cobbold, 1864) is called the eye worm in tropical West Africa. The larvæ appear in the circulating blood during the day. The adult worms move about in the subcutaneous tissues, and are believed to be the cause of "Calabar swellings." Leiper has observed the embryos developing in the salivary glands of two species of mangrove flies (*Chrysops*).

Filaria perstans (Manson, 1891) has been so called because the larvæ persist in the circulating blood day and night. The complete life history is unknown. Both mosquitoes and ticks have been incriminated as vectors. The presence of the parasites causes no apparent symptoms.

The *prevention* of filarial diseases resolves itself fundamentally into a mosquito campaign. So far as is known, the mosquito is the only insect involved. Filariasis exists in malarial districts; in fact, the two diseases are found together. It is therefore possible "to kill two birds with one stone."

³³ *Hyg. Lab. Bull.*, No. 117, 1919.

FLIES

The true flies have but two wings, that is, they belong to the order Diptera. They comprise an enormous number of species. Contrary to popular opinion, flies are poor scavengers. Most flies prefer the sunshine, but species vary greatly in their habits and breeding places. However, surprisingly little is known of the life history and habits of most flies. The subject lacked attraction—especially the maggots or larval stage. The life history of the house

fly in general was, down to 1873, mentioned in only three European works, and few exact facts were given. A. S. Packard, then of Salem, Massachusetts, studied the house fly and gave descriptions of all its stages, showing that the growth of a generation from the egg to the adult occupies from ten to fourteen days. In 1895 Howard further traced the life history and indicated that about 120 eggs are laid by a single female at one time and that a generation is produced every ten days at the summer



FIG. 23.—HOUSE FLY (*Musca domestica*) SHOWING PROBOSCIS IN THE ACT OF EATING SUGAR.

temperatures of Washington. There may be, therefore, twelve generations in a summer. If each female lays only 120 eggs (four such batches may be laid) we have the possibility of countless millions coming from a single fly during a single season. Allowing 2,880 flies to the ounce, it has been estimated that the total product of a single fly in 40 days would equal 140 pounds, provided only one-half of them survived; hence, the logical time to begin fly suppression is in the early spring.

Flies transmit disease in one of several ways. The biting flies, such as the tsetse flies, inoculate the trypanosome of sleeping-sickness directly into the system by piercing the skin with their proboscis. Biting flies, such as *Stomoxys calcitrans*, abound in the United States in stables, houses, and also in nature. They have been implicated as

go-betweens in anthrax, relapsing fever, horse sickness (Pferdesterbe), epithelioma of fowls and other infections. Deer flies (*Chrysops*) transmit tularemia. Other blood-sucking genera, such as *Tabanus*, *Hæmatobia*, etc., are of common

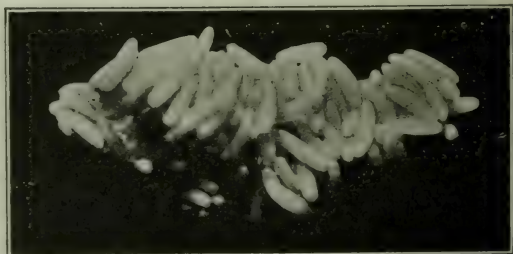


FIG. 24.—EGGS OF HOUSE FLY AS LAID IN A MASS.

occurrence, but are not known to carry any infection regularly. The common house fly does not bite. The non-biting flies are dangerous because they visit, breed or feed upon both excreta and food. There are numerous species which habitually frequent either feces or food, but not both. The house fly, however, does both. These and other non-biting flies transmit diseases by mechanical transfer of infection on their legs, mouth parts, or other body surfaces, but especially with their excreta and sometimes with their vomit. Remedies and preventive measures depend upon the peculiarities in the life history of each particular genus and species.

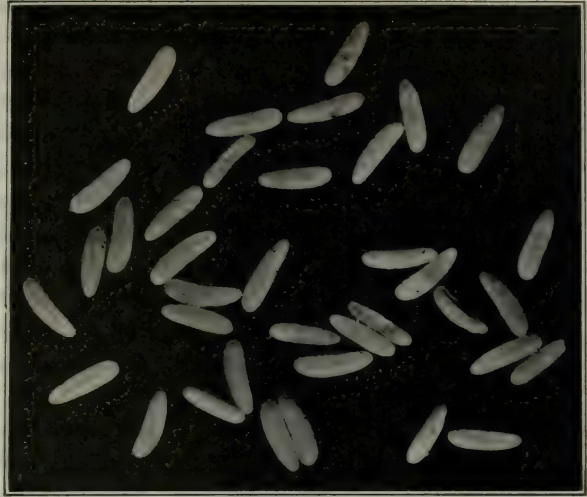


FIG. 25.—EGGS OF HOUSE FLY.

Some have hatched.

Life History of the *Musca domestica*.—The eggs of the common house fly are usually laid in masses (Fig. 24) in certain favorable spots, each mass being the result of deposition by several females. In six to eight hours the eggs

hatch into larvæ (maggots), which grow rapidly and are fully developed in four or five days. Each larva then becomes a pupa in a hard brown case—the puparium. In five days more the pupal case opens and the adult fly appears for a season of activity covering several weeks. It takes about ten days from egg to imago. Most of them die in the early

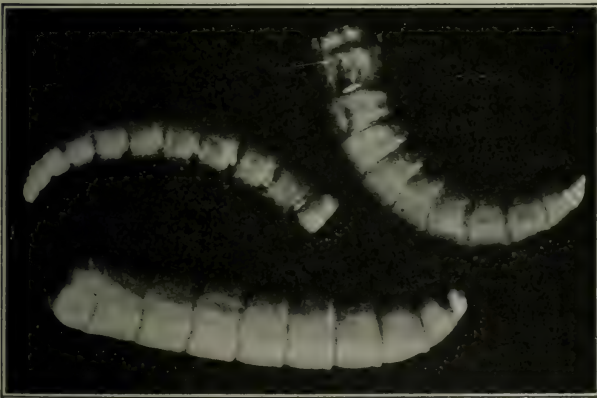


FIG. 26.—LARVÆ OF HOUSE FLY.

autumn, in great part due to a fungus disease, caused by *Empusa muscæ*, which becomes prevalent among the weakened flies at this season of the year. Those remaining out of doors are killed by the first cold nights, those which find their

way into heated buildings gradually die out, and there is no evidence that hibernation of adult flies occurs in this species. They overwinter as larvæ or pupæ in manure heaps or beneath the soil. It is possible for flies to continue breeding throughout the winter in heated locations such as animal houses and the like, where food and breeding materials are present.



FIG. 27.—PUPARIUM OF HOUSE FLY.

The chief breeding place of common house flies is in horse manure. They also have been found to breed in human excrement, fermenting vegetable and putrefying animal matter, in the bedding in poultry pens, in refuse hog hair, in tallow vats, and in garbage and organic material of various kinds—all of which means that if we allow the accumulation of filth we will have house flies.

The house fly does not breed in carcasses, nor in garbage containing much animal matter. It requires a medium with an alkaline reaction.

The larvæ of house flies have a tendency to crawl away from their breeding places; many of them burrow into the loose ground just beneath the manure piles, or crawl under boards or stones, or into dry manure collected under platforms or the like. This tendency of migrating appears three or four days before pupating. The larvæ leave the moist manure for a dry, dark place. This migrating habit is of great advantage to the winged fly, at the time of emergence, as it affords an easy path to freedom. Advantage may be taken of this migrating habit to trap many larvæ. They can be made to leave the manure if it is kept moist, and can be trapped and drowned in a box or tray partly filled with water.

Life History of *Stomoxys calcitrans*.—*Stomoxys calcitrans*, the biting stable fly, is very similar to the house fly in its life history and in appearance during the preparatory stages, but develops more slowly, requiring nearly a month to undergo a complete life cycle. The eggs are laid like those of the house fly in horse manure, but more frequently in fermenting heaps of grass, brewer's refuse ("spent hops"), etc. The adult flies are much like the house fly, but have a sharp, needle-like proboscis. They feed exclusively on mammalian blood and are a great annoyance to horses and cattle in late summer and autumn. They bite persons less frequently, but are of importance on account of their possible relation to transmitting infections.

The Flight of Flies.—Flies are strong on the wing. They travel for miles in all directions, more or less independently of the wind, although they go with the wind in greatest numbers. The strongest attraction is food; the strongest urge, to find places for oviposition. In some cases they fly eight

miles from the point of liberation in less than a single day. Our next-door neighbors, therefore, are by no means the only ones with whom we must make a fly suppression pact.

The maximum flight in the early experiments made in Cambridge, England, was 770 yards across open fenland.³⁴ It was here found that their dispersal was favored by fine weather and warm temperature. In going against the wind flies are attracted perhaps by the odors which may be conveyed. It was further observed that flies do not travel as far in towns as in open country, largely on account of food and shelter afforded by the houses.

English observers proved that house flies may come a mile from an infected dump to the nearest village, and Hodge,³⁵ investigating the abundance of flies on the



FIG. 28.—STABLE FLY.

(*Stomoxys calcitrans*.) (After Brues.)



FIG. 29.—HEAD SHOWING PROBOSCIS (*Stomoxys calcitrans*). After Brues.)

cribs in Lake Erie off Cleveland, came to the conclusion that they are blown at least six miles off shore, and that they gather on the cribs as temporary resting places. Bishopp and Laake³⁶ liberated thousands upon thousands of marked flies, especially the common house fly, *Musca domestica*, in order to ascertain the direction and speed of their travel. The maximum distance of spread from the point of release was, for the several species: *Musca domestica*, 13.1 miles; *Chrysomya macellaria*, 15.1 miles; *Phormia regina*, 10.9 miles; *Lucilia sericata*, 1.2 miles; *L. caesar*, 3.5 miles; *Synthesiomyia brasiliensis*, 0.5 mile; *Sarcophagus* spp., 3 miles; *Ophyra leukostoma*, 7 miles; *O. ænascens*, 4.1 miles.

Flies as Mechanical Carriers of Infection.—Leidy in 1864 attributed the spread of gangrene in hospitals during the Civil War to the agency of the house fly. Shortly thereafter it was discovered that the bite of the gadfly may transmit anthrax from cattle to man. Later

it was found that purulent ophthalmia of the Egyptians is carried by the house fly, and the spread of an infectious conjunctivitis known as "pink-eye"

³⁴ Local Gov. Bd. on Pub. Health Subjects, 1913, n.s., No. 85, 20-41, by Hindley and Merriman; also *J. Hyg.*, 1914, 14: 23.

³⁵ *Science*, 1913, 38: 512.

³⁶ *J. Agric. Research*, 1921, 21: 729.

in the South has been shown by Hubbard to be facilitated by little midges of the genus *Hippelates*.

Typhoid fever and other intestinal infections may be transmitted by the common house fly. Celli in 1888 fed flies with pure cultures of typhoid, tuberculosis and anthrax bacilli, and showed that the virulent bacilli were passed in the dejecta. Kober in 1892 was one of the first to call special attention to the danger of contaminating food supplies by flies coming from the excreta of typhoid patients. The United States Army Commission—Reed, Vaughan, and Shakespeare³⁷—studied the presence of typhoid fever in our camps during the Spanish-American War in the summer of 1898. They concluded that flies undoubtedly serve as carriers of the infection. "Flies swarm over infected fecal matter in the pits and then deposit it and feed upon the food prepared for the soldiers at the mess tents. In some instances, where lime had recently been sprinkled over the contents of the pits, flies with their feet whitened with lime were seen walking over the food."³⁸ Vaughan subsequently stated that he considered that about 15 per cent of the cases of typhoid in the camps were caused by fly transmission.

Alice Hamilton³⁹ isolated typhoid bacilli from five out of eighteen house flies captured in Chicago in the privies and fence near a sick room during a local water-borne outbreak. It has been shown experimentally that living typhoid bacilli may remain in or upon the bodies of flies a long time.

Ficker⁴⁰ isolated *B. typhosus* from flies from a house in which there were eight cases of typhoid fever. He further proved by experimentation that flies fed on typhoid bacilli transmitted the microorganism twenty-three days after feeding. Klein,⁴¹ Faichnie,⁴² Graham Smith,⁴³ Bertarelli,⁴⁴ and others also isolated *B. typhosus* from flies kept in nature, but in proximity to the infection. Howard studied fly abundance in relation to the origin and prevalence of typhoid fever in the District of Columbia in the summer of 1908.⁴⁵ No particular correlation between the prevalence of the flies and the prevalence of the disease could be made out. See also page 114.

Flies undoubtedly spread the infection of typhoid fever, but the importance of the rôle they play in this regard varies considerably with circumstances. In camps, unsewered towns, and overcrowded places in poor sanitary condition the danger from flies may be considerable, but even under the worst conditions it is doubtful whether flies ever play the major rôle or are responsible for the bulk of typhoid fever, as has been stated. In a well-sewered city, such as Washington, we concluded that the flies are probably responsible

³⁷ *Report on Origin and Spread of Typhoid Fever in United States Military Camps in Spanish War of 1898, 1904.*

³⁸ *Am. J. M. Sc.*, 1899, 118: 10.

³⁹ *J. Am. M. Ass.*, 1903, 40: 576.

⁴⁰ *Arch. f. Hyg.*, 1903, 46: 275.

⁴¹ *Brit. M. J.*, 1908, 2: 1150.

⁴² *J. Roy. Army Med. Corps*, 1909, 13: 672.

⁴³ *Rep. Local Govt. Bd., Lond.*, 1910, No. 40.

⁴⁴ *Cent. f. Bakt.*, 1910, 53: 486.

⁴⁵ *U. S. Hyg. Lab. Bull.*, No. 52.

but for an occasional case of the disease. It is very difficult in any particular instance to know quantitatively just how much of the infection is conveyed by flies and how much by contacts. The danger of flies is great enough without the need of exaggeration, and their suppression fully justifies the best energies of the health officer. It is perhaps a mistake to call the common house fly the "typhoid fly," not alone for the reason that the disease is spread in many other ways, but for the reason that the fly is responsible for the spread of many infections other than typhoid fever. Flies undoubtedly play the same rôle in dysentery, cholera, and all other intestinal infections that they do in typhoid fever. Tizzoni and Cattani in 1896 demonstrated active cholera organisms in the dejecta of flies caught in the cholera colonies of Bologna, Italy. These observations were subsequently verified and extended by Simonds, Offelman, McRae, and others.

It is quite evident that flies lighting upon a case of smallpox, or other infections of the skin may very readily transmit the disease to another person. I have actually seen maggots breeding in the open lesions of a case of smallpox treated in huts at Eagle Pass, Texas.

Flies may transmit the infection of erysipelas, anthrax, glanders, and skin infections. It is known that flies may ingest tuberculous sputum and excrete tubercle bacilli which may remain virulent as long as fifteen days. Flies have also been associated with leprosy and many other diseases.

Flies and Bacteria.—Esten and Mason⁴⁶ counted the bacterial population of 415 flies and found that the number of bacteria on a single fly may range all the way from 550 to 6,600,000. Early in the fly season the numbers of bacteria on flies are comparatively small, while later the numbers are very large. The places where flies live also determine largely the number of bacteria they carry. The average of the 415 flies was about one and one-quarter million bacteria. The method of the experiment was to introduce the flies into a sterile bottle and pour into the bottle a known quantity of sterilized water, then shake the bottle to wash the bacteria from the body of the fly. The numbers, therefore, only represent those washed off the outside and not those in the intestinal tract. The experiments of Esten and Mason were designed to simulate the number of microorganisms that would come from a fly falling into milk.

Torrey⁴⁷ found that a single fly may carry from 570 to 4,400,000 bacteria upon its surface, and from 16,000 to 28,000,000 in its intestinal tract. The streptococci in the flies were the prevailing types found in the breeding and feeding places of the house fly. Torrey also obtained three cultures of *B. paratyphosus* which is especially significant.

Bacot,⁴⁸ however, has shown that certain species of bacilli ingested during the larval period of *Musca domestica* can retain their existence while their host is undergoing the process of metamorphosis, and continue their existence

⁴⁶ *Storr's Agricultural Experiment Station*, Bull. No. 51, April, 1908.

⁴⁷ *J. Am. M. Ass.*, 1912, 58: 1445.

⁴⁸ *Tr. Ent. Soc., Lond.*, 1911, Pt. II, 497.

in the gut of the adult fly, but that their number diminishes suddenly after emergence. In a subsequent work Bacot⁴⁹ demonstrated that *Bacillus pyocyaneus* may thus survive. Faichnie⁵⁰ shows how *B. typhosus* may also persist. Ledingham confirms these conclusions, and states that he has recently isolated *B. typhosus* from pupæ, and the larvæ of which have fed on this organism. Glaser⁵¹ found that house fly larvæ on pupating enclose a considerable number of bacteria within them. These bacteria persist through metamorphosis and "pass on" to the adult intestine. The adult house fly (*M. domestica*) is a veritable reservoir of bacteria, whether of recently emerged flies that have not fed, or of wild adults of unknown age.

Graham-Smith⁵² recovered *B. anthracis* from blow flies bred from larvæ fed on meat infected with the organism, but failed to recover *B. typhosus* and *B. enteritidis*.

Diseases Transmitted by Flies and the Methods of Spread.—Flies transmit the virus of disease mechanically and in a great variety of ways. The virus may be carried upon their proboscis or on the surface of their bodies, but it is now believed they usually transfer infection through their dejecta. Flies act as vectors of disease, both by conveying the infection to the lips, nostrils, or wounds, or indirectly by soiling fingers, food and other objects. Biting flies may transfer infection very much as can be done by a hypodermic needle. Some biting flies, as the tsetse fly, are able to convey trypanosomes biologically rather than mechanically, because the parasite is believed to undergo a cycle of development within the insect.

Faichnie⁵³ contends that it is only the excrement of fecal bred flies that can cause enteric fever or bacillary dysentery to any great extent. The flies become infected in their larval stage; therefore, the place where they bred is a matter of far greater importance than the food they feed on. The most hazardous source of fecal bred flies is the night-soil pit.

Root⁵⁴ found that *Endamæba histolytica* and other intestinal protozoa may encyst in flies, and that these encysting forms may survive as long as two days. If, however, a fly containing cysts is drowned in water, milk, soup or other liquid food, the cysts will live still longer—about a week. Buxton⁵⁵ found that more than 4 per cent of flies examined in lower Mesopotamia actually carry human entozoa, and probably at least 0.5 per cent carry the cysts of *Endamæba histolytica*. He believes that the fly in that country is a major factor in the carriage of the bowel disorders which are there very numerous; in fact, he states the egg of any human intestinal worm, or the cyst of any protozoan may be found in the fly if only one looks long enough.

Suppression.—The suppression of the common house fly may be accomplished by striking at its breeding places. In a city this does not present very great difficulty. It resolves itself simply into a matter of cleanliness—organic

⁴⁹ *Parasitol.*, 1911, 4: 68.

⁵¹ *Am. J. Hyg.*, 1923, 3: 469.

⁵³ *So. African Med. Rec.*, 1921, 19: 438.

⁵⁵ *Brit. M. J.*, 1920, 142.

⁵⁰ *J. Roy. Army Med. Corps*, 1909, 13.

⁵² *Rep. Local Govt. Bd.*, n.s., No. 53, 1911.

⁵⁴ *Am. J. Hyg.*, 1921, 50: 131.

cleanliness of our environment. The chief breeding place is in horse manure. This should be given first attention. One neglected stable will furnish a plague of flies for an entire neighborhood. Their suppression in a well-ordered city fortunately is neither expensive nor difficult, but it requires well-trained and capable inspectors with sufficient authority to enforce the regulations. The suppression of flies by voluntary effort through the slow process of education cannot be relied upon.

There has been a notable reduction of the fly nuisance in cities since automobiles have largely replaced horses. In cities, stable manure should be placed in properly covered receptacles and removed at least once a week. This one measure obviates the use of kerosene, chlorid of lime, Paris green, or arsenate of lead, all of which are expensive and uncertain unless used frequently and in liberal amounts; further, they may decrease the fertilizing value of manure.

Garbage should be kept in water-tight cans with good covers and removed frequently, especially in the warm weather. Refuse on city lots, in back yards, in alleys, about wharves, markets, and similar places should be regularly taken away. Householders, provision merchants, storekeepers, and others should be held responsible for the cleanliness and tidiness of their premises, and those who violate these simple and primitive hygienic requirements should have their places cleaned up for them at their own expense.

Fly traps are only supplementary to radical methods of control which strike at the breeding places. Traps work most efficiently where there is no manure, kitchen refuse or other attractions to compete with the bait. The number of flies caught in traps serves as an index of the effectiveness of campaigns against breeding places. Fly trapping should begin early in the spring if it is to be of greatest value. Practically all fly traps are based on the same general principle of easy entrance and difficult exit. Their construction and operation are clearly explained in *Farmers' Bulletin No. 734*, United States Department of Agriculture, 1925.

Manure should be promptly removed; it may be kept covered in a dark place, which discourages the visitation and breeding of flies, and in addition should be carefully screened. Larvæ may be destroyed. The best results are obtained by the use of borax (sodium borate) and calcium colemanite (crude calcium borate).⁵⁶ Both substances possess a marked larvicidal action, but ap-

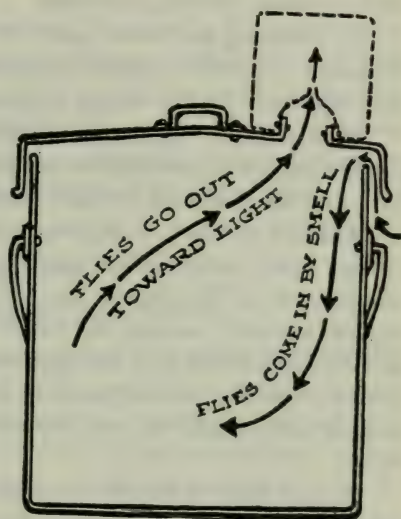


FIG. 30.—THE HODGE FLY TRAP ON A GARBAGE CAN.

⁵⁶ Bull. U. S. Dept. Agric., No. 118, 1914.

pear to exert no permanent injury on bacteria. In order to kill fly eggs and maggots, apply 0.62 pound borax or 0.75 pound calcined colemanite to every ten cubic feet (eight bushels) of manure immediately on its removal from the barn. Apply the borax particularly around the outer edges of the pile with a flour sieve or any fine sieve and sprinkle two or three gallons of water over the borax treated mass. As the maggots congregate at the outer edges of the pile, most of the borax should be applied there. The treatment should be repeated with each addition of fresh manure. Borax may also be applied to floors and crevices in barns, markets, stables, etc., as well as to street sweepings. The borax does not appear to injure the fertilizing value of the manure if it is applied carefully, and not in excess of 0.62 pound per ten cubic feet.

In Nanking and other parts of China, sodium cyanid has controlled the nuisance. About six grams in solution is added weekly to each "kong" which is a receptacle for the family excreta.

Unsatisfactory results are obtained with the use of kerosene, iron sulphate, copper sulphate, lime sulphur mixture, Paris green, sodium fluorid, formaldehyd, and the Isthmian Canal Commission larvicide.

Another method of suppression which has been found quite effective in certain army camps is the simultaneous application of three measures: first, the removal of the manure as early in the morning as possible, before it has become infested; second, the removal of the manure to a considerable distance and building it up as a compact heap in which the pressure and heat tend to prevent breeding, except possibly at the outer edges, but even these will soon become unfavorable in very dry weather; third, the use of fly traps kept well baited.

Manure may be buried or saturated with oil and burned. An efficient and simple method of handling the manure problem is in successful operation at Panama. This consists of concrete pits, six feet deep, into which the manure is dumped from day to day, and each accession promptly covered with a layer of clay and the clay coated with crude oil. Thus tightly sealed, fermentation kills the eggs, larvæ and pupæ of the flies, and effectually prevents reinfestation. The manure is kept fifteen days to three months in these pits and then sold to farmers. When, however, the pit is opened, its contents must be removed and spread on the fields at once, else it will again prove a breeding place.

Flies are thirsty insects and will be attracted to a saucer of water containing a little formaldehyd solution (1.25 to 2.5 per cent). Sodium salicylate (1 per cent) is a muscicide of about equal efficiency.⁵⁷ This simple measure will kill many of them in a room. The salts of barium, cobalt, and other poisons, such as arsenic, potassium bichromate, or quassia infusion, may be used instead of formalin, and are better bait if sweetened. Sticky fly-paper,⁵⁸

⁵⁷ *U. S. Hyg. Lab. Bull.*, No. 108, 1916.

⁵⁸ A satisfactory adhesive preparation may be made by dissolving, by the aid of heat, sixty-five parts of colophony resin in thirty-five parts of castor oil.

fly traps, electric fans, and other well-known measures will help dispose of a certain number of flies, but all these measures are tentative, and attack the problem at the wrong end.

The house fly has a number of natural enemies: various fungi, especially one belonging to the *Entomophthorææ*, which destroys flies in the autumn. Flies also harbor protozoa and nematodes as parasites, which, however, seem to do them little harm. The little bright red objects often seen attached to flies are mites, which are usually only temporary ectoparasites stealing a free ride. When spider webs are not disturbed they catch, and the spiders devour, a large number of flies. The house centipede (*Scutigera*) also sometimes catches and eats flies, as do the common garden toad, some lizards, and a few insectivorous birds.

Flies and similar dipterous insects are responsible for the transmission of a large number of diseases, most of which are discussed elsewhere. It now remains to consider sleeping-sickness, transmitted by the tsetse flies (*Glossina palpalis* and *G. morsitans*), pappataci fever, transmitted by a biting dipterous insect (*Phlebotomus papatasi*), and tularemia (*Crysops*). For convenience a general consideration of the trypanosomes is inserted in this chapter.

SLEEPING-SICKNESS ⁵⁹

(Trypanosomiasis—The Trypanosome Diseases)

The trypanosomes, next to the malarial parasites, are man's most deadly enemies among the protozoa. Two species, *Trypanosoma gambiense* and *Trypanosoma rhodesiense*, cause the terrible scourge of sleeping-sickness, one of the blackest clouds overhanging the civilization of tropical Africa. Both are transmitted by tsetse flies; *T. gambiense* by *Glossina palpalis*, and *T. rhodesiense* by *Glossina morsitans*. The disease is characterized by irregular fever, glandular enlargements, weakness, wasting and slowly increasing lethargy—sleeping-sickness. It has a chronic course, seldom lasting longer than eighteen months, and usually terminating fatally. Some individuals seem to possess a resistance, if not immunity to the infection. The period of incubation is prolonged.

Up to about 1888, sleeping-sickness was confined to a limited area on the west coast of tropical Africa. Stanley, the explorer, was responsible for the introduction of the infection into Uganda and the lake regions of Central Africa, where it had hitherto been unknown. In one district in Central Africa the population was reduced from 300,000 to 100,000 in the course of seven years (1901-1908), and there are records of whole villages and islands being depopulated. The pioneer work of David Bruce, in 1894, on nagana and the tsetse fly is one of the romantic chapters in preventive medicine.

The ravages of sleeping-sickness were well known to the old slave traders,

⁵⁹ This must not be confused with encephalitis lethargica, also popularly but improperly called "sleeping-sickness."

and the presence of "lazy niggers" lying prostrate on wharves and decks with saliva drooling from their mouths, insensible to pain or emotion, was a familiar sight. Negroes with swollen glands in the neck were soon recognized as unprofitable cargo. The fatal "homesickness" of the novelist was in fact sleeping-sickness.

The Parasite.—Trypanosomes are actively wriggling protozoa, too large to get into cells, and are therefore found free in the body fluids. In sleeping-sickness they occur in the cerebrospinal fluid, the lymph glands and sometimes the blood. They are protozoa with an undulating membrane, which terminates in a free tail-like flagellum. There are a great many kinds of trypanosomes inhabiting many different animals. Those in cold-blooded animals apparently have no effect on their hosts, but the species infecting mammals almost always cause disease. Unlike many kinds of pathogenic parasites, most trypanosomes are not specific for one host. Thus, the trypanosome of sleeping-sickness infects not only man, but also monkeys, dogs, rodents, domestic animals and a large number of wild game animals in Africa.

Practically all diseases caused by trypanosomes are transmitted by biting insects, and the parasites undergo remarkable transformations, both in the vertebrate host and in the insect. There are over seventy known species, only a few of which can be distinguished on morphological grounds. They can, however, be differentiated by their pathogenic effects, by their immunological reactions and also by cultural characteristics when grown according to the method of Novy in the water of condensation of blood agar tubes, although some of the pathogenic forms, notably *T. gambiense*, defy artificial cultivation. Novy and McNeal, in 1903, accomplished the remarkable feat of growing trypanosomes in artificial culture media.⁶⁰

Trypanosoma gambiense was discovered by Dutton in 1901-2 during the first or febrile stage of sleeping-sickness, and subsequently studied by Dutton and Todd, who did not at first suspect the relation of this parasite to the disease. This was shown by Castellani in 1903. The trypanosomes are found in the cerebrospinal fluid, in the enlarged lymphatic glands, and also in the circulating blood. They are transmitted by the tsetse fly, *Glossina palpalis*. For many years this infection was limited to West African natives. It was also met with among the slaves imported into America, but has never gained a foothold here because of the absence of the insect vector. It was largely localized in Gambia, Sierra Leone and Liberia, but with the opening up of equatorial Africa, the infection has spread to the Congo basin, Uganda and Rhodesia. The disease is not confined to Negroes; whites are susceptible.

Trypanosoma rhodesiense is even more prevalent but less malignant than *T. gambiense*, and occurs especially in southern Central Africa. It is transmitted by the tsetse fly, *Glossina morsitans*.

Transmission; the Tsetse Fly.—Both the Rhodesian and Gambian types of sleeping-sickness and also many trypanosome diseases of lower animals are

⁶⁰ *Contributions to Medical Research*, Ann Arbor, Mich., 1903, 549.

transmitted by certain species of tsetse flies which act as intermediate hosts for the trypanosomes. *Glossina morsitans* has a more widespread distribution and is less easily controlled than *G. palpalis*. Transmission is not mechanical, but biological, for the trypanosomes undergo part of their life cycle in the fly.

The relation of the tsetse fly to the transmission of this disease rests upon satisfactory evidence. Dutton and Todd, as well as others, found these flies abundant wherever sleeping-sickness existed. Wherever *Glossina palpalis* is absent sleeping-sickness never spreads, as Koch observed; while, on the other hand, if a case is brought to a locality where the tsetse fly prevails, it soon spreads.

The trypanosome undergoes a complex series of changes somewhat resembling those of the malarial parasite, except that sexual reproduction has not been demonstrated. Slender forms make their way to the salivary glands where they rapidly multiply. According to Kinghorn and Yorke, the time required for *Glossina morsitans* to become infective is from eleven to twenty-five days. A temperature between 75° and 85° F. is necessary for the full development of the parasite in the fly, ending in invasion of the salivary glands. Flies seem to lose their power

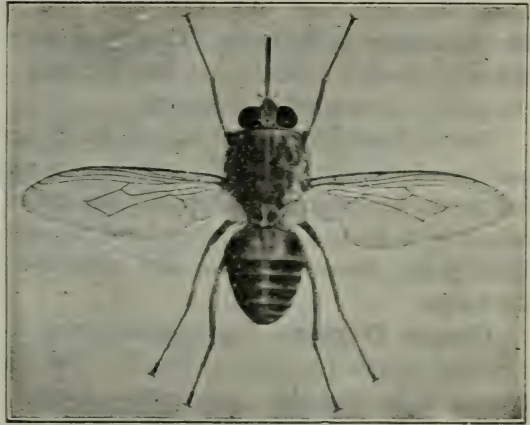


FIG. 31.—TSETSE FLY. (*Glossina palpalis*.)

of transmission soon after feeding on an infected animal, and Bruce considers it thoroughly impossible that mechanical transmission alone could explain the situation. Kleine's experiment on monkeys, confirmed by Bruce, showed that the flies may convey the disease twenty-one days after one feeding upon a monkey infected with sleeping-sickness. In another experiment by Taute, which is reported by Kleine, infection was produced on each of the first three days after feeding. From the fourth to the tenth day no infection resulted. The flies then became infective again and produced the disease from the eleventh to the forty-fourth day. Kleine⁶¹ concludes that the period of development or extrinsic period of incubation in the fly is about twenty days or a little less. Flies remain infective at least seventy-five days. Not all flies which drink blood containing trypanosomes become infective. The proportion is about one in twenty. Of the flies caught in nature in endemic areas, from two to ten in one thousand are capable of transmitting the disease to animals. Novy has emphasized, and Minchim has corroborated the fact, that tsetse

⁶¹ *Bulletin of the Sleeping-Sickness Bureau*, No. 7, 1909.

flies may harbor non-pathogenic as well as pathogenic trypanosomes, a fact which impairs the significance of a great deal of the microscopic work which has been done. As a means of avoiding the accident of dealing with naturally infected flies, it is best to use those which have been bred and raised in the laboratory.

Next to the mosquitoes, the tsetse flies are the most dangerous of the biting flies. The history and destiny of the African continent has been and will be largely controlled by these insects. They are about the size of our common house fly, although somewhat more elongated, and dark brown or yellowish in color. There are fifteen species of glossina. They have a proboscis not unlike that of a mosquito, containing a needle-like puncturing stilette, and a delicate tube in the hypopharynx for carrying the salivary secretions. Tsetse flies are diurnal in habit and are not evenly distributed, but are limited locally to fly belts. They feed mainly on the blood of both warm- and cold-blooded animals, but flies fed only on cold-blooded animals, such as crocodiles, never produce offspring.

Tsetse flies differ from all others in their family in their remarkable manner of reproduction. They do not lay eggs, but the single larva becomes full grown and occupies practically the entire swollen abdomen of the mother before it is born. As soon as born, another larva begins its development. Usually dry, loose soil, in shaded, protected spots, is selected for depositing the larvæ.

Chagas' Disease.—*Trypanosoma cruzi* is the cause of American trypanosomiasis, which prevails in Brazil. The disease bears the name of Carlos Chagas of the Oswaldo Cruz Institute, who studied it. This trypanosome is transmitted by a bloodthirsty bug, *Lamus (Conorhinus)*⁶² *megistus*, also called *Triatoma megista*. It is a large, vicious, biting insect that often attacks the face, and hence the disease is called barbiero fever. They infest all the houses in certain regions, and are found in great numbers in the cracks of the houses of the poor. Both the male and female convey the infection, which occurs chiefly in infants, who, in view of the habits of the insect, are particularly exposed. The disease often runs an acute course, terminating fatally within a few days, but it may become chronic and persist for years, giving rise to the cardiac or the nervous form.

Trypanosomes in Animals.—One of the first trypanosomes to be studied was *T. lewisi*, discovered by Lewis in 1878, which infests the rat without producing any appreciable harm, although the blood may be swarming with these large wriggling parasites.

T. evansi was discovered in 1880 by Griffith Evans, who for the first time showed that a trypanosome may be pathogenic. It is the cause of a very fatal

⁶² Entomologically *Conorhinus* is an hemipterous insect resembling the bedbug rather than the fly, but it is considered here for convenience.

disease of horses and cattle in India, known as surra. It is believed to be transmitted by biting flies (*Stomoxys*).

T. brucei is the cause of nagana, or the fly disease of Africa, an infection which is always fatal in horses and one from which few cattle recover. In 1895 Bruce discovered this trypanosome which bears his name, and which is transmitted by the tsetse fly, *Glossina morsitans*. All animals except man and possibly the goat seem susceptible. The big game animals of the district are carriers, having doubtless developed an immunity, but susceptible domestic animals brought into the fly belt soon succumb. The development of vast areas of Africa is impossible without the control of this disease.

T. equiperdum is the cause of a venereal disease of horses in many parts of the world. It is known as dourine, and is transmitted by coitus. The genital organs show marked edema, which is followed by anemia and paralysis.

T. equinum is the cause of "mal de caderas," a fatal disease of horses in South America.

T. dimorphon is the cause of a disease of horses in Gambia.

There are many other trypanosomes in birds, frogs, fish, reptiles, etc.

The Prevention of Sleeping-Sickness.—The prevention of sleeping-sickness, as well as other diseases due to trypanosomes, depends primarily upon the suppression of the insect vector. The extermination of the tsetse fly seems a hopeless task. The larvæ remain in the body of the mother fly until fully developed and are then dropped on moist soil, in which they burrow to undergo transformation to the adult state; therefore, clearing of the land in limited locations largely diminishes the number of flies. Clearing the brush exposes the earth to the sun, and the surface becomes dry and hard, so that flies die during the pupal period. This measure has limited possibilities, but is useful, as Shirata points out, around ports, in the neighborhood of villages, wharves, and other places.

The tsetse fly may also be fought by suppressing its food supply. It must obtain the blood of some vertebrate animal every two or three days. Koch believed that the disease may be successfully controlled by destruction of the crocodiles, a theory which later research has rendered very unlikely, for the flies feed upon other animals.

It has light-heartedly been advocated to kill off the big game because they are the reservoirs of the trypanosomes. Such a radical and inhuman procedure might well disturb nature's equilibrium and bring unknown trouble in its wake. Furthermore, there is no guarantee that killing off the varied and unique wild life of Africa would be effective, for domestic animals are quite as suitable for tsetse flies to feed upon as are wild game, and they may also subsist on small forest mammals, birds, crocodiles, etc., in the absence of other food.

The elimination of breeding places is the only feasible method for fighting tsetse flies in their early stages. Clearing away brush along fly-infested streams in the case of *Glossina palpalis*, which remains rather closely confined to patches along water courses, is the most valuable measure in connection

with their local destruction. These flies seldom go over fifty yards from such brushy borders of streams, except when following prey, in which case they may go several hundred yards. The effectiveness of this method of extermination was demonstrated by the Portuguese Sleeping-Sickness Commission on the Island of Principe, where tsetse flies were almost exterminated in a four years' campaign. In addition to clearing marshes or bodies of water, the beds of the water courses were straightened and leveled to make the clearing easier, the forests were completely cleared away on a large scale where they seemed to harbor the flies. In addition, some of the men employed in these operations wore on their backs black cloths smeared with sticky bird lime, thus being converted into active traps for capturing flies. Nearly half a million flies were thus caught, and the number captured daily gave a good index of the effectiveness of the preventive measures being used.

The reduction of *Glossina morsitans* is a much more difficult problem, since its habitats, though sharply confined to fly belts, are not so closely limited to the edge of water, and are therefore more difficult to clear. Since, however, the areas occupied are not over a few square miles at the most, complete deforestation of such areas, when near villages or highways, would even be feasible (Chandler).

Control measures also include attack upon the trypanosomes in man. Todd and Wolbach⁶³ suggest a systematic examination of the natives in the endemic area by gland palpation and gland puncture. The latter consists in withdrawing a drop of fluid from one of the enlarged lymphatic glands by means of a hypodermic syringe. The little drop of bloody fluid thus obtained is examined as a fresh preparation under the microscope for trypanosomes. By this method these investigators found at least 0.8 per cent of the population of the Gambia to harbor trypanosomes. If all the infected individuals could be collected in villages for observation, treatment, and isolation, it would do much to limit the disease.

Both the sick and the well should be protected against fly bites. The sick should be isolated in a location where tsetse flies are absent, and always in a well-screened and carefully managed hospital. It is therefore important to isolate all those who carry the infection in the early stages of the disease, whether they feel sick or not.

All persons taken to the hospital and detention station are given a thorough treatment with atoxyl (a combination of arsenious acid and anilin oil). Atoxyl is one-tenth as toxic and contains about three times as much arsenic as arsenious acid alone. The dose is from $\frac{3}{4}$ to 3 grains (0.05-0.2 grams) subcutaneously, combined with antimony in the form of tartar emetic. Tryparamide has a marked effect on *T. gambiense*. It is given intramuscularly in single doses of 0.5 to 5 gms. Good results are also reported from the secret German remedy "Bayer 205."

When it is necessary to travel through fly-infested places where sleeping-

⁶³ *Ann. Trop. M. & Parasitol.*, 1911, 5: 245.

sickness occurs, white fly-proof clothing and veils should be worn. Whenever possible, the fly belt should be passed through in the darkness of night, when the insects are inactive. Railroad trains and steamboats passing through fly belts should be protected by fly-proof screens, an expedient adopted in many parts of Africa at the present time.

TULAREMIA

Tularemia is a disease primarily of wild rabbits, ground squirrels and other rodents, and secondarily of man; caused by *Bacterium tularense*. The infection is insect-borne but man also contracts the disease by handling infected animals or carcasses, which accounts for the incidence among hunters, cooks, market men and laboratory workers.

The incubation period is from two to five days; the onset is sudden with headache, chills, body pains, vomiting and fever. An inflamed painful papule develops at the site of the infection, which soon breaks down liberating a necrotic core and leaving a small punched out ulcer with raised edges. The regional lymph-nodes become painful, swollen and often suppurate. The picture is that of an acute lymphadenitis.

The fever lasts two or three weeks and may reach 104° F. with a transient remission on the third or fourth day, or daily remissions suggesting a septic type of fever. Convalescence is slow and drags along with weakness for several months, sometimes a year. There are no sequelæ and fatal cases in man are rare. Tularemia in laboratory workers resembles typhoid fever, for there is no local reaction at the site of entrance and no glandular enlargement. One attack in man leaves an immunity.

So far as is now known, the disease is confined to the United States and Japan. Human cases and rodent infection have been recognized in California, Utah, Wyoming, Colorado, Idaho, Ohio, Indiana, the District of Columbia, North Carolina, Tennessee, Montana, New Mexico, Mississippi, Virginia, West Virginia, Texas, Arizona, Oregon, Kansas, Iowa, Kentucky, Pennsylvania, Missouri, Georgia, Arkansas, Maryland and Japan. The last twelve states and Japan have been added to the list since March, 1925, indicating that with the dissemination of knowledge of the disease physicians are coming to recognize a condition which has doubtless existed in their midst for years.

History and Synonyms.—McCoy first described the disease in 1911 as a "plague-like disease of rodents" in California, Tulare County; hence the name *tularense*. McCoy and Chapin in 1912 discovered the causative organism, *Bacterium tularense*. Pearse of Utah in 1911 described six human cases under the heading of "insect bites." Vail, Wherry and Lamb of Cincinnati in 1914 described a human case as "*Bacillus tularense* infection of the eye." Francis⁶⁴ in 1919 and 1920 described "deer-fly fever" in Utah and named it "tularemia."

⁶⁴J. Am. M. Ass., 1925, 84: 1243. This article reviews the disease very fully and contains a bibliography which is complete except for the pathology.

Market men call the disease "rabbit fever." The phrase "glandular type of tick fever" was used by Lamb of Idaho in 1923 in designating cases.

Bacterium tularense is a small organism occurring in coccoidal, bacillary and bipolar forms; it is Gram-negative, aërobic, without spores and non-motile; it grows only on coagulated egg yolk or glucose cystine agar. In smears, it stains well with anilin gentian violet and in sections it stains best with Giemsa solution. In three of eight attempts, it passed through Berkefeld filters which held back a small staphylococcus.

The organism is killed at 56° to 58° C. in ten minutes. Formaldehyd solution, cresol and the usual germicides are effective. The virus resisted drying in bedbug feces for twenty-five days. Glycerin preserves its virulence for at least eight months at -14° C. Refrigerated rabbits are infective after three but not after four weeks.

The history of the case and the symptoms may suggest tularemia, but the diagnosis depends upon agglutinins and cultures of *Bacterium tularense*. Specific agglutinins appear in the blood in the second week and remain for a long time—even several years. Material from the site of infection or from the enlarged glands put into guinea-pigs causes their death within a week with caseation of the lymph glands and small areas of focal necrosis studded over the liver and spleen, and *Bacterium tularense* can be recovered in pure culture on coagulated egg yolk or glucose cystine agar.

Animal Reservoir.—The only animals found infected in nature are the ground squirrels of California and Utah and the jack rabbits, snowshoe rabbits and cottontail rabbits of several states, but not domestic rabbits raised in rabbitries. These wild rodents, which constitute the great reservoir of infection manifest, when dying, the characteristic bacteremia, thus affording the necessary condition for ready transfer by blood-sucking insects and ticks.

Modes of Transmission.—*Insect Transmission.*—Transmission from rabbit in nature is by the wood tick, *Dermacentor andersoni*, the rabbit tick, *Hæmaphysalis leporis-palustris* Packard, and the rabbit louse, *Hæmodipus ventricosus*; they keep the infection alive in rabbits throughout the year.

Transmission to man is (1) by the bloodsucking fly, *Chrysops discalis* (deer-fly), commonly found on horses in Utah and in the adjoining states during the months of May, June and July; this fly, after biting an infected rabbit, bites man on an exposed part of the body; (2) by the wood-tick, *Dermacentor andersoni* Stiles, commonly found in Montana and in the adjoining states; this tick feeds on various rodents, domestic animals and on man, and is active during March, April, May and June.

Under laboratory conditions, transmission has been effected among white mice by the mouse louse, *Polyplax serratus*, and by the common bedbug, *Cimex lectularius*; from guinea-pig to guinea-pig by the biting stable fly, *Stomoxys calcitrans*; from ground squirrel to ground squirrel by the squirrel flea, *Ceratophyllus acutus*.

Tick Infections.—Ticks of the species *Dermacentor andersoni* harbor the

infection in their feces, in their circulatory fluid and in the epithelial cells of their guts. The virus is capable not only of surviving the winter in this tick and of being transmitted from stage to stage as the tick develops, but it is transmitted through the egg to the next generation of ticks. When feeding, this tick remains attached to its host for days, during which time infection most probably takes place by the introduction of its feces into the biting wound. The mere handling of a tick may result in an infection: thus, a man pulled a tick from his horse and then removed a foreign body which had blown into his eye; forty-eight hours later the eye became inflamed and he developed a typical case of tularemia.

Fly Infections.—The horsefly, *Chrysops discalis*, in contrast to the tick, remains infective for at most only four days and feeds several times a day, but only for a few minutes at a time. Infection remains confined to his mouth parts and transmission is purely mechanical.

Laboratory Infections.—Man is very susceptible to infection through the lodgment on his hands of the blood and tissues of animals upon which he performs necropsy. In five laboratories the entire personnel so engaged (eighteen in number) contracted the disease. In these cases the infection enters through the skin.

Cutaneous Infection.—The disease can be propagated indefinitely in the laboratory from rodent to rodent through the skin. Infection readily takes place through the unshaven, unabraded and unrubbed skin of a guinea-pig when infectious material is very gently applied; this probably explains the method of infection of laboratory workers.

Presence of an Antecedent Abrasion.—The presence of an abrasion of the skin, either preëxistent or coincident with the time and site of infection, was recalled by only twenty-one of eighty-seven cases which received their infection from dressing or cutting up rabbits; these abrasions consisted of cuts, punctures or scratches by fragment of shattered rabbit bone, knife, splinter of wood, nail, barbed wire, briar or burr. In twenty-three cases that were either fly-bitten, tick-bitten or bitten by coyote or ground squirrel, the bite constituted the abrasion of the skin.

Absence of Antecedent Abrasion.—No antecedent abrasion of conjunctiva was recalled by sixteen cases of primary infection of the conjunctiva or of the skin by fifty cases of primary infection of the skin, all of whom had derived their infection from dressing or cutting up wild rabbits. None of the seventeen cases of laboratory infection manifested an apparent site of infection—much less an abrasion.

In the last analysis, no one really knows even at the present moment whether he has an abrasion of the skin; much less does he know the condition three or four days ago.

Market Infections.—During the months of November, December and January, the "open season" for wild rabbits, these rodents are offered for sale in the markets in large numbers. Of a total of 914 rabbit livers examined in the Washington, D. C., market, seven were found to contain virulent *Bacterium*

tularensis. Seventeen human cases of tularemia were traced to dressing rabbits sold in that market.

Eye Infections.—Seventeen authentic instances are recorded in which the primary seat of infection was the eye, into which the infectious material had been mechanically transferred from rabbits or ticks.

Transmission by Feeding.—Guinea-pigs, rabbits, white mice, opossums, coyotes and, to a less extent, cats and rats, become infected after eating food which has been artificially contaminated with infected animal tissue. White mice readily eat bedbugs and constantly become infected after eating infected bedbugs; mice eat each other and become infected after eating an infected mouse.

Nasal Secretions, Urine and Feces.—Rabbit urine and nasal secretions cause tularemia by subcutaneous injection but not by feeding; mouse urine acts in the same way; tick feces and bedbug feces are very infectious by inoculation into guinea-pigs.

Summary.—Case histories and notes have been recorded for 145 authentic cases, of which forty-five had dressed market rabbits; twenty-three had dressed rabbits which they had shot; nineteen had cut up jack rabbits for dog feed, hog feed, chicken feed or fish bait; thirteen were fly-bitten; eight were tick-bitten; one had removed a foreign body from his eye while pulling ticks from his horse; one was bitten by an insect (species not determined); one was bitten by a coyote; one was bitten by a ground squirrel; eighteen were laboratory workers who had either (1) performed or assisted at necropsies of infected guinea-pigs, rabbits or white mice, or (2) had held infected living rabbits or guinea-pigs or had handled infected living ticks; and fifteen were residents of infected territory in whom the source of infection could not be determined with certainty.

Immunity.—One attack in man confers immunity. The only instance reported of a second attack in man was that of a laboratory worker who developed a local lesion and slight lymphadenitis, but no constitutional manifestations. Susceptible animals exhibit no evidence of immunity; they all die. In nature, dead rabbits are seen in large numbers in an infected area.

The highly susceptible animals are man, monkeys, rabbits (jack, cottontail and snowshoe), ground squirrels (California, Utah and desert), wild mice, white mice, guinea-pigs, pocket gophers, woodchucks and opossums. The faintly susceptible animals are wild rats, white rats, cats, goats and sheep. Those non-susceptible are dogs, calves, swine, horses, pigeons and chickens.

Prevention.—Laboratory workers engaged in performing necropsy of infected animals should wear rubber gloves and observe all other precautions to avoid infection. Cooks, market men and hunters should wear rubber gloves in dressing rabbits. Eradication of jack rabbits in the West is hopeless except that as the land becomes densely populated and comes under intensive cultivation the jack rabbits are naturally forced back from the valleys and seek seclusion in the shrubbery of the foothills. Since the infection is transmitted by wood-ticks through their eggs to the next generation of ticks,

complete eradication of that reservoir of infection seems impossible. Likewise, the control of the deer fly under natural conditions is an impractical task. No preventive vaccine or curative serum has yet been perfected.

The treatment is symptomatic. Rest in bed is the most important. Those who have had the most experience with the enlarged glands do not advise excision, or even incision, until a very evident, soft, thin place appears in the skin overlying the glands.

PAPPATACI FEVER

Phlebotomus fever is an acute specific infection, probably caused by a lepto-spira and conveyed to man by a biting midge (*Phlebotomus*) usually of the species *papatassii*. Long recognized clinically, Doerr and Russ,⁶⁵ and also Doerr, Franz and Taussig described a three-day fever which is prevalent on the shores of the Mediterranean, and is also known in India, Egypt and South America; in fact, the disease is almost universal in the coastal regions in the tropical and subtropical belts of both hemispheres, including the southern United States and California.

The period of incubation is three to five days. The disease is characterized by sudden onset, fever lasting two or three days, headache and aches in the bones. Uncomplicated cases are never fatal. The disease occurs in epidemics with a high incidence rate. Pappataci fever has many clinical resemblances to influenza and also to dengue. One attack confers a definite immunity.

The infection is transmitted through the bite of a dipterous insect, *Phlebotomus papatassii*. This little gnat breeds in caves, cracks and damp cellars. The extrinsic period of incubation is one week in the gnat, which is nocturnal in habit and bites only in houses.

Prevention consists in the abolition of breeding grounds of the gnats if they can be found, destruction of the adult fly, and the use of fine mosquito netting to prevent biting.

FLEAS

Fleas are laterally flattened, wingless creatures related to the Diptera. They pass through a complete metamorphosis: egg, larva, pupa, and imago. The adult female flea deposits her eggs among the hair or fur of the host animal, but, unlike the eggs of many ectoparasites, they are not fastened to the hairs and therefore fall freely to the ground. The eggs are oval, whitish, and smooth and about half a millimeter long. The larvæ escape from the eggs in two to five days. They are able to break the egg shell by a slender process on the top of the head which disappears after the first molt. The larva is a slender, legless, cylindrical creature, whitish or yellowish in color, with a head and thirteen segments. There are a few scattered hairs or bristles on the body, and at the tip is a pair of corneous processes. At the

⁶⁵ *Schiffs u. Tropen Hyg.*, 1909, 13: 693.

front of the head is a pair of biting jaws or mandibles. The larvæ feed on almost any kind of refuse. They have been reared on the sweepings from rooms. There is always some organic matter in such dust, and this is doubtless their nourishment. In houses the larvæ usually crawl into cracks or in carpets, where they feed and grow. Those that infest wild animals probably feed

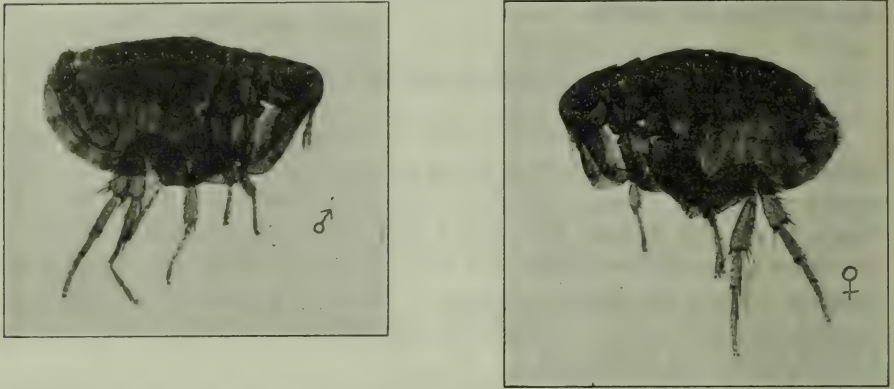


FIG. 32.—THE INDIAN RAT FLEA. (*Xenopsylla cheopis* Rothsc.)

on the refuse in the nests or retreats of these animals. It will be noticed that, unlike the mosquito, the larval and pupal stages of the flea are not aquatic. They remain in the larval stage from a week to ten days, sometimes two weeks, molting the skin three times in this interval. Then they spin flat, white, silken cocoons in which they transform to the pupal stage. In from

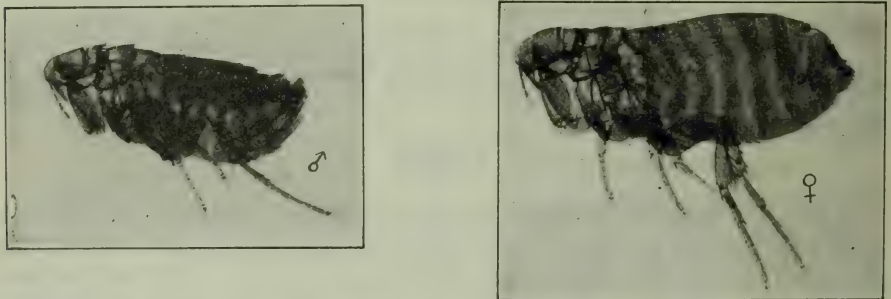


FIG. 33.—THE COMMON RAT FLEA OF EUROPE AND NORTH AMERICA. (*Ceratomyxus fasciatus* Bosc.)

five to eight days the adult flea emerges from the cocoon. The period of their transformation is affected by the temperature and moisture. In warm, damp weather a generation may develop in ten days or two weeks, but usually about eighteen days to three weeks elapse from the egg to the adult. Although some moisture is necessary for their development, an excess is apt to destroy the larvæ. Fleas are most numerous and active during warm

weather, and this doubtless accounts for the seasonal prevalence of bubonic plague and its geographic distribution.

The leaping ability of adult fleas is familiar to all. This, however, has been greatly exaggerated. The British Plague Commission determined that fleas jump three to five inches, never over six inches. No part of the leg is particularly enlarged, so that the jump is made by the entire leg as in the leaf-hoppers, and not by the femur of the hind leg, as in grasshoppers. Fleas do not vary much in size. They are mostly about two to three millimeters long. The adult insect has a hard, strongly chitinated body. The mouth parts resemble somewhat those of the mosquito. Both the male and the female flea are capable of piercing the skin to obtain blood and thus transmit infection. Fleas, as a rule, prefer certain hosts, but are not as particular in this regard as are many parasites. Those species which



FIG. 34.—THE HUMAN FLEA. (*Pulex irritans* Linn.)

are best known are found to attack several hosts, including man. This is one reason that makes them dangerous so far as plague and other infections are concerned. N. C. Rothschild, an indefatigable pioneer in the study of fleas, instead of finance for which his family is eminent, described most of the 680 known species. Formerly all fleas were classified in the single family Pulicidae, genus *Pulex*; now they are arranged in many genera and these genera grouped into families.⁶⁶ *Ctenocephalus canis* (Synonym: *Pulex serraticeps*) occurs all over the world, infesting cats and dogs, also many other animals. They are frequently brought into houses upon domestic animals, and thus become troublesome to man. *Pulex irritans* is the human flea, sometimes called the "house flea" or "common flea." It is common in Europe, but rare in most parts of the United States except on the Pacific Coast. The fleas concerned in the transmission of plague are *Xenopsylla pallida*, the India rat flea, and *Ceratophyllus fasciatus*, the common rat flea of Europe and North America. Plague may also be transmitted by *Ctenocephalus felis*,

⁶⁶ Banks, "The Rat and Its Relation to the Public Health," *Pub. Health Bull.* No. 30, *Pub. Health & Mar. Hosp. Serv.*, p. 69.

the cat flea; *Pulex irritans*, the human flea; *Ceratophyllus acutus*, the squirrel flea, and doubtless other genera and species.

Pulicides.—Adult fleas succumb to most of the general insecticides. Kerosene (coal oil) is a very efficient flea destroyer. An emulsion of petroleum and soft soap, with or without naphthalene, is a good pulicide. Formaldehyd solution, phenol, mercuric bichlorid, and tricresol in the strengths used as disinfectants are of little value in killing fleas. Powdered sulphur seems to be of no use. Mitzmain⁶⁷ has shown that water is of little value in the destruction of mature fleas. Glycerin is also practically inert as a pulicide, but tincture of green soap is very quickly effective.

Hydrocyanic acid is the most efficient of the gases because it kills both the fleas and their eggs. The amount used is two and one-half ounces of sodium cyanid per thousand cubic feet of air space (see page 544). Cyanogen chlorid is equally effective. Sulphur dioxid, three pounds per thousand cubic feet, and also carbon disulphid will kill fleas, but cannot be depended upon to destroy their eggs. Chloroform or ether first anesthetizes fleas, and if continued kills them. This is important for the safe handling of rats, squirrels, and other plague animals. The host may be chloroformed and the fleas and other ectoparasites removed with a comb. The anesthetic may be controlled by practice so that the host will recover and the fleas die, or both recover, or both die, as may be desired.

In flea-infected houses the larvæ, living in the cracks of the floor, etc., may be controlled by sprinkling a thin coating of flake naphthalene on the floor and then leaving the room tightly closed over night. In the morning the naphthalene may be swept up and what remains used again.

RELATION OF PLAGUE TO RATS AND FLEAS

Plague is primarily a disease of the rat⁶⁸ and secondarily of man. This fact is now firmly established not only by the recent experiences, but especially through the admirable studies of the Indian Plague Commission⁶⁹ which established beyond doubt the fact that plague may be and generally is transmitted from rat to rat and from rat to man through the agency of the Indian rat flea, *Xenopsylla pallida*, and sometimes by our rat flea, *Ceratophyllus fasciatus*, and also by the fleas of squirrels, cats, man, etc.

During some plague epidemics it has been noted that the rats die in great numbers before and during the outbreak. It is now known that this epizootic in the rat is true plague. In nature, rats suffer both with acute and chronic plague and there is evidence that they may serve as healthy carriers.

In the laboratory, rats may be infected with plague by ingestion, by application of the virus to mucous or cutaneous surfaces, or by subcutaneous inoculation. In nature, rats may become infected by any of these means, but

⁶⁷ U. S. Pub. Health Rep., 1910, 25: 1039.

⁶⁸ Sometimes of other rodents, as the ground squirrel, marmot, tarbagan, etc.

⁶⁹ J. Hyg., 1906, 6: No. 4; 1907, 7: Nos. 3, 6; 1908, 8: No. 2.

probably flea transmission is the only one that ordinarily operates to any extent.

Rats are great travelers, and have carried the plague to all quarters of the globe. A more complete discussion of the rat and its relation to plague and other diseases will be found on pages 334 to 350.

Within the past few years it has been discovered that, while the rat is the great medium for the spread of plague, the disease was probably preserved from extinction in Thibet by another rodent, the marmot (*Arctomys bobac*). The tarbagan, a fur-bearing rodent, infected trappers and traders, who in turn started the epidemic of pneumonic plague in Manchuria in 1910-11. In California the infection has got into the ground squirrels⁷⁰ in which the disease will doubtless be kept alive for many years to come. In South Africa, certain field rodents are the reservoir. In an isolated district in South America, the guinea-pigs became involved. To realize the full importance of these discoveries, it is only necessary to call to mind that, in order to eradicate plague, a warfare against the rat alone is not sufficient, but must include the rodents mentioned and perhaps others.

Simond in 1897 advanced the theory that plague was carried by fleas. This theory was developed by J. Ashburton Thompson and others and conclusively proved by the Indian Plague Commission. The exact method by which the flea transmits the infection from animal to animal is not always clear, although carefully studied. The mouth parts appear not to remain infected. It is improbable that the salivary secretions contain the micro-organisms. It is known that the plague bacilli may live in the digestive tract and be passed alive and virulent in the dejecta. It is easy to understand how some of the infected dejecta may be rubbed or scratched into the little wound produced by the flea bite.

Bacot and Martin⁷¹ found that *Xenopsylla pallida* and *Ceratophyllus fasciatus* may transmit plague during the act of sucking by regurgitating some of the blood. These investigators found that in a proportion of infected fleas the plague bacilli develop to such an extent in the esophagus and proventriculus as to occlude the alimentary canal at the entrance to the stomach. Fleas in this condition are not prevented from sucking blood as the pump is in the pharynx, but they only succeed in distending an already obstructed esophagus, and on cessation of the pumping act some of the blood is forced back into the wound. Such fleas are persistent in their endeavors to feed, and this renders them particularly dangerous.

When it was found that the common rat flea of Europe, *Ceratophyllus fasciatus*, does not readily bite man, doubt was thrown upon the part played by the flea in plague transmission. These negative results, however, are offset by the convincing positive proofs of the British Plague Commission in India, and by McCoy and Mitzmain in San Francisco, who showed that under certain conditions the rat flea will bite man, especially if the natural

⁷⁰ *Otospermophilus grammurus beecheyi*, syn. *Citellus beecheyi*.

⁷¹ *J. Hyg., Plague Suppl.*, 1914, 3: 423.

food supply is limited, and that these fleas may feed on a man's hand even in the presence of a rat.

Raybaud ⁷² calls attention to the fact that the rat flea (*Ceratophyllus fasciatus*) is able to hibernate for a month or forty-five days without nourishment, and that virulent plague germs may persist unharmed in its stomach during this length of time and even longer. This fact may be of importance for the transmission of plague to a distance.

Bacot and Martin ⁷³ found that infected fleas which were fed regularly might live for fifty days at from 10 to 15° C. and twenty-three days at 27° C. and remain infected at death.

The Commission for the Investigation of the Plague in India ⁷⁴ found that infection conveyed by fleas might take place three weeks after the flea population had any opportunity of imbibing infected blood. Bacot ⁷⁵ has observed



FIG. 35.—A SQUIRREL FLEA. (*Hoplopsyllus anomalus* Baker.)

that fleas (*Ceratophyllus fasciatus*) are able to carry the bacillus of plague for periods up to forty-seven days and subsequently infect a mouse. The indications thus are that plague infection may persist in fleas at least one or two months in cold weather and subsequently give rise to an epizootic.

The development of plague bacilli in the flea, as well as the activity of the flea itself, is restricted both in very hot and very cold weather. Extremes of temperature are therefore not favorable for the spread of the bubonic form of the disease. Fleas are most numerous and most active in warm weather. The pneumonic form is a contact infection of man, independent of an insect vector, and spreads best when the weather is very cold.

According to the observations of Nuttall and Yersin, flies and possibly other insects may occasionally convey the infection. Walker ⁷⁶ considers, as

⁷² *Presse méd.*, 1911, 19: 179.

⁷³ *J. Hyg.*, Plague Suppl., 1914, 3: 437.

⁷⁴ *J. Hyg.*, 1906, 6: 435.

⁷⁵ Observations on the length of time that fleas (*Ceratophyllus fasciatus*) carrying *Bacillus pestis* in their alimentary canals are able to survive in the absence of a host and retain the power to reinfect with plague, *J. Hyg.*, Plague Suppl., 1915, 4: 770.

⁷⁶ *Indian M. Gaz.*, 1910, 45: 93.

the result of experiments, that bedbugs and other biting insects play a rôle in the transmission of plague. Bacot⁷⁷ has demonstrated that bedbugs are capable of carrying plague bacilli and may thus infect mice after a period of forty-eight days' starvation.

RATS AND OTHER RODENTS

Rats, mice, squirrels, and other rodents have become a serious problem in preventive medicine, and their habits and methods of suppression may be considered conveniently at this place. Plague being primarily a disease of rats, the prevention and suppression of this infection resolve themselves into a war upon these and other rodents. There have been epizootics in the guinea-pig in Brazil and in a similar woodland rodent, *Cavia aperea*, in Argentina, coinciding with plague in man. Practically all mammals are susceptible. In southeastern Russia the camel became infected and was able to start an epidemic.⁷⁸ Fowl are immune. For the control of plague it is, therefore, necessary to have a knowledge of the life history and methods of attacking the problem in the lower animals. In addition to plague, rats are a reservoir of trichinosis. They are responsible for infectious jaundice and other diseases, and for the transmission of certain tapeworms and parasites, some of which concern man. Rats also in their wild state suffer from cancer and from leprosy, which, however, have no known connection with the human diseases. Rats and mice are carriers of *B. enteritidis*, which is connected with food infection.

Rodents comprise more than one-third of all living species of mammals, and exceed any other mammalian order in the number of individuals. They have no canine teeth, but strongly developed incisors. Only the front of the incisors is covered with enamel, which keeps them sharp and chisel-like, owing to the more rapid wearing away of the softer dentine. The incisor teeth continue to grow throughout the life of the animal. The most extensive family of rodents is the *Muridae*, which includes the true rats and mice, typified by the genus *Rattus* (syn. *Mus*). Trouessart, in his "Catalogus mammalium," enumerates 250 species of *Rattus* (*Mus*) described before 1905. Since that date a number of new forms have been added.

The genus *Rattus* is characterized by narrow, ungrooved incisors; three small-rooted molars; soft fur mixed with hairs, sometimes with spines; a rudimentary pollex (thumb) having a short nail instead of a claw; a long tail bearing rings or overlapping scales and often naked or nearly so. The ears are rather large, the eyes bright and prominent, and the muzzle somewhat pointed.

The distinction between rats and mice is largely based on size. The house mouse and the domestic rats, however, belong to different genera. Of the many species of the genus *Rattus* and the genus *Mus* only four have developed

⁷⁷ *J. Hyg.*, Plague Suppl., 1915, 4: 779.

⁷⁸ *Ann. de l'Inst. Pasteur*, 1923, 37: 618.

the ability to adapt themselves to such a variety of conditions as to become cosmopolitan. Four have found lodgment in America:

The common house mouse, *Mus musculus musculus*

The English black rat, *Rattus rattus rattus* (syn. *Mus rattus*)

The Egyptian or roof rat, *Rattus rattus alexandrinus* (syn. *Mus alexandrinus*)

The brown rat, *Rattus norvegicus* (syn. *Mus norvegicus*)

The black rat and the roof rat differ from each other mostly in color, and some zoölogists regard them as races of the same species. The brown rat is also known as the gray rat, barn rat, wharf rat, sewer rat, and Norway rat.

The black rat (*Rattus rattus rattus*) has been known in Europe since the twelfth century, and from there has been carried to America. The brown rat (*Rattus norvegicus*) came later, and, as it is more destructive, larger, and more ferocious, it is rapidly driving the black rat before it. The brown rat differs somewhat in habits from the black rat, especially in that it burrows, which protects it against its enemies and renders its suppression more difficult.

The house mouse holds its own everywhere against the brown or Norway rat, as it is able to get into holes too small for the rat to follow. Albinism and melanism occur in all species; pied forms are common. The white rat of the laboratory is an albino form of either *Rattus rattus rattus* or *Rattus norvegicus*. No other wild animal is cosmopolitan and few domestic animals have a world-wide distribution.

Breeding and Prevalence.—The brown rat is more prolific than either the roof rat or the black rat. The brown rat reproduces from three to five times a year, each time bringing forth from six to nine, and sometimes as many as twenty-two or twenty-three, young. They breed more rapidly in temperate and equable climates than in those of great variability. Lantz estimates that starting with one pair of rats breeding three times a year, with an average litter of ten and without deaths, the increase amounts to 20,155,392 rats at the end of three years. Rucker, carrying the figures to five years, obtained 940,369,969,152 rats.

The number of rats is limited only by the food supply and opportunities to nest. Few people have any conception of the enormous numbers of rats in cities and on farms. Although seldom seen in the day time, at night they fairly swarm along river fronts and wharves, as well as in sewers, stables, warehouses, markets, and other places where food may be found. A few instances will illustrate the prolific habits and give an idea of the destructive tendency of rats.

In 1901 an estate near Chichester, England, was badly infested with rats; ⁷⁹ 31,981¹ were killed by traps, poisons, and ferrets, while it is estimated that tenants, at the threshing, destroyed fully 5,000 more. Even then the property was by no means free from rats.

⁷⁹ *The Field*, London, 1902, 100: 545.

At one time Jamaica was overrun with rats to such an extent that famine threatened. The mongoose which was imported soon killed the brown rat, but the black rat was able to escape, owing to its habit of living in trees and on thatched roofs. When the rats became scarce, the mongoose attacked birds and their eggs, which caused a great increase in the insect pests, and the mongoose in turn thus became a nuisance.

During the plague of rats on the island of Jamaica, in 1833, the number killed on a single plantation in a year was 38,000.⁸⁰ The injury to sugar cane on the island caused by the animals was at that time estimated at half a million dollars a year.

The report of the Indian Famine Commission in 1881 affords one of the best illustrations of the number of rats that may infest a country. An extraordinary number of the animals at that time inhabited the Southern Deccan and Mahratta districts of India.⁸¹ The autumn crop of 1878 and the spring crop of 1879 were both below the average, and a large portion of each was destroyed by rats. The resulting scarcity of food led to the payment of rewards for the destruction of the pests, and over 12,000,000 were killed.

The average life of a rat is about two years.

Migration.—The migrations of rats have often been recorded. The brown rat is known in Europe quite generally as the migratory rat; the Germans call it the *Wanderratte*. Pallas relates that in the autumn of 1772 they arrived from the East at Astrakhan, southeastern Russia, in such great numbers and so suddenly that nothing could be done to oppose them. They crossed the Volga in immense troops.

Seasonable movements of rats from houses and barns to the open fields take place in the spring, when green and succulent plant food is ready for them. The return movement takes place in the autumn. This seasonal migration is notable even in large cities. In 1903 a multitude of migrating rats spread over several counties of western Illinois. They traveled in great armies and invaded the farms and villages of Rock Island and Mercer counties, and caused heavy losses during the winter and summer of 1904. In one month Montgomery of Mercer county killed on his farm 3,435 rats, most of which he caught in traps.

In England a general movement of rats inland from the coast occurs every October. This is known to be closely connected with the closing of the herring season. During the fishing the rodents swarm to the coast, attracted by the offal left in cleaning the herring, and when this food fails, the animals troop back to the farms and villages.

An invasion of black rats (*Rattus rattus rattus*) in the Bermuda Islands occurred about the year 1615. Within two years they had increased so alarmingly that none of the islands was free from them. The rodents "devoured everything that came in their way—fruits, plants, and even trees"—so that for a year or two the people were nearly destitute of food. A law was passed

⁸⁰ *New Eng. Farmer*, 1834, 12: 315.

⁸¹ *Brit. M. J.*, 1905, 2: 622.

requiring every man in the island to keep twelve traps. In spite of all efforts the animals continued to increase; but they finally disappeared, so suddenly that it is supposed they must have been victims of a pestilence.

While stationed upon Angel Island in San Francisco harbor I observed several migrations of rats between the army post and the quarantine station, which were about a mile apart and separated by an intervening ridge. Everyone is familiar with the sudden invasion of stores, factories, and other structures with those rodent pests, which causes considerable economic loss.

On Vessels.—Rats are found on all vessels; they are great travelers. It is through this seagoing tendency that the rat has become cosmopolitan. Rats get on board vessels readily as they lie at their dock; sometimes they are carried on board in the cargo. Modern steamers are being built so as to afford little rat harborage.

It is very important to prevent the introduction of rats on vessels at plague-infected ports; it is also important to prevent the passage of rats from ship to shore, particularly if the vessel is from a plague port. In order to accomplish this, it is necessary to exercise particular care. In extreme cases the ship should not approach the dock, but the cargo should be handled by means of lighters. When the ship lies at its moorings in a stream or in the open bay, rats may get on board by swimming, and climbing in through the hawse pipe. Rats rarely swim more than one-quarter to one-third of a mile. If the vessel ties up at the dock, inverted funnels should be placed on the hawsers. The gangplanks should be watched during the day and always taken up at night. Vessels from plague ports should always be treated with sulphur dioxid or hydrocyanic acid gas, preferably when empty, and always before leaving, and also en route, to kill the rats that may be on board. A wise measure in international sanitation would be to require all vessels, whether trading at plague ports or not, to fumigate for rats no less than three or four times a year. For destruction of rats on vessels, see page 539.

Food.—Rats are not strictly herbivorous, as might be inferred from their dentition; they are practically omnivorous. Their bill of fare includes grains and seeds of every kind; flour, meal, and all food products made from them; garden vegetables, mushrooms, bark of growing trees, bulbs, roots, stems, leaves, and flowers of herbaceous plants; eggs, chickens, ducklings, squabs, and young rabbits, milk, butter, and cheese; fresh meat and carrion; fish, frogs, mollusks, and crustaceans; they are also cannibals. This great variety of food explains the ease with which rats maintain themselves in almost any environment.

Habits.—The roof rat (*Rattus rattus alexandrinus*) and the black rat (*Rattus rattus rattus*) are more expert climbers than the brown rat, which is larger and clumsier. In buildings the brown rat keeps mainly to the cellar and lower parts, where it commonly lives in burrows. From these retreats it makes nightly excursions in search of food. The roof rat and the black rat live in the walls or in the space between ceilings and roofs. Rats readily climb trees to obtain fruit. In the tropics the roof rat and the black rat

habitually nest in trees. In the open, rats seem to have defective vision; by daylight they move slowly and uncertainly; on the contrary, at the side of the room and in contact with the wall, they run with great celerity. This fact suggests that the *vibrissæ* (whiskers) serve as feelers, and that the sense of touch in them is extremely delicate. The animals always prefer narrow places as highways—another circumstance which may be made use of in placing traps. Rats fight fiercely when cornered. They sometimes bite sleeping infants and adults (see Rat-Bite Fever, p. 334).

Economic Importance.—The destruction of food, merchandise, and property by rats is so great that this alone would justify active measures of suppression, even though they were not responsible for plague, trichinosis, and other infections. Rats destroy grain while growing; invade stores, destroy flowers, laces, silks, carpets; eat fruits, vegetables, meat, etc., in the market; destroy by pollution ten times as much as they eat; cause conflagration by dragging matches into their holes; gnaw lead pipes and floors of houses; ruin artificial ponds and embankments by burrowing; destroy eggs and young poultry; damage foundations, floors, doors, piers; in short, they have become the worst mammalian pest among us. According to Government experts, the cost of maintenance of a rat is about one-half cent a day, which makes the annual board bill of the rat population in the United States about \$182,000.000. This does not include property and other damage.

SUPPRESSION OF RATS

Rats are very intelligent and cautious. Extermination is a biological impossibility, for killing off large numbers gives the survivors an easier living. We can well be satisfied with suppression and control. Millions of rats have been killed in India, Japan, San Francisco, and other places during the recent plague measures without making an appreciable impress upon the numbers remaining. They may be exterminated and kept out of a limited area, such as a ship, a granary, a stable, a warehouse, a market, or local compound. In the well-built residential sections of a city, with concrete walks, asphalt streets, stone cellars, and few stables, there are very few rats. In ten years of residence in such a district in Washington I never saw or heard of one in the neighborhood.

The measures for the repression and destruction of rats will be considered under: (1) rat-proof buildings, (2) keeping food from rats, (3) natural enemies, (4) traps, (5) poisons, (6) domestic animals, (7) shooting, (8) fumigation, and (9) bacterial viruses.

Rat-proof Buildings.—This is a measure of first importance in the fight against rats. Rats can only gain entrance to a properly constructed cement structure through neglect or ignorance. They come in through drain pipes if left open; through doors, especially from alleys; and through basement windows. Once in, they intrench themselves in out-of-the-way places, nest behind rubbish, and are difficult to dislodge. The lower parts of the outer

doors of public structures, such as markets and wharves, should be reënforced with metal to keep the rats from gnawing through. Basement windows should be screened and doors provided with springs to keep them closed. Screens or wire cloth to keep out the rats must be not less than twenty gauge wire nor greater than one-half an inch mesh. The special points of ingress and egress of rats which must be guarded against besides basement windows and doors are hatches, ventilators, skylights, unused chimney flues, and openings around water, sewer, gas, and steam pipes, and electric wires. Screens should be placed in tunnels and other strategic places to prevent rats wandering from one place to another in large buildings and on ships.⁸²

Foundation walls should be laid without a break around the entire building and should extend not less than eighteen inches beneath the surface of the surrounding soil, and should always be flush with the under-surface of the floor above. Floor joists should be imbedded in this wall or the spaces between the joists filled in and completely closed up to the floor level. Ground areas should be concreted with a layer at least three inches in thickness, finished with a wearing surface of cement about one-half an inch thick. The walls of a wooden house should have one foot of concrete between the sheathing and lathing. All water and drain pipes should be surrounded with cement where they pierce the walls. Rat holes may be closed with a mixture of cement, sand, and broken glass, or sharp bits of crockery and stone.

Buildings may be raised from the surface of the ground on piers, thus rendering them rat-proof. Cribs for grain in the country can be so raised and further protected with metal netting.

The chief refuges for rats in cities are sewers, wharves, stables, provision houses, markets, out-buildings, slaughterhouses, restaurant kitchens, bakery shops, candy factories, and uninhabited structures. These are more important than dwellings. Modern sewers are highways and not nesting places for rats. They find a safe retreat from nearly all enemies under wooden sidewalks. In the country it is important to build corn cribs, barns, and granaries rat-proof with the liberal use of cement, iron sheeting, or galvanized iron netting.

Keeping Food from Rats.—Well-fed rats mature quickly, breed often, and have large litters. A scarcity of food helps all other suppressive measures. Garbage and offal must be disposed of so that rats cannot get at such stuff. Well-covered garbage cans should be required and the garbage frequently removed and burned. Garbage dumps only invite and nourish rats and other vermin. Slaughterhouses are centers of rat propagation. The offal is best disposed of by burning. Care should also be taken as to the disposal of remnants of lunches in office buildings and the disposal of organic waste generally. Produce in provision stores may be protected with wire cages.

Natural Enemies.—The natural enemies of the rat are the larger hawks, owls, snakes, skunks, foxes, coyotes, weasles, minks, dogs, cats, and ferrets.

⁸² See "The Rat-Proofing of Vessels," *U. S. Pub. Health Rep.*, 1925, 40: 1507.

The persistent killing off of the carnivorous birds and mammals that prey upon rats has been an important factor in the increase of the country rat in the United States. Rats actually destroy more eggs, chickens, and game than all the wild animals combined.

Traps.—Experienced trappers use spring, cage and guillotine traps; also barrel and pit traps. One of the best is the old-fashioned wire cage trap. The rats get in but cannot get out. In placing the trap it is advisable to leave a rat in as a decoy. The trap should be placed along runways, or the entrance to the trap may be arranged so that the rats first have to go through a pipe, as they like to explore narrow passages. It requires ingenuity to trap rats successfully. They are very wary and avoid man-smell. To guard against this the traps may be burned and then smeared with the bait, always handling them with tongs or properly prepared gloves. Cheese, bacon, grain, and also meat, fish heads, vegetables, or bread are the best baits. It is advisable to change the bait occasionally. Rats may be caught and held on a mixture of resin and petroleum oil, which is boiled down to the consistency of glue. This is spread about a half-inch thick on boards and placed near holes and along runways.

Poisons.—Poisons are objectionable in dwellings, owing to the odor of the dead rats, but are of service in granaries, stables, wharves, storage depots, garbage dumps and similar places where rat-proofing is difficult or too expensive. Most rat poisons are dangerous to children as well as to chickens and other domestic animals, and, therefore, the greatest care must be exercised in their use. It requires experience in laying out poisons; the old rats are very smart and will refuse the bait unless artfully concealed and judiciously placed.

The principal poisons used for rats are barium carbonate, arsenic, and phosphorus. In several states the law requires that notice of intention to lay poison must be given to persons living in the neighborhood. Poisons for rats should never be placed in open or unsheltered places. In buildings and yards occupied by poultry the following procedure is recommended: Two wooden boxes should be used, one considerably larger than the other, and each having two or more holes in the sides large enough to admit rats. The poisoned bait should be placed in the bottom and near the middle of the smaller box, and the larger box should then be inverted over the other. Rats thus have free access to the bait, but fowls are excluded.

The cheapest and most effective poison is barium carbonate. It is deadly to rats and mice, and is also poisonous to human beings, live stock and poultry, and must therefore be used with care. This may be made into a dough with four parts of meal or flour to one part of barium carbonate. A good plan is to spread the barium carbonate upon fish, on toasted bread (moistened), or upon ordinary bread and butter. In Hawaii, barium carbonate is mixed with flour dough and made into small round cakes less than an inch in diameter and about one-quarter inch thick. A coating of paraffin protects them from dampness and molding, thus insuring their effectiveness for many months. A very

small nibble at one of these poisonous cakes will kill a mouse, while a piece the size of a small pea will kill a rat.

Strychnin has no advantage over barium carbonate, and should not be used, on account of the danger of fatal accidental poisoning among young children.

Arsenic is popular; the powdered white arsenic (arsenious acid) may be used as described for barium; or a stiff dough may be made by mixing twelve parts by weight of corn meal and one part of arsenic with white of egg. An old English formula is one pound of oatmeal, one pound of brown sugar, and a spoonful of arsenic.

Phosphorus is sometimes used. The yellow phosphorus in the proportion of 1 to 4 per cent may be mixed with glucose or other suitable material. Kitano⁸³ soaks the phosphorus into bread, which is cut into pieces containing 0.025 gm. of phosphorus per piece. The use of phosphorus is very dangerous on account of fire and this poison is not recommended by most authorities. Rats poisoned with phosphorus may die on the premises and decompose, contrary to the statements sometimes made in the advertisements.

The following formula is used as a poisonous bait for rats, mice, squirrels, etc.:

Strychnin	1 ounce
Cyanid of potassium.....	2 ounces
Eggs	1 dozen
Honey	1 pint
Wheat or barley.....	30 pounds

Stir eggs well, then mix in honey and again stir. Then put in dry powdered strychnin and cyanid and stir until well mixed. Put wheat in large box or can and pour in the mixture of poison and stir until it is well distributed over the wheat. Stir two or three times during twenty-four hours, then spread out and dry. Before putting it out for squirrels add oil of rhodium, 1 dram.

Poisons and traps reduce the number of rats but do not eliminate them. Their greatest usefulness is ridding large rat-proof structures of contained rats. Keeping food from rats is one of the best methods of suppression.

Domestic Animals.—A well-trained dog may be relied upon to keep the farm premises reasonably free of rats. Small Irish, Scotch, and fox terriers make the best ratters; the ordinary cur and the larger breeds of dogs seldom develop the necessary qualities.

However valuable cats may be as mousers, few of them care to tackle rats. The ordinary house cat is too well fed and too lazy to undertake an encounter with an animal as formidable as the brown rat. Koch has advised the breeding and distribution of cats capable and willing to attack rats.

Shooting.—Many rats may be shot as they come out to forage about sundown. This method is particularly effective in a large building which is sud-

⁸³ *Am. J. Trop. Dis.*, 1916, 3: 637.

denly overrun with the rodents. The shooting of a number of them upon two or three successive nights discourages the remainder, who leave for some other happier hunting ground.

Fumigation.—Rats may be killed with certainty in any inclosed structure by the use of sulphur dioxid, carbon disulphid, hydrocyanic acid gas, or carbon monoxid. The methods of evolving these substances are described in Section IV. Hydrocyanic acid gas and sulphur dioxid are particularly useful to destroy rats on board ships, in cellars, stables, sewers, and places where they abound. Enormous numbers of rats are frequently killed when ships are fumigated with sulphur dioxid. I have seen buckets full thrown overboard from comparatively small vessels. Hobdy counted 310 on a lumber-carrying schooner of only 260 tons burden. The S.S. *Minnehaha*, a vessel only nine months in commission, fumigated in London in May, 1901, yielded a bag of 1,700 rats. See pages 539 to 547.

Bacterial Rat Viruses.—Rats are notoriously resistant to bacterial infection.⁶⁴ Even plague usually fails markedly to diminish their prevalence. An epizootic of bacterial nature, therefore, cannot be classed with the natural enemies of the rat. We are not surprised, then, to learn that the bacterial rat viruses have signally failed.

These bacterial viruses belong to the colon-typhoid group of organisms. They are either identical with or closely related to the original bacillus of mouse typhoid (*B. typhi murium*) discovered by Löffler, or the paratyphoid bacillus, type β , or the *Bacillus enteriditis* of Gärtner, which have been associated with gastro-intestinal disorders.

The claim that these rat viruses are harmless to man needs revision, in view of the instances of sickness and death reported by various observers. The pathogenicity for man depends upon the virulence of the culture, the amount ingested, the nature of the medium in which it grows, and many other factors.

Danysz virus (*B. typhi murium*) is pathogenic for rats under laboratory conditions, but has feeble powers of propagating itself from rat to rat. It rapidly loses its virulence, especially when exposed to light and air. The result depends largely upon the amount ingested.

Under natural conditions these rat viruses may be likened to a chemical poison, with the great disadvantage that they rapidly lose their virulence and are comparatively expensive. They also have the further disadvantage that chemical poisons do not possess of rendering animals immune by the ingestion of amounts that are insufficient to kill or by the ingestion of cultures that have lost their virulence.

Rat Surveys.—Rat surveys should be conducted to determine the presence or absence of rodent plague, especially at seaports. The survey should be in charge of a trained expert, who will need the assistance of trappers

⁶⁴ "The Inefficiency of Bacterial Viruses in the Extermination of Rats," M. J. Rosenau. "The Rat and Its Relation to the Public Health," Bulletin No. 30 of the P. H. & M. H. S., 1910.

and a laboratory. Where plague exists, the warfare against the rodents will require an extended personnel to include rat-proofing and all the measures known for rat suppression.

In a rat survey to determine the existence of plague the number of rats examined should be at least 1,000 for every 10,000 of the human population, and a preliminary survey should be made in order that the most promising locations shall be trapped. Experienced ratters depend upon traps, dogs, cats and ferrets, and poisoned bait in the open. Rat-proofing will keep rats out, but constant vigilance, inspection and attack upon the first sign of a rat are required.

RATS AND DISEASE

RAT-BITE FEVER

Miyake⁸⁵ has described a "rat-bite disease," called in Japan sodoku, or rat-bite fever. Rat-bite fever⁸⁶ is a rare and curious infection sometimes following a rat bite. In all only eighty cases of human infection have been reported in the literature up to 1918. The symptoms come on after the wound has healed. There is inflammation of the bitten part with swelling of the lymph glands, paroxysms of fever of the relapsing type which may last months or years, and an eruption of the skin which usually occurs during the second febrile paroxysm. The case fatality is about 10 per cent.

The cause of rat-bite fever is probably a spirochete described by Japanese investigators—*Spirochaeta morsus muris*.⁸⁷ This spirochete is found in the blood of rats and also in human cases. It is not found in the saliva of infected rats and the infection is supposed to be transmitted during the bite by blood coming from wounds of the gums. Bearing upon a spirochete as the cause of this infection, arsphenamin seems to be specific in the experimental disease in animals; favorable results are reported for man. Mazaki recommends for the prevention of rat-bite fever cauterization of the wound with actual cautery or with strong phenol.

Other infections may follow rat bites. Thus, Schottmüller in 1914 described *Streptothrix muris rattii*. The same or a similar streptothrix was also found by Blake in a fatal case at the Peter Bent Brigham Hospital in Boston, by Ruth Tunnicliff in Chicago, and by Tileston in New Haven.

INFECTIOUS JAUNDICE

(*Spirochaetosis icterohæmorrhagica*) *

Infectious jaundice is an acute infection, caused by the *Spirochaeta icterohæmorrhagica*, and characterized by malaise, prostration and gastro-intestinal symptoms at the onset; by fever of varying degree, and by jaundice which

⁸⁵ Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1902; also Proescher, Internat. Clin. IV, 25th S., 77.

⁸⁶ J. Exper. M., 1916, 23: 39.

⁸⁷ J. Exper. M., 1916, 23: 249; 1917, 25: 33, 45; J. Infect Dis., 1919, 124: 366.

comes on about the fourth day and develops to varying intensity and duration. In severe cases bleeding from mucous surfaces and albuminuria are common. Light cases resemble ordinary catarrhal jaundice. The incubation period in man is about one week (Inada).

Infectious jaundice has been known for a long time to occur among troops, among sewer workers, among miners, and among agricultural laborers working in wet soil, as rice planters; also people who handle food, at least in Japan, are said to be attacked with especial frequency. It was recognized as a clinical entity and reported by a number of writers long before 1886 when Weil described four cases with typical symptoms. This aroused general interest in the disease, which is called by his name. It is not certain what form of infectious jaundice he saw, and the term "Weil's disease" is confusing.

In addition to the above, there are several other epidemic diseases characterized by jaundice, due to a variety of organisms, some of unknown etiology. Epidemic catarrhal jaundice occurs as localized epidemics in the United States and is not associated with *Spirochæta icterohæmorrhagica*. The cause is not known. It has been studied by Blumer and seems to be a definite entity. There is a form of hemorrhagic jaundice with severe features which occurs in some districts on the Mediterranean. It has a high mortality. The cause is not known.

In most outbreaks of infectious jaundice the mortality is low, but in Japan the infection is both more prevalent and more virulent than in Europe or America. Among the Japanese the case fatality rate is as high as 38 per cent, while in European soldiers in the Great War it did not exceed 2 to 5 per cent. The infection is endemic on all continents among rats. The disease in man is associated with moist soil and moderate temperature; that is, few cases occur in the hottest or coldest weather. Sporadic cases are recorded, but usually the disease occurs in epidemic form, often with a high attack incidence. In civil life it is more common in cities than in the country, and among those who live under insanitary conditions. It prevails especially in summer and autumn; occasional cases occur in spring and winter. The distribution is world wide.

An account of an outbreak of jaundice among troops in the War of 1812 has come down to us. Not less than 71,691 cases occurred among white troops of the Union Army in the American Civil War. It followed the Franco-Prussian War. In the South African War 5,648 cases of epidemic jaundice occurred among the British troops. Many cases occurred among the troops on both sides of the Rhine in the World War. It prevailed from Belgium to Gallipoli.

Official statistics give no adequate idea of the prevalence of this disease, as large numbers of men continued on duty throughout their illness. Regimental medical officers have stated that at times as many as one-tenth of their men actually in the trenches were jaundiced. In one battalion there were one hundred cases during October, but only thirty-six were regarded as of sufficient severity to be sent into a field ambulance. It was called trench

jaundice. A number of epidemics of catarrhal jaundice have been reported among the civil population of the United States. A list of these outbreaks is given by Neil.⁸⁸

The cause of infectious jaundice is the *Spirochæta icterohæmorrhagica* of Inada and Ido (1916).⁸⁹ Noguchi⁹⁰ believes the spirochete belongs to a special genus and has named it *Leptospira icterohæmorrhagiæ*. It is morphologically similar to *Leptospira icteroides* which he finds associated with yellow fever. *S. icterohæmorrhagica* is an aërobe, and grows well on special but relatively simple media. It is pathogenic for guinea-pigs.

Diagnosis depends upon the spirochetes, which occur in the blood during the first five or seven days. They may be seen under the microscope, or obtained in pure culture by inoculating blood intraperitoneally into guinea-pigs. The spirochetes appear in the patient's urine after the tenth day, and may persist for weeks. *S. icterohæmorrhagica* may be differentiated from other forms of spirochetes by agglutination.

The Relation of the Disease to the Rat.—Infectious jaundice is a disease primarily of rats, secondarily of man. About 10 per cent or more of all wild rats, wherever examined, harbor the spirochetes. Rats have a remarkable immunity to the disease, gained perhaps through long association between parasite and host. The spirochetes live in the kidneys of the rats and are excreted in the urine. They have also been found in the blood and other organs, and in the mouth.

Noguchi found the spirochetes in rats caught about the Bronx; Jobling and Eggstein found 10 per cent of the rats examined in Nashville to be infected; Neil 10 per cent of the rats in Washington; and Otteraaen found the parasites in the rats of Chicago. This, therefore, reveals a latent danger to which we have been constantly exposed, but from which we can escape as long as sanitary conditions are satisfactory.

The credit of first finding the *Spirochæta icterohæmorrhagica* in rodents belongs to the Japanese investigators who demonstrated these parasites in the kidneys of field mice. Further investigations in the coal mining regions of Japan showed that 40 per cent of the wild rats harbored the organism, and that many cases of infectious jaundice in human beings occur in this region.

Transmission.—Just how the disease is usually transmitted from rat to rat and from rat to man is not entirely clear. There are several possibilities. There is scant evidence that the infection is transmitted from man to man.

The spirochete which is excreted in the urine of the rat contaminates the soil and also water and food, and infection takes place either through the skin or by the mouth. Susceptible animals may be infected by either route. The infection will pass through the unbroken skin of guinea-pigs. The spirochete is frail,⁹¹ but may live three (Noguchi) to seven days (Reiter), in moist

⁸⁸ U. S. Pub. Health Rep., 1918, 33: 717. Contains a complete bibliography to date.

⁸⁹ J. Exper. M., 1916, 23: 377.

⁹⁰ J. Exper. M., 1917, 25: 755.

⁹¹ J. Exper. M., 1918, 27: 609.

soil; it soon dies when dried. This probably explains the relation of the infection to moist soil and a moderate temperature. It also makes clear the reason why the wet trenches, overrun with rats, favored the transfer of the disease from rats to man during the World War.

The spirochete has been found in the mouth of rats and the infection has been transmitted to guinea-pigs by the bites of such rats. This probably is not the usual method of spread in nature except from rat to rat.

Foulerton⁹² found *Spirochæta icterohæmorrhagiæ* in the feces of infected guinea-pigs, and its presence in the feces of cases of spirochetel jaundice in man may be assumed.

There is no adequate evidence that any insect plays a part in the transmission of the disease, although the experimental evidence in this regard is by no means complete. Mosquitoes, bedbugs and horseflies have all proved to be incapable of transmitting the spirochete. Reiter claims to have transmitted the disease by means of a biting fly, *Hæmatopota pluvius*.

Prevention consists in warfare against rats. The disease is not known to be communicated directly from man to man. Sanitation both in civil and military life to guard against contamination with rat excrement is an essential element of prophylaxis. Shoes should be worn, especially in wet soil and where the infection abounds. Food must be carefully guarded against rats and mice, for infection by the mouth occurs, and food soiled with rat urine is hazardous. The heat of cooking is sufficient to kill the spirochete. Contaminated water may be a source of infection through bathing, washing, etc.

There is no specific treatment. Prophylaxis is fundamentally a question of biologic cleanliness.

RAT LEPROSY

An infection resembling leprosy occurs spontaneously among rats and bears a close resemblance to the disease in man, but rat leprosy apparently is not communicable to man. For a further discussion of rat leprosy see page 417.

TRICHINOSIS

The three most important hosts for *Trichinella spiralis* are man, swine, and rats. The infection is spread mainly by one animal eating the flesh of another. It is, therefore, evident that if the disease occurred only in hogs and man it could be controlled. Hence, a well-directed public health campaign against trichinosis should consider the eradication of rats, especially around slaughterhouses, butcher shops, hog pens, and similar places.

Trichinosis is very common among rats; they become infected by eating each other, by eating scraps of pork found on the offal pile of slaughterhouses, butcher shops, or in swill. Swine become infected by eating rats and

⁹² *J. Path. & Bacteriol.*, 1919, 23: 78.

infected offal. Man becomes infected almost exclusively by eating pork or boar meat that has not been thoroughly cooked. The subject is fully discussed on page 753.

Food infection due to the Gärtner group of bacilli may be associated with rats and mice, for these rodents may be carriers of these microorganisms. The opportunity for food to become contaminated with rat and mice feces is frequent in the slaughterhouse and butcher shop, in the grocery and home, and in storage and transportation (see page 652).

OTHER PARASITES

Rats and mice may harbor eleven species of internal parasites which also occur in man. Several of these are of academic importance only.

Those which concern us principally, in addition to *Trichinella spiralis*, are *Hymenolepis nana*, *Hymenolepis diminuta*, and *Lambliia intestinalis*. However, Simon⁹³ believes there are specific differences between human giardiasis and the mouse form, and that human infection is of human origin.

Rats are also susceptible to experimental infection with *Trypanosoma gambiense*, the cause of sleeping-sickness, but are not known to play any part in the spread of this disease under natural conditions.

Darling has shown the relationship between the relapsing fever of Panama and the rat, which is susceptible to *Spirochata novyi*, the cause of this disease. The tick, *Ornithodoros talaje*, the nymphal stage of which is found on rats, transmits the spirochete.

Lynch⁹⁴ states that the rat (*Rattus norvegicus*) suffers from spontaneous amebic dysentery similar to that occurring in man (see page 140). The rat is, therefore, a possible disseminator of dysentery amebæ pathogenic for man.

Rats have also been accused of dragging typhoid from the sewers to our food. The connection may be close but the possibility distant. An outbreak of typhoid fever in an asylum was, in fact, claimed to this source by Mills.⁹⁵

PLAGUE IN RATS

It is now known that rats and rat fleas are responsible for most cases of human plague of the bubonic type, and that rats are the most frequent medium by which plague is carried from one locality to another. They also convey the plague infection to other rodents, such as ground squirrels.

The clinical manifestations of plague in rats are not very evident. It is generally said that a plague-infected rat staggers about with a drunken gait, loses fear of its natural enemies, and is readily captured. Rats experimentally

⁹³ *Am. J. Hyg.*, 1922, 2: 406.

⁹⁴ *J. Am. M. Ass.*, 1915, 65: 2232.

⁹⁵ *Pub. Health Bull.*, No. 103, June, 1919.

infected show no marked manifestations of illness until shortly before death, when they become quiet, crouch in the corner of the cage, and try to hide. It is rather surprising that comparatively few dead plague rats are found in endemic centers. In the San Francisco campaign McCoy estimates that certainly not more than 20 per cent of the infected rodents were found dead, the remainder being trapped.

Rats suffer both with acute plague and chronic plague, the lesions of which differ.

The diagnosis of plague in rats may be made macrascopically. The Indian Plague Commission, which had the opportunity of examining an enormous number of plague rats in Bombay and elsewhere in India, state that "the results of tests carried out for the purpose of comparison make it manifest that the naked eye is markedly superior to the microscopic method as an aid in diagnosis, and as the result of our experience we are prepared to make a diagnosis of plague on the strength of the macrascopical appearance alone, even though the other results of cutaneous inoculation and culture are negative and the animals show signs of putrefaction." The experience of McCoy and others in the Federal Plague Laboratory in San Francisco leads to the same conclusion. It should be remembered, however, that occasionally plague occurs in rats without gross lesions. In fact, infected rats may show only obscure or no signs of infection. This has been observed by Dunbar and Kister, by McCoy, and also by Williams and Kemmerer.⁹⁶ In any critical case the bacteriological confirmation is essential.

Acute plague in rats is characterized by engorgement of the subcutaneous blood-vessels and a diffuse pink color of the subcutaneous structures and muscles. The diagnosis may often be inferred at the first incision. The lymphatic glands of the neck, axilla, groin, or pelvis are enlarged and frequently surrounded by a hemorrhagic exudate and edema. The liver is granular with focal necroses, the spleen enlarged and friable, and pleural effusions are common.

Chronic plague in rats has been encountered in a considerable number of cases among *Rattus rattus rattus* in the Punjab villages of Kasel and Dhand. It has not been found in California, but has been observed in New Orleans. In the chronic disease the lesions consist of purulent or caseous foci, usually in the viscera; that is, they occur as splenic nodules and abscesses, or mesenteric abscesses. Sometimes the abscesses are situated in the regions of the peripheral lymph glands. Plague bacilli are either absent or very scanty upon microscopic examination in these abscesses, but they may be recovered by cultural methods or more surely by inoculating the material into susceptible animals. Some workers consider scars in the spleen as evidence of chronic plague or of recovery from this disease. The relation between chronic rat plague and the recurrence of acute plague among rats, if any, has not been demonstrated.

Rats may be infected by the ingestion of infective material or the appli-

⁹⁶ U. S. Pub. Health Rep., 1923, 38: 1873.

cation of virulent plague bacilli to a mucous or cutaneous surface, or by subcutaneous injection of the microörganism. The infection may also be transferred from rat to rat through the agency of the flea. In nature transference probably takes place commonly through the flea, and rarely in any other way.

Contrary to the general impression, the wild rat has a considerable resistance to plague infection. The Indian Plague Commission found that 59 per cent were immune when inoculated by the subcutaneous method from the spleen of infected rats. A series of experiments conducted in the Federal laboratory in San Francisco also showed a high grade of immunity in about 15 per cent of small rats and about 50 per cent of large rats when inoculated with highly virulent material. The experiments demonstrated that this immunity is not acquired through a prior attack of the disease, but must be a natural immunity.

The natural subsidence of plague among rats in any community is a point about which much more evidence must be obtained before we can speak with any degree of authority. It may be due to a lack of susceptible material, possibly to a loss of virulence of the organism, but it seems more probable that it is due to a change in the number or activities of the rat fleas.

SQUIRRELS AND PLAGUE

In August, 1903, a blacksmith died of plague probably contracted from a squirrel in Contra Costa County, California. In 1904 Currie demonstrated the susceptibility of the ground squirrel to bubonic plague. In 1908 McCoy and Wherry discovered natural plague in ground squirrels. It was then learned that thousands of squirrels had died of some disease during 1904, 1905, and 1906. This epizootic was doubtless plague. It is now realized that plague has become endemic in California in the squirrel. It is also believed that the disease has been kept alive in the endemic foci of Tibet in another rodent, the marmot (*Arctomys bobac*). The eradication of plague must, therefore, consider these and perhaps other susceptible wild animals.

California is overrun with three species of ground squirrels. The commonest is the *Otospermogrammus beecheyi*; syn. *Citellus beecheyi*. They live in colonies in burrows or warrens. The booby owl is a frequent companion occupying the same burrow, and possibly they spread the infection by carrying fleas. Squirrels become infected through fleas from each other and from rats. The squirrel flea (*Ceratophyllus acutus*) attacks man just as the rat flea does. The infection may also be conveyed to man through squirrel bites, as in the case of the child in Los Angeles studied by Stimson. Squirrels make good food for man, but since the danger has been realized the shooting or trapping of them for food purposes is now forbidden in California.

An outbreak of pneumonic plague in Oakland, California, during August and September, 1919, started with a man who went squirrel hunting in Berkeley Hills on August 11th and 13th, and took ill August 15th. The

outbreak involved fourteen cases, thirteen of whom died.⁹⁷ Another localized epidemic of the same type, but more extensive, occurred in Los Angeles, California, in November, 1924. In this episode, it is believed that the squirrels infected the rats, which in turn passed the disease on to a man, who started the epidemic.

Plague in the squirrel may be recognized⁹⁸ by the gross anatomical lesions in the lymphatic glands, the liver, and lungs. Involvement of the lungs is common in the squirrel. Many cases are subacute or chronic. Smear preparations from squirrels dead of plague are frequently negative for plague-like bacilli. The diagnosis may, therefore, be made more surely by animal experimentation. Subcutaneous inoculation is surer than the cutaneous method, as the latter often fails on account of the comparatively few plague bacilli present in squirrel lesions.

Squirrels may be destroyed by various means. One of the most successful is to saturate a ball of cotton waste the size of an orange with carbon disulphid and place it in the warren; then close the opening with wet clay. Officers of the Public Health Service working in California have devised an apparatus for vaporizing carbon disulphid and pumping the gas into the burrows. This method is reported to be much more successful than any other that has been employed. Poisoned bait, such as strychnin, phosphorus, or cyanid of potassium, is effective. Traps are not very successful, as the squirrel is wary. Natural enemies, such as the coyote, wolf, badger, skunk, mountain lion, the cobra snake, and red-tailed hawk should be encouraged.⁹⁹

THE PLAGUE

(Peste)

Plague is an infection primarily of rats and other rodents, secondarily of man; caused by *Bacillus pestis*,¹⁰⁰ which belongs to the hemorrhagic septicemia group of bacteria, so called because they are apt to produce hemorrhagic areas throughout the body. When the hemorrhages occur in the skin they are called "plague spots," which gave the name "Black Death" to the disease in the Middle Ages. In addition to this specific definition, the term plague still has a generic meaning. There is much confusion in the literature, because formerly all serious epidemics were called plague and we still speak of them as plagues.

There are two chief forms: (1) bubonic plague, (2) pneumonic plague.

Bubonic Plague.—This is the common type of the disease. It is characterized by fever, depression, great prostration, and painful, inflammatory

⁹⁷ *Am. J. Pub. Health*, 1920, 10: 599.

⁹⁸ *J. Infect. Dis.*, 1909, 5: 485.

⁹⁹ In this chapter material has been freely drawn from "The Rat and Its Relation to the Public Health," Public Health and Marine Hospital Service, 1910, particularly articles by Lantz, McCoy, Brinckerhoff, Banks, Stiles, Rucker, Creel, Hobdy, Kerr, and Rosenau.

¹⁰⁰ According to modern nomenclature *Pasturella pestis*.

enlargement of the lymph-nodes (buboes). The glands of the groin are most commonly affected (54 per cent) because the flea bites are usually on the legs; next, those of the axilla, then the cervical; occasionally, multiple. When suppuration takes place it is regarded as a favorable sign. There is a high leukocytosis. The period of incubation is usually five to seven days. In this type, the plague bacillus is locked up in the glands, blood and other tissues and organs of the body, and is not eliminated in the usual excretions. Bubonic plague therefore is not contagious and is not spread from man to man, but from rodent to man through the flea.

In man the bubonic type of plague exhibits distinct seasonal prevalence; for example, in Bombay, while the disease is present throughout the year, the great bulk of the cases occur during the winter and spring months and the number subsides as the temperature rises; when the mean reaches about 85° F. the disease is relatively infrequent. In the United States, either on the Pacific Coast or on the Gulf Coast, the cases are grouped chiefly in the summer and early fall. Robertson¹⁰¹ has shown that in countries with a mean mid-winter temperature of 45° F. or below, the disease is occasional, accidental and distinctly self-limited. Bubonic plague seems to occur in colder regions only for short periods under unusual conditions. In other words, it is essentially a disease of the tropics and subtropics, flourishing best in moderately warm, dry weather. The geographic distribution and seasonal prevalence correspond to the number and activity of the fleas. Another reason given why bubonic plague is especially prevalent in warm countries is the closer contact between rats and man.

Pneumonic Plague.—Clinically, the pneumonic form of the disease resembles croupous or lobar pneumonia, except that the sputum is more bloody and less tenacious. Few cases recover, death usually occurring in two to four days. The period of incubation is short—one to three days. The bacilli are contained in enormous numbers in the copious sputum. The disease is transmitted directly by close association with a patient having plague pneumonia. The channel of entrance is usually through the respiratory tract, although a secondary plague pneumonia may develop during the course of a case of the bubonic type.

Pneumonic plague may assume epidemic proportions, especially in the cold weather and under circumstances where people come in close association. This was the case in the Manchurian epidemic of 1910-11 (60,000 deaths), which occurred during the winter and was one of the most virulent epidemics of modern times, the case fatality being practically 100 per cent. Other extensive epidemics have occurred, notably in middle China in 1917-18, 15,000 deaths; and a second outbreak in Manchuria in 1920-21, with 9,000 deaths. Extensive epidemics of pneumonic plague are restricted to northern climates, as northern Asia and the northern higher regions of India. A limited outbreak, which started from an infected squirrel, was reported from California in 1919 and a somewhat larger epidemic in 1924.

¹⁰¹ U. S. Pub. Health Rep., 1923, 38: 1519.

Pneumonic plague is usually a cold weather disease, following the seasonal prevalence of other diseases of the respiratory tract. On the other hand, Egypt and other parts of Africa have had small outbreaks of this type.

Septicemic Plague.—This form of the disease is due to an overwhelming infection of the blood with plague bacilli, which kills in a few days, before the buboes have time to appear. Hemorrhages are common.

Pestis minor.—Pestis minor, or ambulant plague, is a mild form of the disease with fever and buboes which may suppurate, but with symptoms so slight that the patient does not go to bed.

Other Forms.—An intestinal form of plague occasionally occurs, causing diarrhea and the features of typhoid fever. In another variety the chief manifestation may be in the skin and subcutaneous tissues.

Diagnosis.—The diagnosis of plague is readily made by finding the bacilli in the blood or lesions. One attack confers a definite immunity. The period of detention for the purposes of quarantine is seven days.

In considering the prevention of plague, it is necessary to recognize that the different types of the disease are contracted and spread in different ways. Bubonic plague, which is the common type, is flea borne; pneumonic plague is a contact infection. The first is not contagious from man to man, the second is very contagious. Pneumonic plague and bubonic plague usually run true to type; that is, ordinarily the pneumonic form remains pneumonic, and the bubonic is predominantly bubonic or septicemic. Pneumonic plague may have its origin in a case of the bubonic type which has developed a secondary plague pneumonia. In the 1920-21 Manchurian epidemic, observers on the ground noted the gradual evolution of the plague from the bubonic, through the septicemic into the pneumonic form. However, the relationship between bubonic and pneumonic plague is obscure. Pneumonic plague rarely occurs from rat infection, but may arise from squirrel infection, because in the squirrels there is a tendency to pulmonary localization. There is even a suspicion that the pneumonic type of the disease is due to a particular strain, or separate variety of plague bacillus. Mixed outbreaks have occurred in Africa in recent years, but generally the tendency is for the two types to remain distinct during epidemics.

To summarize, then, bubonic plague is an insect-borne disease and has an entirely different epidemiology from pneumonic plague, which is a contact infection.

History.—The Philistines made offerings of golden images of the mice that marred the land and of their emerods to stay a pestilence. The epidemiology of this outbreak, which started on the seacoast at Ashdod and moved inland, smiting at Beth-shemesh 50,070, is clearly described in the Book of Samuel. The plague of Athens and the pestilence in the reign of Marcus Aurelius, according to Payne, may not have been this disease. Epidemics of varying severity occurred in Europe for over 1,100 years—from the sixth century in the days of Justinian to the middle of the seventeenth century. The most devastating was the "Black Death" of the fourteenth century which

overran Europe and destroyed one-fourth of the population. The disease gradually subsided and disappeared from Europe and became endemic in a few remote parts of the world (page 344). We thought it had passed away, when suddenly, in 1894, it reappeared at Hongkong and from there again spread over the world. This recrudescence of a disease thought to be extinct is one of the most striking facts in epidemiology.

In 1664-65, the Black Death in London carried off 70,000 of a population then numbering 500,000. A graphic though imaginative description is given by Defoe in *A Journal of the Plague Year*. Numerous references to the disease will be found in Pepys' Diary. Benvenuto Cellini describes his own case in his autobiography. The disease profoundly affected the economic, social and political history of Europe. Plague started in Stratford-on-Avon in July, 1564, when Shakespeare was a baby three months old. From July to December of that year, 237 deaths are recorded in the parish register of the little vicarage of Avon. The infection swept away entire families. Fortunately, not a Shakespeare is on the list. How much has mankind lost throughout the world's long history by the untimely death of genius on account of preventable infections!

The Present Pandemic.—The present pandemic is the most widespread of all, having been carried to the four quarters of the globe by trade and travel from Hongkong where it suddenly reappeared in 1894. It is quite as virulent as the Black Death of the fourteenth century. Thus, in India, over 10,000,000 deaths were reported in the twenty-five years 1898-1923.

PLAGUE MORTALITY IN INDIA, 1898-1923 *

(1921 Population: 319,075,132)

Years	Deaths
1898-1903	1,707,456
1903-1908	4,325,237
1908-1913	2,042,127
1913-1918	2,179,401
1918-1923	530,170
TOTAL (25 years)	10,784,391

* Epidemiological Intelligence No. 8, Health Organization of the League of Nations, August, 1924.

Endemic Foci.—There are four historic endemic foci in which plague has slumbered for ages. One is on the eastern slope of the Himalayas, in the province of Yunnan. The great epidemic in Hongkong in 1894 came from this center. A second endemic focus near, and perhaps connected with the first is on the western slope of the Himalayas. From here the infection was carried to Bombay in 1896, where it still prevails. A third plague focus exists from about the center of Arabia to near Mesopotamia. From here the infection was dragged to Samarkand, the Black Sea, and Persia. The fourth endemic area was discovered by Koch in 1898 in the interior of Africa, near

the source of the White Nile in Uganda. We must now add to this other endemic foci, for plague has obtained a foothold in California in the ground squirrels. The disease also has gained a foothold in the Gulf States—Louisiana, Texas and Florida, but was readily eradicated; and is endemic in many parts of India, Africa, South America and Asia. The number of cases in the United States and their occurrence is shown in the following table:

PLAGUE (BUBONIC TYPE): HUMAN CASES IN THE UNITED STATES

Year	California	New Orleans	Texas	Florida	Total
1900	22	22
1901	26	26
1902	35	35
1903	17	17
1904	10	10
1907	170	170
1908	7	7
1909	1	1
1910	2	2
1911	3	3
1913	2	2
1914	...	30	30
1915	1	1	2
1919	1	12	13
1920	1	7	31	10	49
TOTAL	298	50	31	10	389*

* This includes seventeen cases of squirrel origin.

The *Bacillus pestis* (*Pasteurella pestis*), discovered by Yersin during the Hongkong epidemic of 1894, has more than fulfilled Koch's laws. Several accidents in which pure cultures have been inoculated into man, producing all the symptoms and lesions of the disease, have added to the proof that this organism is the cause of plague (Vienna, 1898, Ann Arbor, 1902, and also in laboratories in Russia, Berlin, and Japan). The plague bacillus is comparatively easy to isolate, grows readily on artificial culture media, and has characteristics that readily distinguish it from all other species. It is a short rod with rounded ends, with a tendency to bipolar staining, not motile, decolorized by Gram's method, and grows well at room temperature.

Recognition of the plague bacillus rests upon the following characteristics: (1) Curious involution forms upon salt agar within twenty-four hours; (2) stalactite growth in liquid media; (3) characteristic lesions produced by experimental plague in guinea-pigs, rabbits, rats, etc. Kolle's method consists in rubbing the material containing the plague bacillus upon a shaved area of the skin of a guinea-pig. The plague bacilli penetrate the skin, leaving other organisms behind. The skin of the guinea-pig thus acts as a differential filter. (4) The final test of the identity of the plague bacillus is the fact that its pathogenicity may be neutralized by the use of antiplague serum.

The plague bacillus has no spore; its resistance corresponds about to that of the colon bacillus. It does not live a saprophytic existence in nature. It

is readily killed by drying, sunlight, heat and the usual germicides. In the feces of dead rats it may live for two months, in sputum from pneumonic cases ten days, and under certain circumstances from six to fifteen days on fabrics and surfaces. There is, however, comparatively little danger of contracting the disease through fomites.

Immunity.—One attack of plague usually protects for life. Occasionally second attacks are noted in the same person, but they are usually mild. This is an old observation and led to the employment of persons with a plague history or a plague scar in hospitals and laboratories.

Artificial immunity of either an active or passive nature may be acquired by various procedures. The passive immunity produced by the injection of antiplague serum lasts only about three to four weeks. The active immunity produced by vaccination of cultures may be depended upon for about six months.

Haffkine's prophylactic consists of a killed culture of the plague bacillus, which is injected subcutaneously. Haffkine used a bouillon culture, six weeks old, grown at 25-30° C. and killed at 65° C., for one hour. One-half of one per cent of phenol is then added. From 2 to 3.5 c.c. (this was later increased to 20 c.c.) of this vaccine are injected subcutaneously. Ten days later a second injection of a still larger amount is given.

The German Plague Commission prepared their prophylactic vaccine from a fresh virulent agar culture, suspending the killed bacilli in salt solution or bouillon. Lustig and Galeoti extract the immunizing substance from the bacterial cell (endotoxin) with weak potassium hydroxid. Terni and Bandi recommend the peritoneal exudate of plague-infected guinea-pigs. Shiga prefers a combined active and passive immunity produced with killed cultures and antipest serum, because this mixed immunizing process has the advantage of producing milder reactions.

Kolle and also Strong started out from the principle that a much higher degree of immunity is produced by living microorganisms than dead ones, and recommended the use of live attenuated cultures.

The reactions which follow vaccination with a plague culture, whether alive or dead, are sometimes marked. The symptoms consist of a rise in temperature to 39° C., malaise, depression, and headache, and swelling and pain at the site of the inoculation. The symptoms usually pass away in twenty-four to forty-eight hours.

McCoy and Chapin¹⁰² state that there is no evidence indicating that vaccination for plague has ever controlled an outbreak. A community should not be allowed to delude itself into the belief that plague may be controlled in this manner.

Yersin's serum is obtained from a horse that has received repeated injections of plague cultures; at first killed plague cultures, afterward living bacilli, are used. At most this antitoxic serum is weak; it has feeble and

¹⁰² U. S. Pub. Health Rep., 1920, 35: 1647.

transient protective properties, and doubtful curative power. Very large quantities must be administered early in the disease to obtain any effect at all.

Management of a Plague Epidemic.—The handling of a plague epidemic is conducted along two definite lines of activity. One is to find and care for the human cases, the other consists in a warfare against rats. All three species of the common rat may transmit plague. With us, it is mainly the Norway rat; in Africa it is *Rattus rattus rattus*. The organization and general management of a plague campaign do not differ radically from similar work in other epidemics (see page 523). Cases of the disease must be sought for and

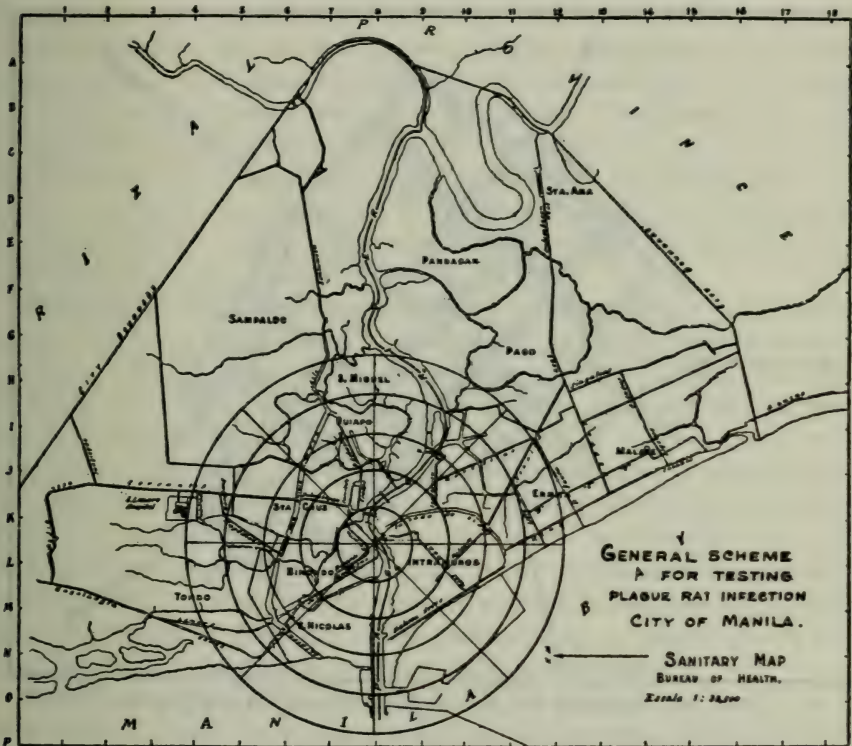


FIG. 36.—A GENERAL SCHEME FOR TESTING PLAGUE RAT INFECTION, CITY OF MANILA.

early diagnosis confirmed; all deaths from no matter what cause must be investigated, and the body examined by an expert before burial is permitted. This is particularly important in outbreaks of the pneumonic type. A bacteriological laboratory is a *sine qua non*. Cases of the disease should be isolated and the usual disinfection of excreta and surroundings exercised. Particular care must be taken that the isolation wards are vermin-free. The place from which the case is removed should be given a preliminary disinfection with sulphur dioxide or other substance that may be depended upon to kill rats and fleas, and a search made in the neighborhood for other cases both in man and rodents.

The campaign against the rat is expensive and difficult, but must be vigorously prosecuted to insure success. The rat warfare may be briefly summarized as a simultaneous attack upon the habitation and food supply of the rat; the destruction of rat burrows and nesting places; the separation of the rat from his food supply by concreting and screening such places as stables, warehouses, markets, restaurants, etc.; the prevention of the entry of the rat into human habitations by the use of concrete, wire netting, or other barriers; and the use of poisons, traps, etc. For further consideration concerning rats and their eradication see page 329. All the rats that are caught in traps or

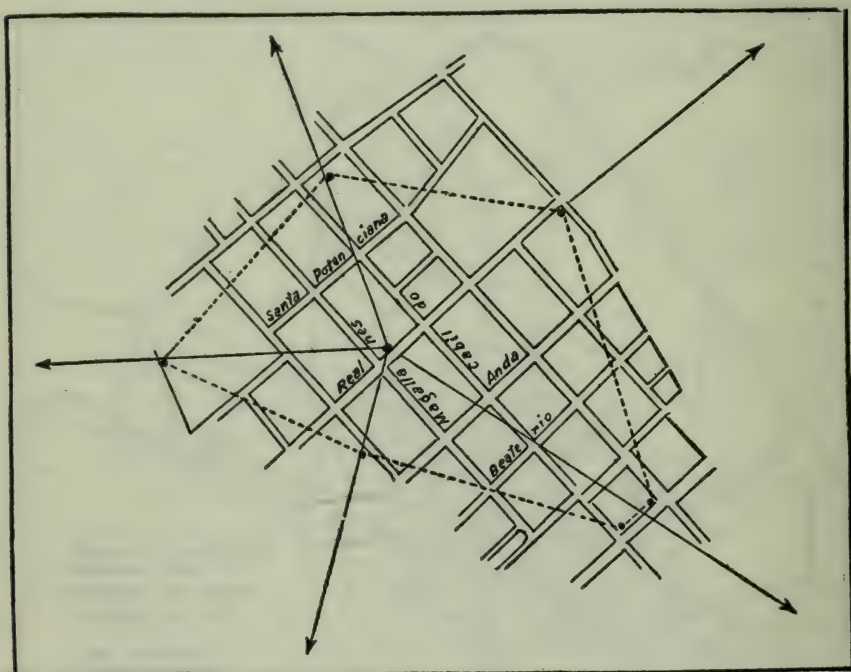


FIG. 37.—ISOLATED PLAGUE-INFESTED CENTER, MANILA, P. I.

found dead are brought to the bacteriological laboratory, where they are examined and careful records kept concerning the species, the location, the place where the rat was caught, the character of the infection, etc. As it is a hopeless task to exterminate rats from a large city, Heiser has proposed a practical plan which proved effective in Manila. A list of the places in which the plague-infected rats were found was made. Each was regarded as a center of infection. Radiating lines, usually five in number, were prolonged from this center, evenly placed like the spokes of a wheel. Rats were caught along these lines and examined. Plague rats were seldom found more than a few blocks away. The furthestmost points at which the infected rats were found were then connected with a line, as is roughly shown in the diagram, Figs. 36 and 37. The place inclosed by the dotted line was regarded as a section of

infection. The entire rat-catching force was then concentrated along the border of the infected section, that is, along the dotted line. They then commenced to move toward the center, catching the rats as they closed in. Behind them rat-proofing was carried out. One section after another was treated in this way, until they had all been wiped out. Once weekly thereafter rats were caught in the previously infected sections and at other places, especially those which had been infected in years gone by. Since the above system was adopted, plague has disappeared in the city of Manila, and at a cost of only a small fraction of that of a general rat extermination campaign. A campaign along these same general lines has also been successful in New Orleans.

The pneumonic type of the disease requires a campaign of an entirely different nature. It is often a family infection; at least those in close contact with cases are apt to come down with the disease. The infected area must be rigorously isolated, contacts kept under observation and quarantined for at least five days, and all suspicious deaths investigated bacteriologically. Carriers are not known to play a rôle in pneumonic plague, but the possibility must be considered. On the basis of American experience, the pulmonary type tends to be self-limiting under our conditions.

Maritime Quarantine.—Plague infection is frequently carried overseas in vessels. When this happens it is more apt to be due to the disease in the rat than man. Maritime quarantine, therefore, finds its greatest justification in keeping out plague. To be successful, measures must be directed almost entirely against the rat. Rats may be kept down on board a vessel by the frequent use of sulphur dioxide. All vessels trading with a plague-infected port should have each cargo compartment fumigated with this gas, or better, hydrocyanic acid, at least when it is empty, at the port of departure. The vessel must be again fumigated on arrival. Both at the infected port and at the port of arrival, care must be taken to prevent the ingress and egress of rats. The period of detention of the personnel for a plague ship is seven days. For further details concerning quarantine see page 529.

Prevention—Summary.—The principles and many of the details for the prevention of plague have been stated in the foregoing pages and need not be repeated.

Personal prophylaxis consists in avoiding the infected regions and guarding against flea bites. Physicians and nurses should remember that the pneumonic form of the disease is highly "contagious" in the ordinary sense of the term. Attendants and persons who come in contact with such cases may protect themselves with Haffkine's prophylactic. Individual measures to guard against droplet infection, such as the wearing of masks or veils of cheesecloth, may be resorted to in dealing with the pneumonic form of the disease. The bubonic and septicemic forms of the disease are not directly communicable.

Cases of plague should be isolated in a well-screened, vermin-proof room. Fabrics and other objects which become contaminated with the discharges should be thoroughly disinfected by proper methods.

It is important to have prompt reports of all cases of suspected plague, and the diagnosis must be confirmed by bacteriological methods. In all plague centers there should be a special hospital and also a laboratory where diagnostic work may be carried on; this is an essential part of the equipment for a successful campaign.

The prevention of plague, after all, is reduced to warfare against rats and fleas. This has been fully discussed. All seaport towns having communication with plague countries should examine rats caught about the wharves and other places for plague. This, in fact, should be one of the routine duties of the port sanitary authorities. Plague may slumber in the rats for years before human cases occur; indeed, there may be fairly extensive infection among rodents and very few human cases. Other preventive measures are obvious from the nature of the infection and its mode of transmission, or have already been stated in the preceding pages.

TICKS

Ticks are *Arachnoidea*, of the order *Acarina*, family *Ixodidae*, subfamily *Argasinae* and *Ixodinae*. Quite a number of genera and species are known to attack man.

Ticks, or wood lice, are not true Insecta, but belong to the Acarinoids which include the mites, and are closely allied to spiders and itchmites (scabies). Ticks have an unsegmented body with eight legs in the adult stage and six legs in the larval stage. In some of their habits they resemble bed-bugs. So far as is known, they take no vegetable food, but live on blood. Ticks are ectoparasites of man and many animals. They frequently hang tenaciously to the skin, in which they partly bury themselves. If covered with oil or vaselin, thus closing their breathing pores situated behind the fourth pair of legs, they may be induced to release their hold. If pulled off roughly the head (capitulum) is likely to break off and remain in the skin. Sulphur in some form is useful to destroy ticks in the adult stage. Sulphur ointment is particularly obnoxious to this group of parasites. Arsenic and crude oil also act as poisons to the tick, and may be used by local application.

Tick paralysis in man and animals as a result of tick bites has been reported from South Africa and Australia, and also from Oregon and British Columbia. It has been reproduced experimentally in the sheep by allowing the spotted fever tick, *Dermacentor andersoni*, to bite along the spinal column. Cases in children have been reported (MacCormack).¹⁰³

Life Cycle.—The life cycle of the tick consists of four distinct stages, viz., egg (embryo), larva, nymph, and adult. The eggs are invariably deposited on the ground in large masses. The larvæ which emerge from the eggs are minute six-legged creatures. The larvæ attach themselves to a suitable host, upon which they feed, then usually drop to the ground and molt, becoming nymphs.

¹⁰³ *J. Am. M. Ass.*, 1921, 77: 260.

The nymphs have eight legs. The nymph climbs a blade of grass and waits until it can attach itself to a host, engorges blood, usually drops, molts its skin and becomes adult. Ticks take one, sometimes two and rarely more years to mature. Those that drop between molts may be delayed a long time before finding another host. The egg is the over-wintering stage, but the later stages may hibernate. The free-living stages prefer shady places, and are destroyed by sunshine; hence, clearing the land and cultivation are good measures of eradication.

Methods of Transmission.—It was first shown by Smith and Kilborne that in the case of Texas fever the microorganism within the adult tick passes into the egg and is, therefore, transmitted “hereditarily” to the next generation. The infection of Rocky Mountain spotted fever, of canine piroplasmosis, and probably also that of African tick fever, is also transmitted by the female to the next generation. Tick-borne diseases are not always transmitted in nature in this way. The virus may be transferred directly by the larva, the nymph, or the adult. Thus, some ticks leave their host repeatedly, and the parasites they draw from one animal may be injected into another animal either during the same or at a subsequent stage in the development of the tick.

Control of Ticks on Cattle: Dipping.—Ticks upon domestic stock may be controlled by dipping, spraying, or by hand methods. The arsenical dip has practically displaced all others for the destruction of ticks in the various parts of the world. Arsenical solutions containing sodium arsenite in amounts equivalent to about 0.2 per cent arsenious oxid (As_2O_3), or less according to the frequency of dipping and other conditions, give the best results in killing ticks without injury to the stock. This strength of arsenic, however, will not kill or prevent egg-laying by all the engorged females. Crude petroleum oils have been used to a considerable extent. They are more expensive than the arsenical dip, and dangerous to cattle under some conditions. Serious losses have followed the use of heavy oils in dry regions, or where it has been necessary to drive the cattle any considerable distance after dipping.

A commonly used formula for arsenical dip is as follows:

Sodium carbonate (sal soda)	24 pounds
Arsenic trioxid (white arsenic)	8 pounds
Pine tar	1 to 2 gallons
Water	sufficient to make 500 gallons

The above formula may be used when treatments are given not oftener than every two weeks, but in some tick-borne diseases more frequent dippings are necessary owing to peculiarities in the life histories of the various species of ticks, and weaker solutions, although less effective in destroying ticks, must be used to avoid injury to the cattle.

Sometimes dipping is not practical. Instead of driving cattle considerable distances to dipping vats it will be found sufficient to treat them thoroughly by hand methods. The procedure consists simply in applying the arsenical

mixture liberally by means of rags, mops, or brushes, or by means of spray pumps. Crude oil may be used by hand instead of the arsenical solution.

TICK-BORNE DISEASES

The following diseases transmitted by ticks will be given brief consideration: Texas fever (*Margaropus annulatus*), South African tick fever (*Ornithodoros savignyi*), Rocky Mountain spotted fever (*Dermacentor andersoni*), and relapsing fever (*Ornithodoros moubata*); although it is probable that the latter disease is also transmitted by *Argas persicus* and perhaps other biting insects.

TEXAS FEVER

Texas fever or splenetic fever is also known as bovine malaria, tick fever, and hemoglobinuria. The disease does not affect man. It is confined to

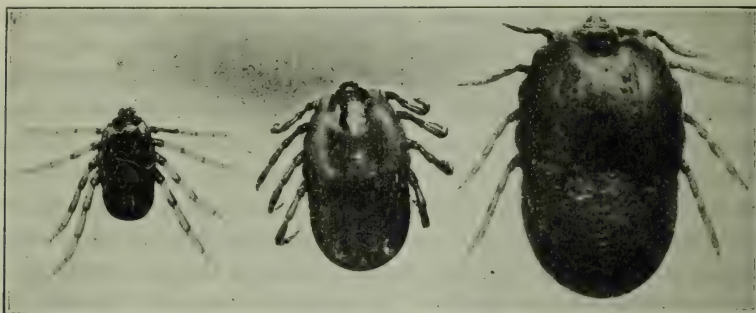


FIG. 38.—THE TEXAS FEVER TICK. (*Margaropus annulatus*.)

cattle, and is of very great economic importance. Texas fever is an infection which should be understood by all sanitarians, on account of its scientific and historic importance. The cause of this infection and its mode of transmission were ascertained in 1893 by Smith and Kilborne. The discovery that the tick is the intermediate host of Texas fever opened an entirely new principle in the sanitary sciences.

Texas fever is caused by a protozoön parasite. This parasite was first named *Pyrosoma bigeminum* on account of the twin-like, pear-shaped forms commonly seen in the red corpuscles. This genus was changed by Patton in 1895 to *Piroplasma*. These terms having been preoccupied, the present name of the parasite is *Babesia bigemina*.¹⁰⁴

The contagium is carried by the cattle tick, *Boöphilus bovis*, now *Margaropus annulatus*. This tick lives upon the skin and feeds upon the infected blood, becomes sexually mature at the last molt; the female drops to the ground and lays about 2,000 eggs; the newly hatched larvæ attach themselves to the skin of a fresh host, which they infect. This explains the long

¹⁰⁴ These various names are given for the reason that they are all found in the literature.

extrinsic period of incubation in this disease, forty to sixty days, thirty days of which are required for the development of the larvæ and the remainder for the development of the parasite within the host.

ROCKY MOUNTAIN SPOTTED FEVER

Rocky mountain spotted fever is an interesting infection, with a very limited geographic distribution. The symptoms closely resemble those of typhus fever, including an eruption, first macular, then petechial. This may go on to gangrene of the skin, due to thrombi of the peripheral vessels. Irritability and hyperesthesia of the skin are common symptoms and are due to infiltration of the peripheral nerves. There is no leukocytosis, but an increased number of mononuclear cells.

The disease is very mild in Idaho and very virulent in Montana, which has a case fatality as high as 90 per cent. About 500 cases occur in the United States a year. The disease prevails especially in the spring.

Rocky Mountain spotted fever may be distinguished from typhus fever in the guinea-pig: After intraperitoneal injection of infected blood, the period of incubation in Rocky Mountain spotted fever is three days, while in typhus it is nine to eleven days; typhus does not show the scrotal and foot lesions of Rocky Mountain spotted fever; the virus of typhus is much less virulent for guinea-pigs; there is no cross immunity: the two diseases are therefore distinct.

Rocky Mountain spotted fever is focalized on the western slope of the Bitter Root Valley, Montana, where it is most prevalent and fatal; the infection has spread over seven states in the Rocky Mountain region, namely, Colorado, Idaho, Montana, Nebraska, Utah, Oregon, and Wyoming, and is also found in California, South Dakota, Washington and British Columbia.

Wilson and Chowning first suggested that the tick acts as the carrier of Rocky Mountain spotted fever. This was proved by Ricketts in 1906, who showed that the particular tick is *Dermacentor andersoni*.¹⁰⁵ Other ticks (*Dermacentor modestus* and *Dermacentor marginatus*) also transmit the infection. The infection may be transmitted by the larva, the nymph, and both the adult male and female ticks. The disease is transmitted to susceptible small animals by the larvæ and nymphs; but the mouth parts of these immature forms are probably not strong enough to feed on man and other large animals, which are infected by the adult ticks. The infection is also transmitted "hereditarily" through the ticks to their larvæ. The disease has been transmitted from man to monkey and the guinea-pig. Rabbits, ground squirrels, woodchucks, and other animals are susceptible.

Infected ticks have actually been found in nature. Ricketts found one out of 296; Maver, two out of 402; and McClintic, six out of 1,037. The ticks were collected from known infected districts and fed on guinea-pigs

¹⁰⁵ Also called *D. occidentalis* and *D. venustus*.

in groups. McClintic died of Rocky Mountain spotted fever while studying the disease.

Maver has proved by experiment that different species of ticks collected



FIG. 39.—ROCKY MOUNTAIN SPOTTED FEVER TICK (*Dermacentor venustus*).

1, Adult female, unengorged, dorsal view; 2, Adult male, dorsal view; 3, Adult female, unengorged, ventral view; 4, Adult male, ventral view; 5, Adult female in act of depositing eggs.

from various regions [*Dermacentor marginatus* (Utah), *Amblyomma americanum* (Missouri), and *Dermacentor variabilis* (Massachusetts)] are able to transmit the virus of Rocky Mountain spotted fever. The inference is that

the disease may find favorable conditions for its existence in localities other than those to which it is now limited.

Rocky Mountain spotted fever evidently belongs to a family of diseases. It is closely related to typhus fever, transmitted by lice; to Japanese river fever, transmitted by a small mite; and to a disease called pseudo typhus in Sumatra, as well as to other insect-borne infections.

One attack of the disease establishes a very high degree of immunity. No authentic case of a subsequent attack in man is known, and laboratory animals have always been found completely immune after a primary infection. The blood-serum of recovered cases contains protective properties of a rather high degree for guinea-pigs (King). The virus is not filtrable through a Berkefeld filter.

Ricketts found "bodies" in the blood of human and experimental cases and also in the tissues and eggs of infected ticks. These observations have been confirmed by Wolbach,¹⁰⁶ who finds bacillus-like bodies in the lesions of Rocky Mountain spotted fever. The characteristic form is a short rod, in pairs and clusters, growing in great numbers in the lesions of the blood-vessels, testicle, skin, and subcutaneous tissues. Wolbach later identified these bodies as *Rickettsia*, which are described on page 368. Noguchi¹⁰⁷ has cultivated rickettsia-like microorganisms isolated from the Rocky Mountain spotted fever tick, *Dermacentor andersoni*.

The organism has been grown in tissue plasma cultures.¹⁰⁸ It invades all the tissues of the tick, even the ova and the spermatozoa, which accounts for "hereditary" transmission of the infection. In man these rickettsia bodies are mainly localized in the smaller peripheral blood-vessels, causing proliferating endarteritis.

The prevention of Rocky Mountain spotted fever is directed entirely against the tick. Ticks are to be avoided in the infected region. If it is necessary to work in the fields and woods and about animals where these ticks abound, the bites should at once be cauterized with strong carbolic acid. In endemic regions the entire body and clothing should be examined twice daily for ticks.

The ultimate control of Rocky Mountain spotted fever depends upon the suppression of the *Dermacentor andersoni* and related ticks in infected areas. This, perhaps, is not so hopeless a task as may at first seem likely.¹⁰⁹ Henshaw and Birdseye¹¹⁰ found ticks either in the immature or adult stage upon twenty species of five hundred mammals examined in and around Bitter Root Valley. The mammalian hosts of fever ticks fall naturally into two groups: those that harbor chiefly adult ticks and those that harbor the younger stages. In the former class belong mountain goats, bears, coyotes, badgers, woodchucks, and possibly elk, deer, mountain sheep, rabbits, and domestic stock, such as horses,

¹⁰⁶ *J. Med. Research*, 1916, 34: 121; 1919, 41: 1.

¹⁰⁷ *J. Exper. M.*, 1926, 43: 515.

¹⁰⁸ *J. Med. Research*, 1923, 44: 231.

¹⁰⁹ Fortunately the *Dermacentor andersoni* is the only tick in the endemic region which attacks man.

¹¹⁰ U. S. Dept. of Agriculture, Bureau of Biology Survey, *Cir.* 82.

cattle, and sheep. Those of the second class, harboring the nymphs and larvæ, are mainly rodents and comprise ground squirrels, woodchucks, chipmunks, pine squirrels, mice, and wood rats. These smaller animals are too agile to permit the adult ticks to remain upon them.

Unquestionably the great bulk of fever ticks (*Dermacentor andersoni*) which become engorged in the Bitter Root Valley do so on domestic stock—horses, cattle, sheep, and sometimes dogs. They obtain the ticks from the pastures and other uncultivated land infested by wild animals. It is obvious, therefore, that, if the domestic animals in the valley are rendered tick-free by dipping, spraying, or by some other equally effective method, the chances of the infection of human beings will be vastly lessened.

The measures proposed for the eradication of the tick are as follows: Clearing and cultivation of tillable land; burning over of foothills and "slashings"; killing of small wild mammals; dipping of domestic animals in arsenical dip; spraying and removing ticks by hand from domestic animals. Each one of these measures has a rational basis in the bionomics of the tick *Dermacentor*, but although all of these methods have been attempted on the west side of the Bitter Root Valley for three years or more they have not greatly diminished the number of ticks to be found nor the number of deaths from spotted fever. The extent and inaccessibility of the infested territory, and the consequent expense have rendered the problem difficult. Fricks¹¹¹ recommends sheep grazing to diminish the number of ticks, for the reason that ticks die upon sheep, and many of the engorged females are not fertilized on account of the difficulty experienced by the males in propelling themselves through the thick wool in search of the females.

The serum of convalescent animals is protective. Noguchi¹¹² induced immunity in guinea-pigs with freshly prepared mixtures of spotted fever virus and immune rabbit serum in neutral or superneutral proportions. Spencer and Parker¹¹³ found that an emulsion of infected fed adult ticks treated with 0.5 per cent phenol will protect guinea-pigs against 1 c.c. of blood virus. In a further study¹¹⁴ they showed that it will also protect rabbits and monkeys. This vaccine has been administered to thirty-four adults to test its prophylactic value.

JAPANESE RIVER FEVER

Japanese river fever occurs along river bottoms with a preference for sandy soil. It is also called Japanese flood fever or tsutsugamushi disease, and has a similar clinical picture to typhus fever as well as to Rocky Mountain spotted fever. The mortality is about 20 per cent. The disease also occurs in Sumatra, Formosa and probably China. Sellards¹¹⁵ has grown rickettsia-like bodies from a strain of this disease from guinea-pigs.

¹¹¹ *Bur. Entomol. Bull.*, No. 105.

¹¹² *J. Exper. M.*, 1923, 38: 605.

¹¹³ *U. S. Pub. Health Rep.*, 1924, 39: 3027. ¹¹⁴ *U. S. Pub. Health Rep.*, 1925, 40: 2159.

¹¹⁵ *Am. J. Trop. Med.*, 1923, 3: 529.

The disease is transmitted by the bite of a very small, reddish, hairy mite, known as the Kedani mite, *Trombicula akamushi* (*Microtrombidium akamushi*). Field mice are the reservoir of the infection, and the mites live in great numbers on these rodents in the infected areas in Japan. A small area of necrosis results at the point of the mite bite and the neighboring lymph glands are enlarged.

RELAPSING FEVER

Relapsing fever comprises a group of specific infections caused by spirochetes in the blood, characterized by relapsing attacks of fever lasting about a week and recurring at like intervals. It is transmitted by various biting bugs: lice, ticks, bedbugs, etc.

Relapsing fever, also called famine fever, tick fever, and seven-day fever, is found upon all the five continents of the globe, with the possible exception of Australia. It was formerly epidemic where insanitary conditions were rife, especially in Ireland and Russia. In 1920, 1,570,604 cases were reported in Russia, without Ukraine. The infection prevails in India, where Vandyke Carter of Bombay made his classic investigations. In Africa it ranks next to malaria and sleeping-sickness. In India it is hardly less severe, and in Europe and America it is a mild disease. Relapsing fever was epidemic in New York and Philadelphia in 1869. It has not reappeared in epidemic form, but cases occasionally occur in various parts of the United States. The disease has receded from civilization where sanitation is practical and hygiene is observed.

The term "relapsing fever" includes a group of tropical, febrile infections caused by different, but very closely allied, spirochetes—*Spiroschaudinnia*, now *Borrelia*. The European relapsing fever is caused by *B. recurrentis* and transmitted by bedbugs and lice. The relapsing fevers of East and West Africa are caused by *B. duttoni* and transmitted by the tick (*Ornithodoros moubata*). The North African, or Algerian relapsing fever is caused by *B. berbera* and is transmitted by lice (*Pediculus humanus*, var. *capitis* and *corporis*). The relapsing fever of Asia and India is caused by *B. carteri* and is transmitted by lice. The American form is due to *B. novyi*, and the Persian form to *B. persica*. These various spirochetes resemble each other morphologically, but show a difference in pathogenicity to laboratory animals and are further distinguished from each other by specific agglutinins. Clinically the relapsing fevers resemble each other, except that some now are more virulent than others. Epidemiologically the disease resembles typhus fever; in fact, in Europe where relapsing fever prevails the doctors call it *typhus recurrens*.

Relapsing fever is characterized by sudden onset, intense frontal headache and pains of back and limbs. The fever continues from three to five days and falls by crisis. The temperature remains normal for about a week, when the fever repeats itself. There may be four or five such relapses, sometimes ten. The spirochetes are found in the peripheral blood only during the febrile period.

Obermeier in 1868 discovered the "spirillum" in the blood—*Spirillum*¹¹⁶ *obermeieri*, the cause of European relapsing fever. Carter and Koch in 1878 showed that the infection may be transferred to apes by the inoculation of the blood of a patient. Münch and Moczutkowski transferred the disease by the inoculation of relapsing fever blood to healthy individuals. Koch and also Dutton and Todd succeeded in demonstrating that the spirochetes of African relapsing fever multiplied in the tick (*Ornithodoros moubata*), and that the bite of this tick may convey the disease to healthy men. The spirochetes are found in the coxal glands and in the feces of the tick, which are rubbed into the wound made by the tick bite. The spirochete is infectious for the wild rat, and the tick *Ornithodoros talaje* occurs on rats. Other insects, as bedbugs, fleas, biting flies, and lice, may convey the infection. Lice do not infect by biting, but through their feces or when the insects are crushed and the infection rubbed into a scratched surface of the skin.

Leishman¹¹⁷ has demonstrated that *Borrelia duttoni* may be transmitted "hereditarily" in the tick. He has obtained positive results in the second generation, the bites of which were infective for mice and monkeys. Attempts to carry the infection to the third generation in the tick have so far failed. Leishman considers the hereditary transmission of the infection as biological evidence that the spirochetes belong to the protozoa rather than the bacteria.

Schuberg and Manteufe¹¹⁸ found that a temperature of 22° C. is not favorable for the spirochete in *Ornithodoros moubata*. This was shown by experiments upon rats in which the infection through the bite of the tick disappeared more quickly at 22° C. than at higher temperatures.

One attack protects against subsequent attacks. Second attacks occur among Negroes in Africa after years but are very light. The only susceptible animals are man, the apes, mice, and rats.

The prevention of relapsing fever is based upon sanitation of the environment and personal and domestic cleanliness and the avoidance of tick and other bug bites. Personal prophylaxis depends upon keeping aloof from vermin-infested places, especially where the disease prevails. Manson suggests that a mosquito net, a vermin-free bed well off the ground, and a night light are indispensable in Africa, where the nocturnal habits of *Ornithodoros moubata* render the hours of sleep especially dangerous. This tick has habits somewhat similar to those of a bedbug, and lives in cracks of the walls and floors of the native huts. Arsphenamin is a specific remedy.

Other diseases caused by spirochetes are rat-bite fever, syphilis, yaws, infectious jaundice, yellow fever (?), Vincent's angina; also certain types of bronchitis, ulcers, etc. Spirochetes are intermediate between bacteria and protozoa.

¹¹⁶ *Spirillum* was changed to *Spirochæta* and then to *Spiroschaudinna*; it is now *Borrelia recurrentis*.

¹¹⁷ *Lancet*, 1910, 1: 11.

¹¹⁸ *Ztschr. f. Immunitätsforsch.*, Orig. Bd. 4, 1910, 512.

LICE

Lice are degraded, wingless insects, and are divided into two groups according to their method of feeding. The *Mallophaga* include biting lice, like the bird lice which feed on the hair and feathers of animals, but do not suck blood. As far as is known, these lice do not transmit disease. The *Anoplura*, or sucking lice, feed upon blood, and are the group concerned in the transmission of infection. Lice do not travel much of their own accord, but keep fairly close to one host.

Human lice belong to three species: *Pediculus capitis*, head lice; *Pediculus vestimenti* or *corporis*, clothes or body lice; *Phthirius pubis*, pubic or crab lice. Nuttall ¹¹⁹ and Bacot ¹²⁰ regard the head louse and clothes louse as races of the same species, and so Nuttall united them under the title *Pediculus humanus*, designating the head louse as *capitis* and the clothes louse as *corporis*.

Life History.—The life histories of *Pediculus capitis*, *P. corporis*, and *Phthirius pubis* are similar, in that the insects, after emerging from the egg, undergo three molts before attaining sexual maturity.

The eggs or nits are laid on fibers of clothing or body hair. They prefer to lay eggs on rough material such as felt, wool or flannel, but will deposit eggs on silk. The eggs are ovoid, about $\frac{1}{25}$ of an inch long, with a granulated cap or operculum; they are firmly cemented to the hair. The freshly laid egg is almost transparent, but as the embryo develops it assumes a yellowish color. The empty shell is hard and remains attached after the louse has emerged. The shell and the cement is resistant to chemicals, no solution will remove it without first destroying the hair or fiber to which it is attached. At the temperature which ordinarily exists between the skin and the clothing, the eggs hatch in from seven to ten days, but if kept in a cooler atmosphere, the incubation period is lengthened. The first molt occurs after two days; the second, two days later; and the third, after three days. A complete cycle from egg to egg takes about sixteen days. There is no grub (larval) stage as in fleas.

Oviposition in *Pediculus humanus* commences twenty-four to thirty-six hours after the emergence of the female from the third larval skin. The number of eggs laid depends upon the food supply and the temperature at which the female is maintained. Under optimum natural conditions, the louse lays four or five eggs a day for four to five weeks. Bacot ¹²¹ states that a female louse under ideal conditions might have four thousand offspring during her lifetime. The average life of a louse is from thirty-five to forty days, probably a little less for the males. Development of eggs is eight days at 32° C., which is the optimum temperature. This period may be lengthened or shortened by varying the temperature. Therefore, persons who remove their clothing at night will become less heavily infested than those who wear their

¹¹⁹ *Parasitology*, 9: 293; 10: No. 1, No. 4, 375-382, 383.

¹²⁰ *Brit. M. J.*, 1916, 1: 788.

¹²¹ *Parasitology*, 1917, 9: 228; *Proc. Roy. Soc., Med.*, 1917, 10: 61.

clothing continuously. The periodic cooling of the clothing and contained lice leads to their progeny being materially reduced.

Lice feed immediately after emerging from the egg. A young louse will die within twenty-four hours if no food is obtainable, while a well fed louse can live ten days away from its host. Lice feed many times during the day. They feed most frequently at night when the host is at rest. When lice become ravenous with hunger they feed to excess and may rupture their intestinal canal. The louse depends upon the salivary secretion to dilate the capillaries so that blood flows freely. While feeding, the greedy insect passes excreta, which contains a large proportion of undigested red blood cells.

Spread of Infestation.—Vermin infestation is spread either by contact with infested persons themselves, their clothing or their personal effects. One vermin-infested man may spread lice to many of his associates. The soldiers abroad believed that trenches and dugouts were “lousy” and that they obtained their infestation from them. This was not exactly true, for the infestation was obtained owing to the overcrowding in these places. Lice desert their host when the person has fever or dies.¹²² In the first instance the excessive heat drives them off, and in the latter the lack of food supply. Lice may be dislodged by brushing and so fall to the ground. It has been found that lice buried at a depth of four inches will crawl to the surface. They may be blown by wind. Lice are ordinarily not found in bedding and blankets unless recently occupied by vermin-infested individuals. Clothes lice will migrate at night from one pile of clothes to another. They may come into the house with the family laundry, or be transferred from one person to another in sleeping cars, hotels, crowded street cars, and the like.

Lice are most often found in those parts of the garment which are in closest contact with the body, such as the fork of the trousers, waistline, armpits and neck. They are found in the inner as well as the outer garments. Lice may be found on any garment or article worn by an infested man. In conducting inspections for lousiness, it is important to remember this, and to bear in mind that the body louse may lay its eggs on the hair of the head as well as any other hairy part of the body. This is of importance, and neglect of it leads to unsatisfactory results. The delousing of clothing alone is not sufficient.

Clothes or Body Lice.—*Pediculus corporis* is often called the “body louse” or “clothes louse,” or the “grayback” of Civil War days, or “cootie” in the World War. This louse, also known as *Pediculus vestimenti*, is a parasite which depends upon human blood for sustenance and man’s body and clothing for prolonged life and reproduction. The size varies with its maturity; a newly hatched louse is about the size of a pinhead, while a full grown, well fed louse is about one-sixth of an inch in length. The louse has a smooth, hard, chitinous covering, which is impenetrable to most chemicals.

The body is divided into head, thorax and abdomen. At the sides of the

¹²² Observed by Plotz during typhus fever epidemic in Serbia and Bulgaria, 1915-16.

head are two antennæ, the mouth has a long sharp stylet or stabber which is used for puncturing the skin. This stylet consists of three parts which are so formed as to make a hollow tube through which the blood flows. Attached to the thorax are six legs which are joined, and at each end is a single large claw. The abdomen is divided into six or eight segments. The terminal one is indented in the female and rounded in the male. The abdomen of the female is broader than that of the male. There is some evidence that there are more females than males. Both sexes bite and convey disease.

Head Lice.—*Pediculus capitis*, or the head louse, is perhaps the commonest variety of louse in civil life. Sobel¹²³ states that about 22 per cent of

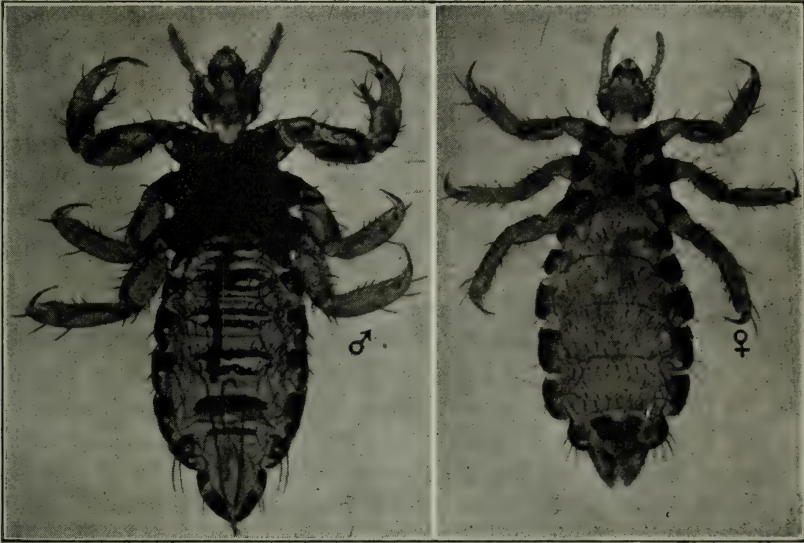


FIG. 40.—*PEDICULUS HUMANUS* VAR. *CORPORIS*.

the school children in New York are infested, some 150,000 to 185,000 cases being reported during the years 1909 to 1912. *P. capitis* shows no material difference in its biology from *P. corporis*. It lays fewer eggs and is perhaps shorter lived. It is found mostly in children, especially in girls on account of their long hair, and in old people. It is fond of the temporal and occipital regions. Although the hairy head is the common habitat, still it may be found on other parts of the body, in which case it would be difficult to say whether it was *corporis* or *capitis*. This insect is spread by contact such as occurs in schools, and by brushes, combs and hats. The common clothes-hook in school houses may be a method of transfer. If hair is worn short infestation rarely occurs. It has been reported by Goldberger¹²⁴ that the head louse may transfer typhus fever.

An effective treatment is to anoint the head with a mixture of equal

¹²³ N. York M. J., 1913, 98: 656.

¹²⁴ U. S. Pub. Health Rep., 1912, 27: 297.

parts of kerosene and vinegar or kerosene and olive oil; phenol 2.5 per cent, or lysol 1 per cent, for one or two hours, may then be applied. The head must be wrapped in a towel over night and shampooed in the morning. The treatment may have to be repeated after about ten days to destroy lice which have hatched in the meantime, but usually the eggs are destroyed as well as the adult lice. Combing with a fine warm comb will remove the nits. In males the hair should be cut (see page 370).

Crab Lice.—*Phthirus pubis*, the crab louse, looks unlike *Pediculus* and presents the general appearance of a crab. It is about one-sixteenth of an inch long. It is usually found in the pubic and perianal region, but may be found over the abdomen and chest, axillæ and down over the thighs; even the eyebrows may be infested. The nit is laid near the base of the hair. *Phthirus* feeds almost continuously, and hence dies rapidly when removed. A female lays about twenty-five eggs in her lifetime. Eggs hatch in about seven days. Development is the same as *Pediculus*. This insect is transmitted mainly by contact in lodging houses, houses of prostitution, bath tubs, and from toilet seats. It has not been known to transmit disease. Treatment consists in shaving. If skin irritation is present apply some bland ointment. The use of blue ointment may cause skin irritation and therefore should not be so commonly employed.

Lice and Transmission of Disease.—Lice transmit typhus fever; they are also concerned in the transmission of relapsing fevers and trench fever, and perhaps in other infections. Lice most likely transmit disease by way of the excreta, the virus gaining entrance through the punctured wound made by the louse while feeding, or scratched in by the individual. Mueller and Urizio¹²⁵ were able to transmit typhus fever even without the bite of the insect. Mueller himself contracted typhus fever as the result of an accident, in which an emulsion of lice feces spilled on his hands. The period of incubation was seventeen days. It is possible that syphilis and other infections may be transmitted mechanically by lice and other biting insects.

The effects of the presence of lice upon men differ according to individual susceptibility. Persons who are constantly vermin infested are immunized against the salivary secretion, and the local reaction to the bites is very slight. Sometimes only a slight puncture wound is discernible. In persons who have never been lousy before, the local reaction is intense and indicated by an urticarial wheal or hemorrhagic spot. Lice bites in themselves may cause a mild febrile reaction and a generalized eruption resembling measles.¹²⁶

Delousing.—The best delousing methods should not only destroy lice and their eggs, but also the viruses transmitted by the insect. Fortunately, the viruses of relapsing fever, typhus fever and trench fever are comparatively frail; they are destroyed at 70° C. moist heat for thirty minutes. Lice and nits are killed at this temperature; also at 55° C. dry heat in five minutes. It is a comparatively simple matter to kill lice and their nits—only the

¹²⁵ *Riforma med.*, 1919, 35: 734.

¹²⁶ *Arch. Int. Med.*, 1919, 24: 419.

administrative aspect of the problem presents any real difficulty. The most frequently used methods are heat, chemicals or storage.

Heat.—Dry heat is convenient, but not effective in large-scale operations because it lacks the power of penetration. Dry heat has the advantage in the case of leather material and rubber goods, which may be thus disinfested without injury. Shoes are rarely infested; in fact, leather and rubber articles as a rule need not be treated except in the presence of heavy infestations or an epidemic. Steam penetrates better, is quicker, surer; and it also disinfects.

Heat may be applied in a great variety of ways: boiling water, Serbian barrel, steam, flat iron, hot oven, hot-air boxes or huts, steam disinfectors.

The *flat iron*, which was employed in some of the armies in the World War, is effective, but impracticable on a large scale as it is time-consuming. If used in connection with a delousing plant, the number of ironers would have to equal the number of bathers so that the clothes would be ready for the men when they came out of the bath. It takes about fifteen minutes to iron a uniform and underclothes.

Hot ovens have been used. This method may be applied on a small scale. Care must be observed not to scorch the clothing. A very effective hot-air method is the hot-air hut, described by Captain Orr of the Canadian Army.¹²⁷ The penetration with dry heat is not as complete as when using steam. Stagnant hot air is less effective than circulating hot air. In the front area, in dugouts and trenches, the hot box¹²⁸ may be used. This box is based on the principle of the fireless cooker, and used by heating a piece of metal, and placing the clothing, which is protected from coming in contact with the metal, over it. The temperature obtained is sufficient to destroy lice and eggs.

Hot Water.—Lice and eggs immersed in water at 70° C. for thirty minutes are killed with certainty; this method is not practical on a large scale.

Steam Disinfectors.—The large commercial steam disinfectors are the best and quickest way of delousing large quantities of clothing. With the aid of the vacuum, penetration is sure and the materials are both disinfested and disinfected. Steam may also be used in improvised methods in a freight car, obtaining the steam from the locomotive; or in the compartments of a ship.

The *steam laundry* is a good delousing apparatus, provided live steam is run into the wash wheel. It takes about fifteen minutes to insure a proper temperature.

The *Serbian barrel* is one of the best improvised methods for delousing with steam. It was used during the typhus campaign in Serbia and Bulgaria. It consists of a large barrel, the bottom of which is freely perforated, while the top is removed and replaced by a weighted lid. At the lower end is a sand bag collar to prevent the escape of steam, which enters the barrel from a metal boiler upon which it rests, both barrel and boiler being imbedded at their junction in the brick work forming the furnace. The furnace may be

¹²⁷ *Parasitology*, 1918, 10: 441.

¹²⁸ Devised by Harry Plotz, War Department.

made long and narrow, with a chimney at one end, and the boilers and barrels placed in series. It is important that steam be generated rapidly. The clothing to be deloused is put in at the top and the lid placed on tightly. After the steam is generated, the clothing remains in the barrel for one hour. All clothing, except leather material, rubber and celluloid, may be handled in this manner.

Storage.—The lice starve and die on clothing hung in storage. Nuttall¹²⁹ advises clothing stored in a dry temperature for two to three weeks. Since

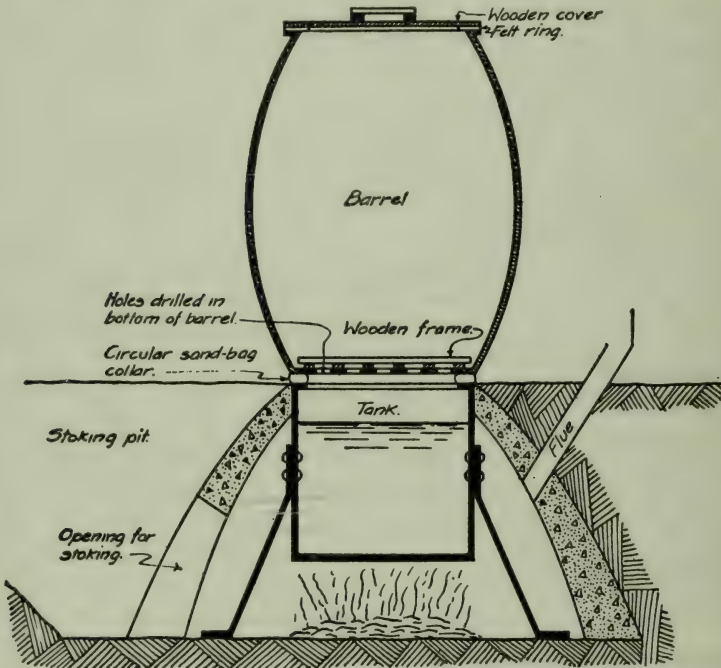


FIG. 41.—SERBIAN BARREL.

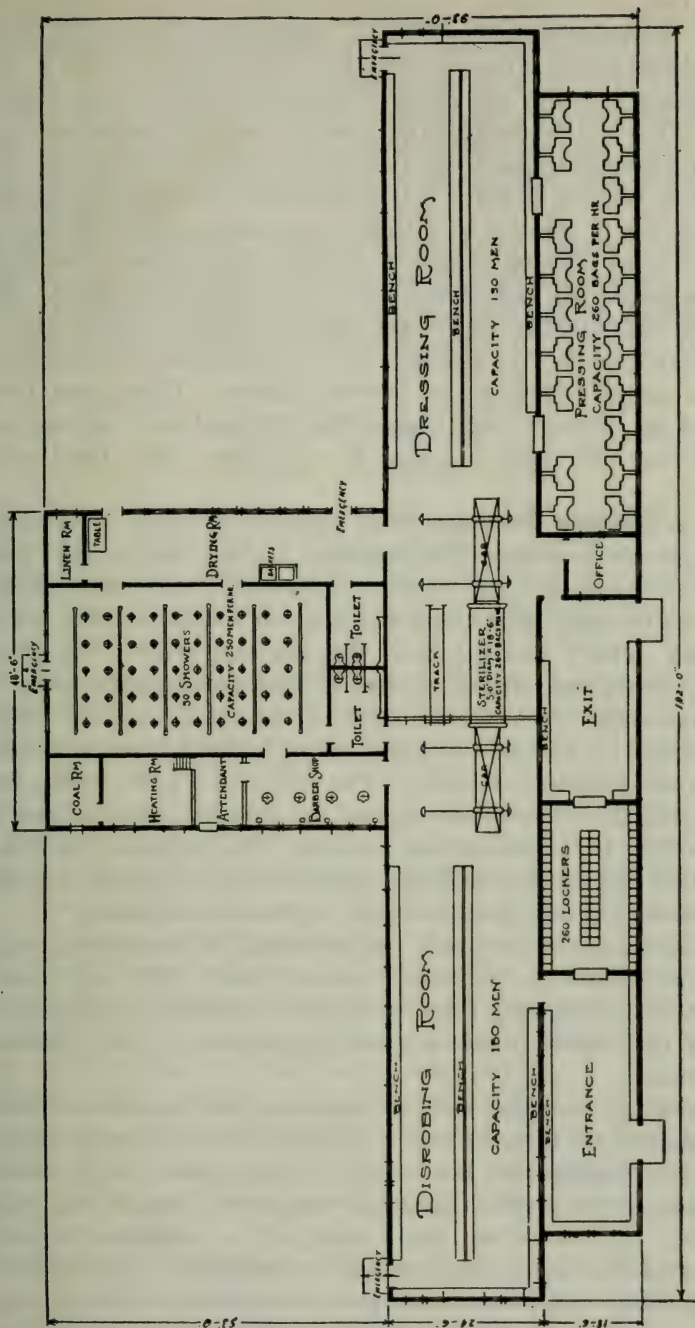
hatching has been delayed thirty-five days by low temperature, it would be safe to allow thirty to forty days during cool weather.

Chemicals are used for two purposes; to destroy lice and nits on clothing and hair, or to repel them. Repellants for the most part are disappointing and experience has shown that most of them are worthless. Some may destroy lice, but rarely do they destroy nits.

The most effective substance for delousing is kerosene; naphthalene, carbon disulphid and ammonia are also effective. Powders, such as the N. C. I. powder of the English, consisting of naphthalene 96 per cent, creosote 2 per cent, and iodoform 2 per cent, are irritating and not always effective.

Various *fumigation methods* have been recommended, but experience has

¹²⁹ *Parasitology*, 1917, 9: 293; 1918, 10: 1, 4, 375, 383.



AREA 9945 SQ. FT.

FIG. 42.—DELOUSING PLANT.

shown that most of the gases, as sulphur dioxid and formaldehyd, do not destroy nits. Hydrocyanic acid gas, in the proportions of 10 ounces of sodium cyanid per thousand cubic feet, is sufficient to kill adult lice and to prevent eggs from hatching, when the clothes are freely exposed to the fumes for two hours. When clothes are tightly packed in trunks, 6 ounces per hundred cubic feet with twenty-five inch vacuum should be used for two hours. Special apparatus with vacuum to force the hydrocyanic acid gas into packages to destroy insect life is effective in killing bollworms in cotton bales and will also destroy lice in clothing packed in trunks. The method is valuable because the packages are undisturbed and the contents uninjured. Success depends upon concentration of the gas, degree of initial vacuum and length of time of exposure.¹³⁰ As this gas is very dangerous, it should not be employed in camps. It does not destroy bacteria. Chlorpicrin has been recommended by Moore,¹³¹ who states that it penetrates clothing and is said to kill lice and eggs. He uses 4 c.c. to one cubic foot for thirty minutes.

Lice as a Military Problem.—The control of vermin infestation and louse-borne diseases was one of the important sanitary and medical problems of the World War. As an army problem, the unprecedented scale of the war, combined with the conditions under which it was fought, led to a prevalence of lice among soldiers that has never been equaled before. No army was spared from widespread vermin infestation, and from typhus fever, trench fever and relapsing fever. Lice disturbed the morale of the soldier by causing irritation of the skin and scratching, often followed by infection, loss of sleep and impaired efficiency. The loss of life from typhus fever in Serbia and Roumania was enormous, and the non-effective rate from trench fever was so high as to cause serious concern. The Germans found that the best protection against lice and the diseases they carry was to establish elaborate delousing plants through which the troops were passed.

Vermin infestation in our army was prevalent. It is said that practically every man that reached the front area became infested. Any large assemblage of soldiers is likely to contain a few verminous individuals, who are primarily the cause of the trouble, which is greatly aggravated by the crowding and camp conditions.

The method of excluding vermin infestation and louse-borne disease by troops returning to the United States was handled in the following manner:¹³² All troops were detained for two weeks at foreign ports before boarding a transport, during which time contagious disease was isolated and delousing was practiced. All troops were again examined on transports as soon after departure as possible, and reëxamined again after six days. During the intervening time, each soldier was instructed to examine his clothing daily for vermin. On arrival in the United States, civilians were carefully kept from

¹³⁰ *U. S. Pub. Health Rep.*, 1925, 40: 335.

¹³¹ *J. Lab. and Clin. Med.*, 1917, 3: 261.

¹³² War Department Order.

coming in contact with soldiers. All soldiers were sent to the nearest camp, where delousing was again practiced. An area was set aside for the receipt of "unclean" troops, and following delousing they were placed in a clean area. Up to July, 1919, about 2,000,000 troops were returned to the United States, and not a single case of a louse-borne disease was introduced into this country, and vermin infestation was not carried into civil communities by demobilized soldiers.

The delousing plant¹³³ devised by Major Harry Plotz is shown in Figure 42. It can handle 200 soldiers and their equipment an hour. The United States Public Health Service also has efficient plants at its maritime quarantine stations, and traveling railroad equipment for work on the Mexican border.

TYPHUS FEVER (*Typhus exanthematicus*)

Typhus fever is an acute specific infection caused by *Rickettsia prowazeki* and transmitted by lice. It is characterized by sudden onset, a macular or hemorrhagic rash which appears on the third to fifth day, and a continued fever lasting about two weeks which usually ends in crisis. The period of incubation is from five to twenty days, with an average of twelve. Typhus fever prevails during cold weather; the disease dies down in summer. One attack confers a definite immunity, second attacks being very unusual.

Synonyms.—Typhus fever is also known as hospital fever, spotted fever, jail fever, camp fever, ship fever, and in Mexico as tabardillo. In Europe physicians say *typhus abdominalis*, *typhus exanthematicus* and *typhus recurrens* to distinguish typhoid, typhus and relapsing fever, respectively.

History.—Typhus fever was formerly confused with typhoid fever. Louis in 1829 named typhoid fever, but it remained for one of his pupils, Gerhard, clearly to lay down the difference between the two diseases. Previous to that time, typhus fever was prominent and prevailing, while typhoid fever was unknown as such and probably did not occur in great epidemics. Up to the time of the World War, the situation was reversed; typhoid fever had become pandemic, while typhus fever had receded with civilization and improvements in sanitation. Since the beginning of the World War, epidemics of typhus fever have occurred in Serbia, Bulgaria, Turkey, Russia, Poland, Germany and Mexico. The history of this disease has been repeated again. War followed by poverty and distress associated with vermin infestation favors the spread of typhus fever.

Distribution and Prevalence.—Typhus fever is a disease of cool and temperate climate, and is found in Europe, Asia, Africa and America, the most common foci being Russia and the Balkans, Ireland, Poland, Galicia, Spain, Italy and Mexico. In ten years, 1871-1880, in Ireland 7,495 deaths were reported from typhus fever; in three years, 1909-1911, the number had

¹³³ The details of construction and operation are described in the *J. Am. M. Ass.*, 1919, 72: 324.

fallen to 143. It is reported that Serbia lost 150,000 people from this disease alone during the winter of 1915-1916. In five years, 1919-1923, 6,995,178 cases were reported in Russia, without Ukraine.

Typhus fever prevailed in epidemic form in the United States in New York in 1881-82, and again in 1892-93, and in Philadelphia in 1883. Since then, except for a few sporadic cases in immigrants, the disease was believed to be non-existent in the United States. In 1896 Brill reported cases of an unknown fever in New York, which was called Brill's disease until it was determined that the cases were nothing more or less than mild typhus fever. It is now evident that the disease did not die out, but has been endemic in mild form in New York, Boston and other seaports along the Atlantic Coast, as well as in Southern California and along the Rio Grande.^{133a}

Typhus fever, when prevalent in epidemic form, has been said by the older writers to be one of the most highly contagious of febrile diseases. Before the mode of transmission was understood, doctors and nurses and others in close contact with the disease were almost invariably stricken. Typhus fever claims more victims in the medical profession than any other epidemic disease. The sad case of Ricketts, who lost his life in endeavoring to unravel this pathologic puzzle in Mexico, is still fresh in our minds. Lest we forget, others who have sacrificed their lives in studying this disease are Donnelly and Macgruder in Serbia, Husk in Mexico, Jochmann and Prowazek in Germany, Bacot in Egypt, and Conneff, Cornet, Luthje and Schussler.

Etiology.—*Rickettsia*.—*Rickettsia* represent a new group of microorganisms. They are small, bacteria-like bodies, possibly belonging to the Protozoa. They stain with difficulty; best with Giemsa or one of the modifications of Romanowski's stain. *Rickettsia* occur in pairs and clusters within the intestinal cells of the insect in typhus and Rocky Mountain spotted fever, and without the intestinal cells in trench fever. They are small: a pair measures about one μ long. They do not grow in artificial culture media, but have been cultivated in tissue. They are frail and easily killed. This is an interesting instance of a great group of microorganisms primarily adapted to insect tissues, in that they show great specificity for their insect hosts. There are occasional representatives which have become pathogenic for man. This interesting and important group of microscopic parasites is just being disclosed, and our knowledge of them is therefore meager.

Rickettsia prowazeki found in typhus fever and *Dermacentroxenus rickettsi* found in Rocky Mountain spotted fever differ from *Rickettsia pediculi*, associated with trench fever, in that the former are found especially in the epithelial cells of the alimentary canal of the louse, while the latter occurs only in the lumen of the alimentary canal, and not in the cells of the louse.¹³⁴ *Rickettsia prowazeki* may harm the louse by disabling so many gastric cells that the insect may starve to death, dying in about three or four weeks, whereas ordinarily they live three to four months.

^{133a} Maxcy, U. S. Pub. Health Rep., 1926, 41: 1213.

¹³⁴ J. Am. M. Ass., 1925, 84: 723.

Rickettsia prowazeki is now believed to be the cause of typhus fever. It was named by Da Rocha Lima¹³⁵ in memory of Ricketts and Prowazek, both of whom succumbed to typhus while investigating the disease. The virus as it exists in the circulating blood does not pass through a Berkefeld filter. It is not killed by freezing for eight days, but is deprived of virulence by heating at 55° C. for fifteen minutes.

Wolbach¹³⁶ states that the pathology of typhus fever consists largely of lesions of the blood-vessels. There is disintegration of the endothelium and proliferation causing thrombi and hemorrhages, also perivascular infiltration with cells. These lesions in the brain account for the stupor and delirium, which are prominent symptoms in typhus fever. Similar lesions of the blood-vessels of the skin produce the macular and hemorrhagic eruption. The thrombi give rise to gangrene which is often symmetrical.

There is a puzzling relationship between *Rickettsia prowazeki* and *Proteus X* in connection with the etiology of typhus fever.

The Weil-Felix Reaction.—The Weil-Felix reaction depends upon the agglutinating property of serum upon certain strains of the proteus group isolated in 1915 by Weil and Felix from the stools and urine of typhus fever patients, and designated by them X₂ and X₁₉. The proteus-like bacillus is found associated with typhus fever, but is not the cause of the disease.¹³⁷ The reaction in typhus fever appears early, usually before the sixth day, and therefore has diagnostic significance.

The Experimental Disease in Animals.—The disease may be transmitted by blood inoculations to man, chimpanzees, lower monkeys and guinea-pigs. The reaction in animals is quite typical, characterized by an incubation period which varies from seven to ten days in guinea-pigs, and rise of temperature from seven to eleven days, usually ending by crisis. An animal that has gone through such a reaction is immune. Many guinea-pigs are naturally immune.

Transmission.—Tobias Coberus, in the beginning of the seventeenth century, associated the louse with typhus fever, but Nicolle, Compté and Conseil, in 1909, were the first to report the transmission of typhus fever by the bite of the louse (*P. vestimenti*). Their work was suggested by the successful transmission of relapsing fever through lice, by Sergeant. Since the work of Nicolle and his collaborators, their results have been confirmed by Ricketts and Wilder, Anderson and Goldberger, and others. Goldberger has shown that the head louse (*P. capitis*) may also transmit this infection. The period of infectivity in lice, following exposure on typhus patients, is reported to vary from two to ten days by different observers. *Rickettsia* bodies have been found in the stomach wall of typhus infected lice by Prowazek, Da Rocha Lima, Wolbach and others.

¹³⁵ *Deutsch. med. Wchnschr.*, 1919, 732.

¹³⁶ *Int. J. Pub. Health*, 1920, 1: 2; *J. Med. Research*, 1919, 41: 197; *J. Infect. Dis.*, 17: 1. Report of the Typhus Fever Research Commission of the League of Red Cross Societies to Poland, Harvard University Press, Cambridge, Mass., 1922.

¹³⁷ *Wien. klin. Wchnschr.*, 1916, 29: 2.

The feces of typhus-infected lice are infective for animals. The disease is most likely introduced through the feces, the virus gaining entrance through the punctured wound made by the louse while sucking, or scratched in by the host.

Since the transmission of the disease by the louse has been shown, we can understand why great epidemics of typhus fever occurred in the dark, dirty ages, and also why it prevails in epidemic form only in overcrowded, filthy, unhygienic surroundings, and the truth is readily understood of the oft quoted sentence of Hirsch that "the history of typhus is the history of human wretchedness."

Prevention.—The disease has been greatly decreased in civilized centers with a diminution of lousiness. The prevention of typhus now focuses itself upon the eradication of the body louse—a question of personal cleanliness. Typhus fever is also a social problem in that it is so closely interwoven with squalor, ignorance and poverty. Measures primarily directed to the destruction of lice and their eggs are described on page 362.

In the presence of a typhus epidemic, a separate typhus hospital should be established and a separate building or enclosure for typhus contacts. When a case of typhus fever is discovered, the patient should be removed to the typhus hospital, never treated at home. All the clothing should be removed in the receiving ward, and all articles of apparel taken away. This material should be disinfected by steam and placed in a storeroom; never allowed to be taken into the ward. The hair of the head, axillary and pubic region should be clipped and carefully collected and burned. The patient is then bathed, using warm water and a kerosene soap mixture. Patients can be rapidly handled if the receiving department is divided into a receiving room, barber shop, bathroom, drying room, and examining room. The patient is given clean pajamas in the examining room and then sent to the ward. Stress is laid on this procedure of admitting patients, in order to keep the ward safe.

Daily vermin examinations are made on the patients and in the bedding, because lice wander from the host during fever. This, no doubt, accounts for the high incidence among physicians and nurses. All attendants in the wards and all nurses and doctors should wear louse-proof suits. This is a one-piece garment made of heavy muslin, which goes over and covers the shoes and is tied about the neck. A hood may be attached. Cotton gloves are sewed in the sleeves by machine stitching. Nurses should wear the same garment with a skirt. The openings should be closed with a broad strip of adhesive plaster. A sterilized garment should be put on each time the attendant, doctor or nurse enters the ward. On leaving the ward, the garment is removed by an attendant, preferably a typhus immune, placed in a bag and steamed. A shower room and clothes closet should be provided near the exit of the ward, where nurses may change their costumes. Under no circumstances should visitors be allowed into the ward. In typhus-stricken countries, one must be extremely cautious of the attendants. As these people

usually come from the peasant class, where the epidemic prevails, their habits of cleanliness are usually very primitive. They must be taught the value of cleanliness, supplied with proper living quarters and repeatedly inspected for vermin. They should be isolated during the epidemic. It is preferable to have typhus immunes as attendants in the hospital.

The room from which the patient is removed should be fumigated with sulphur, or, better, hydrocyanic acid gas, and cleaned with kerosene, and should then be closed for two weeks if possible. A portable steam disinfector is useful to disinfest and disinfect fabrics, etc., in a typhus campaign. Carriages and wagons which carry patients to the hospital should be carefully inspected for lice and cleaned. If patients are removed on trains, the cars should be cleaned by vacuum cleaner and washed. During an epidemic, all plush on railway cars should be removed; abroad only freight cars or third-class cars are employed.

All contacts should be isolated for fourteen days at least; twenty-one days would be better, in a separate building or camp, being carefully deloused before entering. They should be directed to examine their clothing daily for lice and should be inspected for vermin by medical officers daily. Their temperature should be taken twice daily and daily inspections made for the first symptoms of the disease.

An educational campaign should be carried on in the cities by lectures in public places and schools, pamphlets, sermons and moving pictures. As typhus fever is usually associated with ignorance, poverty and distress, sanitary instructions as well as proper clothing and food should be supplied the stricken people.

The fact should be kept constantly in mind that the louse is the focus of the epidemic campaign, just as the mosquito is for the prevention of malaria, and our efforts towards prophylaxis should be concentrated on this point. Even with the knowledge of the mode of transmission of typhus fever, individual prophylaxis is still somewhat difficult, especially where infected insects abound in thickly populated centers.

No satisfactory vaccine or serum is available for protection.

Quarantine for typhus is discussed on page 534; delousing immigrants and troops (page 366).

TRENCH FEVER

Trench fever is a specific infection due to a filtrable virus transmitted by the clothes louse *Pediculus humanus*, var. *corporis*. The disease is characterized in its early febrile stage by recurrent pyrexia, headache, giddiness, a slow pulse in comparison to the degree of fever, sweating, polyuria, and a moderate leukocytosis at the height of the fever. Trench fever is never fatal, and complete recovery usually takes place. A certain percentage of the patients, however, pass into a stage of chronic ill health; that is, they suffer with recurrent pains in the limbs, headache and nervous manifestations, such as mental depression, excessive tendency to sweating, disordered action of the heart, mild

degree of anemia, and some loss of weight. The infection in some cases is very persistent, and acute febrile relapses may occur after months of quiescence. The period of incubation is fourteen to thirty days.

The Trench Fever Commission of the American Red Cross¹³⁸ concluded, as a result of extensive researches, as follows:

1. Trench fever is a specific infectious disease; it is not a modified form of typhoid or paratyphoid fever, and is not related, from an etiological standpoint, to these diseases.

2. The organism causing the disease is a resistant, filtrable virus.

3. The virus causing trench fever is present particularly in the plasma of the blood of trench fever cases, and such plasma will produce the disease on inoculation into healthy individuals.

4. The disease is transmitted naturally by the louse *Pediculus humanus*, Linn., var. *corporis*, and this is the important and common means of transmission. The louse may transmit the disease by its bite alone, the usual manner of infection, or the disease may be produced artificially by scarifying the skin and rubbing in a small amount of the infected louse excrement.

5. A man may be entirely free from lice at the time he develops trench fever, the louse that infected him having left him some time previously as its host, and the louse need only remain upon the individual for a short period of time in order to infect him.

6. The virus of trench fever is also sometimes present in the urine of trench fever cases, and occasionally in the sputum, and the disease may be produced in man by the introduction of the virus in the urine or sputum through the scarified or otherwise abraded skin.

7. Since the urine and sometimes the sputum of trench fever patients are infective, these should be sterilized in order to avoid the possibility of accidental infection from them.

8. In order to prevent trench fever or limit its spread, and thus save man-power for the armies, greater efforts must be made to keep soldiers in general from infestation with lice.

The method of transmission of trench fever was still further elucidated by the work of Byam and his associates on the British Trench Fever Commission.¹³⁹ They found that biting by infected lice was not of itself sufficient to transmit the disease, but that the excreta of such lice, rubbed into the scarified skin, would do so with great regularity. The excreta were found to be infective only after an interval of seven days or more after feeding on the trench fever patient; this fact suggests the possibility that the organism undergoes a development cycle within the louse. Lice were found to remain infective for a period of at least twenty-three days after feeding on the

¹³⁸ *Trench Fever*, Report of Commission, Medical Research Committee, American Red Cross: Major Richard P. Strong, Major Homer Swift, Major Eugene L. Opie, Captain Ward J. MacNeal, Captain Walter Baetjer, Captain A. M. Pappenheimer, Captain A. D. Peacock, and Lieutenant David Rappoport, Oxford University Press, 1918.

¹³⁹ *J. Am. M. Ass.*, 1918, 71: 21, 110, 188.

patients. These experiments indicate that infection in nature probably occurs by rubbing in the louse excreta in the process of scratching.

The cause of trench fever is believed to be *Rickettsia pediculi*. They occur in the intestinal tract, but not in the epithelial cells of the gut of the louse. The lesions of the disease are largely vascular, similar to those described for typhus fever and Rocky Mountain spotted fever.

Trench fever is a war disease. While it is not fatal, the morbidity resulting from it exceeded that from any other disease on the western front in the World War. No epidemics of the disease have been reported in civil life.

The prevention of trench fever resolves itself primarily upon an attack against lice. In addition, urine, sputum and other secretions are infective and disinfection must be practiced. Otherwise, prevention in all essential particulars is a parallel of that of typhus fever.

BEDBUGS

The bedbug has been carried by man to all parts of the inhabited world. It has become a true domesticated animal and has accommodated itself well to the environment of human habitations. The bedbug has no wings and a very flat body, which enables it to hide in the narrowest chinks and cracks of beds and walls. It may subsist for incredibly long periods of time without food. It is nocturnal in its habits. There are a number of species of *Cimex*, two of which attack man: one is the common bedbug, *Cimex lectularius*, found in all temperate climates; the other is the tropical or Indian bedbug, *Cimex hemipterus (rotundatus)*, prevalent in many tropical countries, including southern Asia, Africa, the West Indies and South America.

The pronounced odor of this insect is produced by certain glands opening on the back of the abdomen in young bugs and on the under side of the metasternum in the adults. The odor is common to most members of the group to which this insect belongs. It is useful in plant bugs, protecting them from their enemies.

The bedbug¹⁴⁰ undergoes an incomplete metamorphosis, the young being very similar to their parents in appearance, structure, and habits. The eggs are white, oval objects having a little projecting rim around one edge, and are laid in batches of from six to fifty, in cracks and crevices where the bugs go for concealment. The eggs hatch in a week or ten days and the young escape by pushing the lid within the projecting rim from the shell. At first the larvæ are yellowish-white, nearly transparent, the brown color of the more mature insect increasing with the later molts. During the course of development the skin is shed five times, and with the last molt the minute wing pads, characteristic of the adult insect, make their appearance. Marlatt found that under favorable conditions about seven weeks elapse from the egg to the adult insect, and that the time between molts averages about eight

¹⁴⁰ U. S. Pub. Health Rep., 1920, 35: 2964.

This form is characterized by the severity of the associated anemia. It is caused by *Leishmania infantum*.

Laveran believes that the so-called Indian and Mediterranean forms of the disease are identical. This view, however, is not shared by other authorities. Both these forms are generalized infections and had a high mortality (70 to 98 per cent) before the use of tartar emetic.

3. *Oriental sore* occurs throughout the tropical belt and passes under many names, such as tropical ulcer, Delhi boil, Aleppo boil, Biskra button, Bagdad sore, and espundia. Oriental sore usually occurs upon the exposed surface of the skin and starts as a papule, which subsequently ulcerates. The ulcer persists for months or years, finally healing spontaneously. It is thus a local and self-limited form of leishmaniasis. The parasite (*Leishmania tropica*) is found in great numbers in the ulcer, but apparently never becomes disseminated throughout the body. In Central and South America a form of leishmaniasis (espundia) occurs, which often extends to the mucous membrane of the nose and palate, causing extensive destruction of tissue with serious deformity. It is caused by *Leishmania americana*.

The parasite, for a time commonly known as the Leishman-Donovan body, is found chiefly in the endothelial cells of the liver, spleen, bone marrow and lymphatic glands. It also occurs in the white corpuscles of the circulating blood. It is a rounded or ovoid protozoön, and contains two deeply stained granules representing nuclei. This form represents the non-motile phase of the life cycle of a flagellate. It was first demonstrated by Leishman¹⁴² in 1900, from a case of "Dumdum fever." Donovan¹⁴³ in 1903, found similar bodies in cases in Madras. James Homer Wright¹⁴⁴ was the first to demonstrate the parasite in a case of Oriental sore, at the Massachusetts General Hospital, in Boston. The nature of these parasites was not understood until Rogers¹⁴⁵ succeeded in 1904 in cultivating them in citrated blood and showed them to be flagellated. Later on, Novy's method¹⁴⁶ for the cultivation of trypanosomes was found to be especially useful. On Novy's medium, the parasite increases in size, develops a single flagellum, and becomes actively motile. The medium known as N. N. N. (Nicolle, Novy, McNeal) is the easiest to prepare and gives the best results. It consists of agar (16 gms), NaCl (6 gms), and water (9,000 c.c.), which is distributed in tubes, sterilized and cooled to 40°-50° C., when one-third its volume of rabbit blood obtained by cardiac puncture is added.

No marked differences have been discovered between the three "species" of *Leishmania* in morphologic or cultural characteristics. Immune reactions are feeble or wanting, which corresponds to the action of other animal parasites. Noguchi has shown that the different species may be differentiated by

¹⁴² *Brit. M. J.*, 1903, 1, 1253.

¹⁴³ *Lancet*, 1904, 2: 744; *Brit. M. J.*, 1904, 2: 651.

¹⁴⁴ *J. Med. Research*, 1903, 10: 472.

¹⁴⁵ *Quar. J. Micr. Sc.*, 1904, 48: 367.

¹⁴⁶ *Contributions to Medical Research*, dedicated to V. C. Vaughan, Ann Arbor, Michigan, 1903, p. 549.

agglutination. A certain degree of immunity is present. Thus, Jewish physicians in Bagdad have long practiced inoculation of the virus upon the leg in order to prevent disfigurement of the face.

The period of incubation is irregular, and somewhat uncertain; in Oriental sore it may be months.

The precise mode of transmission of leishmaniasis is still in doubt. The disease is limited to man, except that it occurs naturally in dogs, especially in the region of the Mediterranean. It has been suggested that the dog flea, *Ctenocephalus canis* may be the intermediate host in this form of the infection. It may be transmitted experimentally to monkeys, dogs and mice. The disease can be reproduced in man and animals by inoculation of pure cultures, or tissue containing the parasites. It has long been observed that in regions where it is endemic, Oriental sore may follow injury to the skin, however slight, such as abrasions or insect bites.

Leishmaniasis is probably an insect-borne disease, although wound infection doubtless also occurs. Experiments designed to transmit the disease by blood-sucking insects have thus far proved unsuccessful. The development of the parasite has been observed in various blood-sucking arthropods. Patton¹⁴⁷ suspects the bedbug as the transmitter and finds that *Leishmania donovani* can develop into the flagellate stage in the digestive tract of the bedbug, although it survives for only a short time in this host. Shortt and Swaminath¹⁴⁸ infected a mouse with the dissected intestines of an infected bug.

Temporary development has also been found to occur in mosquitoes, but no development whatever takes place in lice or fleas. It is a difficult problem to differentiate the developing forms of *Leishmania donovani* from herpetomonads which naturally occur in these insects. Knowles, Napier and Smith¹⁴⁹ have demonstrated that *Leishmania donovani* passes into its flagellate form in the gut of the sandfly, *Phlebotomus argentipes*. Transmission experiments are now under way and it seems probable that the sandfly is the responsible vector. Isolation of a few hundred yards is sufficient to prevent the spread of the disease.

A great advance in the treatment and prevention of leishmaniasis was made when it was discovered that tartar emetic and other compounds of antimony are as specific for all forms of this infection as is quinin for malaria and asphenamin for syphilis. Vianna¹⁵⁰ in 1913 reported the effects of tartar emetic upon the disease occurring in Brazil. This was soon confirmed by Castellani,¹⁵¹ 1914, and many others. The treatment now commonly employed is the intravenous injection of chemically pure potassium or sodium antimony tartrate twice a week in 1 or 2 per cent solution, freshly prepared. The first dose is one-half grain (0.03 gm.) gradually increased to grains ii or iii

¹⁴⁷ *Scient. Mem. Med. Off. India*, Nos. 27, 31, 1907-08.

¹⁴⁸ *Indian J. M. Research*, 1924, 11: 965.

¹⁴⁹ *Ind. Med. Research Memoirs*, No. 4, Feb. 1926, p. 113.

¹⁵⁰ *Arch. brasil. de med.*, 2: 426; also, *Bol. de Soc. brasil de dermat.*, 1913, 2: No. 1.

¹⁵¹ *Report to the Advisory Com. for the Tropical Research Fund*, 1914; *Revista di Pediatria*, 1915, fasc. 4.

(0.06-0.12 gms.), until grains xxx (2 gms.) have been given, or until symptoms of poisoning appear.

The prevention of leishmaniasis consists first in early diagnosis and specific treatment. Such measures generally applied would greatly decrease the amount of the infection and its liability to spread. A warfare against bed-bugs, mosquitoes and other biting insects must be carried on, for even if some insect is not the intermediate host, the wound produced from a bite may be the point of entrance of the parasite which is very prevalent in the endemic centers. All wounds, however trivial, should be promptly treated and adequately protected.

It seems probable that leishmaniasis is an insect-borne infection and the sandfly is under suspicion. Preventive measures must therefore be directed toward the intermediate host.

It must be remembered that dogs around the Mediterranean basin have the infection and it is possible that children may contract the disease from them through their feces.

The parasites abound in the ulcers of the skin and intestinal mucosa, from which they may be discharged in great numbers. Infection through various forms of contact, direct and indirect, must be considered and guarded against. Instances are recorded in which Oriental sore has been transmitted indirectly through the use of a towel and other objects. Intestinal infection is also suspected. Houses should be disinfested and disinfected. The prevention of leishmaniasis is largely a question of cleanliness.

ROACHES

Roaches are among the commonest and most offensive of the insects which frequent human habitations. They are under suspicion of conveying several infections. There are no less than a thousand species of the family *Blattidæ*. Four of these have become domesticated and cosmopolitan. They are the American roach, *Periplaneta americana*; the Australian roach, *Periplaneta australasiæ*; the Oriental roach, *Blatta orientalis*; and the German roach, *Blatella germanica*. The first two resemble each other, except that the Australian roach is brighter and has a more definitely limited yellow band on the prothorax, and yellow dash on the side of the wings.

Blatta orientalis, or black beetle, is the common European and English species. The female is nearly wingless in the adult state. *Blatella germanica*, the German roach, has become world-wide in distribution: in this country it is styled the Croton bug. It is now the commonest and best known of the domestic roaches. It is light brown in color, and characteristically marked on the thorax with two dark brown stripes. It is the smallest of the domestic roaches, and multiplies more rapidly than any of them. It is also more active and wary than the larger species, and more difficult to eradicate.

Structure and Habits.—Roaches are smooth, slippery insects, in shape broad and flattened. The head is bent downward so that the mouth parts are

directed backward, and the eyes directly downward, conforming to their groveling habits. The antennæ are very long and slender, often having upward of one hundred joints. The males usually have two pairs of wings. The legs are long and powerful, and armed with numerous strong bristles or spines. The mouth parts are well developed and have strong biting jaws like those of a grasshopper, enabling these insects to eat all sorts of substances.

House roaches are particularly abundant in pantries, kitchens, bakeries, and other warm places. They are nocturnal in habits. Their numbers are often not realized unless they are surprised in their midnight feasts. Domestic roaches are practically omnivorous, feeding on almost any dead animal matter, cereal products and food materials of all sorts. They are believed to be cannibalistic. They eat or gnaw woolens, leather, and frequently damage the cloth and leather bindings of books. They soil everything with which they come in contact, leaving a nauseous, roachy odor. Food so tainted is beyond redemption. This odor comes partly from the excrement, but chiefly from a dark-colored fluid exuded from the mouth of the insect, and also in part from the scent glands. Occasionally they migrate, which accounts for the way in which new houses frequently become suddenly overrun.

The eggs are brought together within the abdomen of the mother into an egg capsule, which is a hard, horny pod. It is retained in this position sometimes for weeks, or until the young larvæ are ready to emerge. The young are very much like the adults, except in point of size, and in lacking wings. They pass through a number of molts, sometimes as many as seven. The development of the roach is slow, and probably under the most favorable conditions rarely more than one generation per year is produced. The abundance of roaches therefore depends upon their unusual ability to preserve themselves from ordinary means of destruction, and by the scarcity of natural enemies, such as mice, rats, guinea-fowl, tree frog, ichneumon flies, and other insectivorous animals.

The warfare against roaches consists of: (1) cleanliness; (2) elimination of breeding and hiding places; (3) fumigation; (4) poisons; (5) traps; (6) natural enemies. Scrupulous cleanliness and the keeping of food in jars or places inaccessible to the roach is of prime importance. All unnecessary corners, cracks and imperfections in the structure of the building that favor breeding and hiding places must be eliminated. Roaches may be killed with hydrocyanic acid gas, and also with sulphur dioxid—several fumigations are sometimes necessary. The best poison for roaches is sodium fluorid. It is, however, poisonous to man and has been the cause of accidental poisoning (see page 689). This substance very finely ground and mixed with meal of some sort forms the basis of most roach powders found upon the market. The powder should be liberally dusted in all corners, drawers, closets and runways. Sodium fluorid is only toxic to insects when taken internally; it is ineffective against bedbugs probably because that insect cannot be induced to eat it. The following is a good formula: sodium fluorid, four pounds; powdered licorice, eight ounces; powdered borax, one pound; pyrethrum, four

pounds; cornstarch, two pounds. Other poisons sometimes used are: pyrethrum powder, flowers of sulphur, phosphorus paste, containing 1 or 2 per cent of phosphorus in sweetened flour. Plaster-of-Paris, one part, flour three or four parts, may be set near a convenient flat plate containing pure water. The insects eat the mixture, become thirsty and drink, when the plaster-of-Paris sets and clogs the intestines. Many roaches may be trapped in a deep, smooth basin or jar. Sticks leading up to the rim of the trap make runways for the insects, which slip into the trap but cannot get out. The best bait for these traps is stale beer. The Croton bug is too wise to be thus trapped.

The roach has been shown by Fibiger¹⁵² to become infested with a round worm. When these infested roaches are eaten by rats, a cancer-like growth develops in the stomachs of the rats, apparently due to the irritating presence of these worms. Herms and Nelson¹⁵³ and also Longfellow¹⁵⁴ have shown the possibility of the conveyance of typhoid and other infections by means of the roach. When we consider that house roaches feed upon all kinds of breadstuffs, milk and its products, meat, clothing, cooked and raw food; that they migrate from one apartment to another, following water and drain pipes, from cellar and sickroom to living-rooms and bedrooms; that they infest kitchens, storerooms and toilets, opportunity is evidently offered to drag infection mechanically from one place to another. Roaches must therefore be regarded as a sanitary menace.

Barber¹⁵⁵ found that cockroaches feeding on the feces of cholera patients may harbor the cholera vibrios in the insect intestine and discharge them on food; Toda¹⁵⁶ has shown that such vibrios may be passed in the feces of the insect, even up to seventy-two hours after the infective feed. Other bacteria, amebæ and worms have been found in the dejecta of roaches.

REFERENCES

The literature upon insects and insect-borne diseases is very widely distributed.

Many of the entomological facts contained in this chapter have been taken from *The Insect Book* by L. O. Howard and the many excellent publications of Howard and his colleagues of the Bureau of Entomology, Department of Agriculture. The Government publications may be had upon application to the Superintendent of Documents, Washington, D. C. Many of the facts concerning the prevention and destruction of mosquitoes have been taken from articles in the *Public Health Reports* of the United States Public Health Service. In the chapter upon insecticides free reference has been made to my own book upon *Disinfection and Disinfectants*, as well as to my other writings and unpublished work in different phases of this subject. I have also drawn freely upon Chandler's excellent book, *Animal Parasites and Human Disease*. Other references are cited in the text. See also:

- FANTHAM, H. B., STEPHENS, J. W. W., and THEOBALD, F. V. *The Animal Parasites of Man*, New York, 1920.
 CASTELLANI, A., and CHALMERS, A. J. *Manual of Tropical Medicine*, London, 1919.

¹⁵² *Berl. klin. Wchnschr.*, 1913, 50: 289.

¹⁵³ *Am. J. Pub. Health*, 1913, 3: 929.

¹⁵⁴ *Ibid.*, 58.

¹⁵⁵ *Philippine J. Sc.*, 1914, 9: 1.

¹⁵⁶ *J. Hyg.*, 1923, 21: 359.

CHAPTER V

MISCELLANEOUS DISEASES

INFANTILE PARALYSIS

(*Acute Anterior Poliomyelitis*)

Infantile paralysis is an acute generalized infection, due to a filtrable virus, occurring both in epidemics and sporadically. The virus attacks the nervous system, with a special tendency to localization in the anterior horns of the gray matter of the spinal cord, hence the name anterior poliomyelitis. Gastro-intestinal symptoms and fever lasting three or four days often precede the paralysis, although a child may go to bed apparently well and wake up in the morning with paralysis and a slight fever. One attack confers a definite immunity; second attacks are not known.

From the standpoint of prevention, it is important to note that social and hygienic conditions apparently have no influence whatever in determining the infection. All classes are affected in about equal proportion. Sir Walter Scott gives a graphic account of his own case.¹

Historical.—Infantile paralysis has the earmarks of a new infection, although instances of sudden paralysis in babies, said to be due to carelessness of the nurse, are found in the literature of antiquity and some of these are assumed to have been cases of infantile paralysis. It is quite certain that the disease has become more and more common and more widespread of late years. The increase cannot be wholly accounted for by the fact that the disease is now better known and more readily recognized than formerly. Until quite recently, infantile paralysis was included in the textbooks with the organic nervous diseases, its infectious nature not being suspected.

Infantile paralysis is often called the Heine-Medin disease, from the fact that Heine² of Connstadt, in 1840, first established the disease as a clinical entity, and Medin,³ a Swedish physician, in 1890, was the first carefully to study an epidemic and to recognize various clinical types, as the cerebral, bulbar, polyneuritic, and ataxic forms. Bergenholtz, in 1881, described an outbreak of eighteen cases in Sweden with sufficient accuracy to establish its epidemic nature. The first outbreak described in the United States was reported by Caverly,⁴ in 1894, in Vermont. Wickman,⁵ of Swe-

¹ *The Life of Sir Walter Scott*, J. G. Lockhart, p. 13.

² *Beobachtungen über Lähmungszustände der unteren Extremitäten und deren Behandlung*. Stuttgart, 1840, F. H. Kohler.

³ *Verhandl. d. x. internat. med. Cong.*, 1890, Berlin, 1891, II, 6, Abth., 37-47.

⁴ *J. Am. M. Ass.*, 1896, 36: 1.

⁵ *Beiträge zur Kenntniss der Heine-Medinschen Krankheit*, Berlin, 1907.

den, in 1905-06, defined mild, non-paralytic types not before recognized, made the first systematic study of the disease from an epidemiological point of view, and found evidence that it was contagious, though usually slightly so. He directed especial attention to several factors in its spread, viz.: routes of travel, public gatherings of children, abortive or ambulant cases, and healthy intermediate carriers. In the spring of 1909 Landsteiner and Popper⁶ succeeded in transmitting the disease to two monkeys by inoculating them with the spinal cord of a child who had died of infantile paralysis. Later in the year Flexner and Lewis⁷ obtained the same results, and further transmitted the infection from monkey to monkey through an indefinite number of passages. To Harbitz and Scheel of Norway we are indebted for formulating the pathologic anatomy of the affection.

The Disease.—The first stage comes on abruptly and is characterized by fever, often with nausea and vomiting, irritability and stiffness of the neck; and sometimes pain and tenderness in the extremities. This stage lasts a few days—usually three or four—when the paralysis appears. Not infrequently the paralysis is the first symptom noticed. Non-paralytic attacks are called “abortive” and are usually recognized only when occurring in an epidemic or in association with frank clinical cases. The spinal fluid during the acute stage shows an increase in the number of cells—about two hundred per cubic millimeter—which distinguishes infantile paralysis from cerebrospinal fever and other acute forms of meningitis. Sensory disturbances are rare. The paralysis usually remains stationary for a few weeks and then shows a tendency to marked improvement or even recovery during the next few months. As long as there is pain or tenderness, active motion, massage and electricity are contra-indicated. The affected parts should be kept at rest during this phase and careful attention should be given to position so as to prevent contractures. Some residual paralyses respond to physiotherapy, but others are apt to be permanent. The crippled children left each year in the wake of infantile paralysis present an important problem which is well met by special clinics patterned after the Harvard Infantile Paralysis Commission in Massachusetts and similar efforts in Vermont and New York.

Incubation.—The incubation is assumed to be short. It is variously stated at from one to three days, up to fourteen days; commonly seven days. My colleagues Aycock and Eaton⁸ estimate that the incubation period is from ten to eighteen days, average fourteen days. This conclusion is based upon a study of the interval between multiple cases in families. The period of incubation of the experimental disease in monkeys is three to twelve days. It is sometimes over fourteen days, and occasionally prolonged; if the animal sickens in less than four days, the diagnosis must be regarded with caution. It is unlikely that the period of incubation of the natural disease in man is shorter than that of the experimental disease in monkeys. I therefore think that epi-

⁶ *Ztschr. f. Immunitätsforsch.*, Orig., 1909, 2: 377.

⁷ *J. Am. M. Ass.*, 1909, 43: 639.

⁸ *Am. J. Hyg.*, 1925, 5: 724.

demologic studies should consider happenings about two weeks prior to onset.

Epidemics and Prevalence.—Epidemics have been more severe and the case rates have been higher in small towns and rural districts than in the more densely populated cities. Even in the cities the disease does not especially strike the crowded districts. Cold countries having marked seasonal variations in temperature have been most affected. The present pandemic of infantile paralysis apparently started in Sweden (1881) and extended over northern Europe and to the northeastern portion of the United States, which areas have been most heavily affected. The disease gradually spread until it now exists in all quarters of the world. The distribution, however, is quite unequal and irregular.

Following the first record of the disease in an epidemic form in Sweden in 1881, some groups of cases were reported in 1882 in Italy, and in 1886 in Norway, Germany and France. These were small outbreaks. In 1887, Medin described an epidemic in Stockholm of forty-four cases, and this is the first important work on the subject. In 1894, an epidemic of 132 cases occurred in Rutland, Vermont, which was recorded by Caverly. Small outbreaks are recorded in the "nineties" in Italy, France, Australia, England, and America, and a larger one occurred in Vienna (42 cases), in 1898, and in Norway and Sweden in 1899. The last was described by Wickman. In the middle of the next decade, 1900 to 1910, the outbreaks increased in size and number; in fact, during the years 1903-1907 it may be said that the disease was pandemic in Norway and Sweden. During the years 1907-1910, large epidemics occurred in the states of New York, Massachusetts, Iowa and Minnesota. At the same time epidemics of the disease were recorded in Australia (Stephens), in Vienna (Zappert), Westphalia (Krause and Reckzek), in Paris, (Netter), in Austria (Furnatt, Potpeschnigg, Lindner and Mallu), Switzerland (Hagenback), and in Russia (Jogichess).

The progress of the disease is shown in the following table:

Years	Cases	Outbreaks	Average Number of Cases per Outbreak
1880-1884	23	2	11.5
1885-1889	93	7	13.
1890-1894	151	4	38.
1895-1899	345	23	15.
1900-1904	349	9	39.
1905-1909	8,054	25	322.

The United States has been sorely afflicted. Of the 8,054 cases reported in five years (1905-1909), we contributed 5,514 cases, or about five-sevenths of the total number. Since 1910, the disease has shown a still further rapid increase. From 1910 to 1914 (inclusive), 18,800 cases were reported in the United States; and 31,500 cases in the next two years, 1915 and 1916. The disease was progressive, epidemics grew in extent and intensity, until it

became pandemic, and in 1916 swept the United States, involving numbers far in excess of anything hitherto recorded. There were 29,000 cases and 6,000 deaths; Massachusetts reported 1,926 cases, and New York City alone over 8,928 cases and 2,407 deaths.⁹

The epidemic of 1916 was the worst known in the history of the disease. Since that time, the story of infantile paralysis shows that the virus is sown broadcast, that no region is spared, but that the outbreaks are smaller and sporadic cases crop out capriciously. The following table shows the number of cases reported in the registration area of the United States since 1912:

Year	Cases	Year	Cases
1912	2,598	1920	2,291
1913	1,953	1921	6,229
1914	1,258	1922	2,221
1915	1,749	1923	3,245
1916	27,363	1924	5,199
1917	4,167	1925	5,615
1918	2,514	1926	1,875 (estimated)
1919	1,895		

Geographic and Seasonal Distribution.—One of the many peculiarities about infantile paralysis is that while it is a warm weather disease, it spares warm countries; a comparatively small number of cases have been reported from the tropics. The disease has a world-wide distribution; every country is seeded with the virus, some more heavily than others. Northern Europe and the northern part of the United States have suffered most. In the summer of 1924 there was an unusually severe outbreak in Iceland, while in December 1924, and January, 1925, an epidemic occurred in New Zealand.

Infantile paralysis is strikingly a warm weather disease. During the winter cases occur sporadically; the number increases as summer advances. The curve rises in July and reaches its peak in August or early September, and declines rapidly with the advent of cold weather. This seasonal periodicity repeats itself with marked regularity in all endemic regions of the temperate zones. The recurrence is as regular as the crops, but the amount of the harvest cannot be foretold. A community having an epidemic one year is spared the next. Aycock and Eaton¹⁰ noted a minor rise in the spring (usually in March), making the curve bimodal.

While the disease is most prevalent in the warm, dry months—from May to November in the northern hemisphere and November to May in the southern hemisphere—cold weather outbreaks occasionally occur. In the United States, late fall and winter foci usually represent the tailings of a previous

⁹ See the splendid epidemiological study of the epidemic of 1916, *United States Public Health Bulletin, No. 91*, July, 1918, by Lavinder, Freeman and Frost. Also the "Epidemic of Poliomyelitis in New York City in 1916," published by the Department of Health, New York City.

¹⁰ *Am. J. Hyg.*, 1924, 4: 356.

summer epidemic. A severe outbreak (3,840 cases and 380 deaths) occurred in Sweden in 1911 in the late fall, October to December. Sporadic cases occur in the cold winter weather.

The attack rate is inversely proportional to the concentration of population. That is, infantile paralysis is predominantly a disease of smaller communities and rural sections, the notable exception being the great epidemic of 1916 in the United States, which struck New York City with unusual force. In general, the incidence rates are lowest in metropolitan cities, higher in small towns and highest in villages and in the country.

The disease is not influenced by social or economic conditions. It prevails in good and bad sanitary situations. It spares neither rich nor poor, clean nor dirty, wise nor foolish, strong nor weak.

Sex and Age.—About 56 per cent of all cases reported are males and about 44 per cent females. This ratio is quite constant at different ages, in different sections of the country, in urban and rural outbreaks, in moderate and severe epidemics and in different races.

Infantile paralysis is a disease of childhood, probably because the young are more susceptible to the infection. In this regard, it seems to be like diphtheria and scarlet fever. Sixty-five per cent of all cases of infantile paralysis occur under five years of age and 95 per cent under ten years; only 5 per cent occur over ten, and these mostly between ten and twenty years. Any age may be affected, however; infantile paralysis sometimes attacks grandfathers. The disease occurs at a somewhat earlier age in the city than in the country, the attack rate being relatively higher in city children under six years of age.

Case Fatality.—Infantile paralysis is a malignant disease both as regards its lethality and its sequelæ. Of thirty-eight epidemics occurring in various parts of the world from 1894 to 1921, comprising 20,568 cases, the case fatality rate ranged from 30.7 per cent to 5 per cent, the average of all being 20.8 per cent. According to morbidity and mortality records, the disease appears to be more virulent in winter and spring than in summer. Thus, the case fatality rate in the United States was 86 per cent in March and 20 per cent in August, 1921.¹¹ This difference may be more apparent than real, and due to lack of recognition and incomplete reporting of mild cases during the off season. It is of interest to note that during the great New York epidemic of 1916, the case fatality rate was about the same (27.39 per cent) during the rise, peak and fall of the curve.

The case fatality rate is lowest from one to five, the age of greatest incidence of the disease. It is high under one year, and becomes proportionately higher over five as age advances. This may be an expression of susceptibility, or more likely due to failure to recognize and report mild cases in infants and adults.

¹¹ *Am. J. Hyg.*, 1924, 4: 681.

CASE FATALITY OF INFANTILE PARALYSIS IN A SERIES OF WELL-REPORTED EPIDEMICS

Year	Place of Epidemic	Reported by	Number of Cases	Number of Deaths	Case Fatality Rate
1894	Vermont	Caverly	132	18	13.
1905	Sweden	Wickman	1,031	145	14.1
1905	Norway	Wickman	719	111	15.4
1906	Norway	Wickman	467	124	26.57
1907	Norway	Wickman	204	31	15.2
1908	Norway	Wickman	59	10	16.9
1908	Wisconsin	Frauenthal and Manning	408	62	15.3
1908	Austria	Locker	68	8	11.7
1908	Minnesota	Hamilton	150	13	8.6
1908	Vienna and Lower Austria	Zappert, Leiner and Weisner	555	61	10.99
1909	Bristol, England	Parker	37	2	5.
1909	Norway	Wickman	51	8	15.68
1909	Cuba	Lebrede and Ricio	140	11	7.89
1909	Massachusetts	Lovett	628	51	8.
1909	Paris	Netter	100	5	5.
1909	Arnsberg, Germany	Krause	436	66	15.1
1909	Germany	Mueller	130	20	16.
1910	Nauru, Melanesia	Muller	700	38	5.4
1910	Massachusetts	Lovett and Sheppard	601	54	8.98
1910	Mason City, Iowa	Frost	57	7	12.28
1910	Norway	Wickman	57	13	22.81
1911	Cincinnati and environs	Frost	150	46	30.7
1911	Norway	Wickman	1,158	216	18.7
1911	Vermont	Caverly	27	8	29.6
1912	Norway	Wickman	183	34	18.58
1912	Bavaria	Uffenheimer	197	20	10.2
1913	Norway	Leegaard	221	30	13.58
1914	Massachusetts	Massachusetts State Board of Health	151	45	29.8
1914	Vermont	Caverly	304	53	17.3
1915	Vermont	Caverly	44	11	25.
1915	Massachusetts	Massachusetts State Board of Health	135	32	23.7
1916	Vermont	Caverly	64	12	18.7
1916	New York City	Emerson	8,881	2,434	27.39
1916	Massachusetts	Massachusetts State Board of Health	1,927	452	23.4
1917	Vermont	Caverly	171	18	10.5
1919	Vermont	Dalton and Aycock	15	0	0
1921	Vermont	Aycock	63	9	14.28
TOTAL			20,568	4,304	20.8

Spread in Families, Multiple Cases.—Infantile paralysis, as a rule, does not spread in families. Only 4.3 per cent of 8,634 families attacked in New York in 1916 had more than one case. Most students of the disease have noticed that when multiple cases do occur in a family, they usually come down together or within a short time of each other. This signifies simultaneous infection from a common source. Aycock and Eaton¹² studied 576 cases in 253 families, and found that 80 per cent occur within seven or eight days of each other, indicating common-source infection; the remaining 20 per cent occur after an interval of ten to eighteen days from the primary

¹² *Am. J. Hyg.*, 1925, 5: 724.

case, suggesting infection from the primary case. Only exceptionally does infantile paralysis tend to spread within the family. The situation with measles is just the opposite, for in that disease the infection is usually brought into the family by one child, from which there is a heavy secondary incidence, indicative of its contagious nature. Furthermore, infantile paralysis usually attacks the younger children in the family, whereas measles is as a rule brought home by the six-year-old child, and then spreads to the younger brothers and sisters.

The Virus.—The nature of the virus is unknown; we therefore lack an exact criterion by which to recognize the disease with certainty. The virus is filtrable through the pores of the finest Chamberland candles. Flexner and Noguchi¹³ described minute globoid bodies arranged in pairs, chains and masses in artificial cultures.

The virus is present in greatest virulence or concentration in the spinal cord and also the brain of infected persons and animals. It has also been demonstrated in other organs and tissues, as, for instance, the mucous membrane of the nose and pharynx, the mesenteric glands, the axillary and inguinal lymph-nodes, also in the blood, and in the cerebrospinal fluid; in fact, the virus may be widespread and has even been found in the intestinal secretions. The suspicion that the alvine discharges may, therefore, be virulent is sufficient indication that they should be disinfected in all cases until further knowledge of the subject is at hand.

Resistance of the Virus.—It is killed by a temperature of 45° to 50° C. in half an hour; also by comparatively weak disinfectants, such as a 1:500 solution of permanganate of potash; 1 per cent menthol in oil; a powder containing menthol, 0.5 per cent salol; 5 per cent boric acid; 20 per cent (Landsteiner and Levaditi); and a dilution of 1 per cent of peroxid of hydrogen. The virus is not destroyed by very low temperatures nor by drying over caustic potash, or *in vacuo* for a considerable period. A virulent cord has been kept for three to six years in 50 per cent glycerin without losing its activity, resembling in this respect rabies, vaccinia, and other filtrable viruses, and differing for the most part from non-spore-bearing pathogenic bacteria which are usually killed by glycerin in a short while. The virus remains virulent in ordinary water for thirty-one days,¹⁴ and the same length of time in sterile milk.

The Experimental Disease.—Infantile paralysis is not known to occur in nature in any other animal than man, although an animal reservoir is suspected. Of all animals tested under laboratory conditions, only certain monkeys are susceptible. The old world monkeys, especially *Rhesus*, are most suitable for experimental purposes; the new world species are quite resistant. The disease in the monkey is a faithful reproduction of infantile paralysis as seen in children.

The experimental disease in monkeys may be produced by injecting the

¹³ *J. Am. M. Ass.*, 1913, 60: 362.

¹⁴ Levaditi and Pasti, *Ann. de l'Inst. Pasteur*, 1911, 25: 805.

virus directly into the central nervous system, preferably the brain. Monkeys may also be infected by introducing the virus subcutaneously or into the peritoneal cavity, and even by intravenous inoculation. They have been infected by placing virulent material upon the healthy mucous membrane of the nose, by injection into the anterior chamber of the eye, and also by inhalation of the infectious material forced into the trachea, and finally by introducing the virus into the stomach, along with an opiate, to restrain peristalsis. Leiner and Weisner have infected monkeys through the uninjured nasal mucous membrane. I have obtained similar results. Monkeys have so far never been known to contract the disease "spontaneously," even though kept in small cages in closest contact with infected monkeys. There are many distempers in the lower animals, some of them with paralytic symptoms, but so far as known none are associated with the virus of infantile paralysis.

Immunity.—One attack of infantile paralysis confers a high degree of immunity. Second attacks are unknown. Monkeys which have recovered from the infection show a high degree of immunity, and their blood-serum contains antibodies capable of rendering the virus harmless. If the blood-serum of an immune monkey is mixed with an emulsion of virulent spinal cord and the mixture allowed to stand for several hours, the virus is no longer capable of producing the disease in susceptible animals. This property has been used by Anderson and Frost to corroborate the clinical diagnosis in abortive cases. Monkeys, however, often fail to respond to injection with fresh virus. Negative results must therefore be studied critically.

No racial immunity to the infection is known, although infantile paralysis has been for the most part confined to the white race. The unequal racial distribution may be due to the fact that the temperate zones and cooler regions are mostly inhabited by whites. However, in Cuba in 1907-08, fewer cases were reported among the blacks than the whites; thus, of seventy-two cases, sixty were whites, four of mixed races and eight Negroes.

Modes of Transmission.—*Contact Theory* (based upon the assumption that the virus is discharged from the mouth and nose and enters through the same channel).—There is evidence to support the theory that the disease is directly transmissible from person to person and there is a suspicion that healthy carriers play an important rôle in spreading the infection. This view was enunciated by Wickman and received support through the experiments of Kling, Pettersson and Wernstedt, and also Flexner. It is known that the mucous membrane of the nose and throat contains the virus, and in one case the salivary glands were shown to be infective. Osgood and Lucas demonstrated that the nasal mucous membrane of two monkeys experimentally inoculated with poliomyelitis remained infective for six weeks in one case and five and one-half months in another. This observation strengthens the suspicion of the existence of chronic human carriers. If healthy carriers continue to spread the infection months after the attack, it increases the difficulty of suppressing the disease, and further renders doubtful the efficiency of strict isolation and prophylactic measures directed only to persons in the

acute stage of the disease. The fact that the mucous membrane contains the virus is not, however, proof that it is discharged in sufficient amount in the secretions from the mouth and nose to be a menace. In a series of eighteen cases Rosenau, Sheppard and Amoss¹⁵ were unable to demonstrate the virus in the nasal and buccal secretions obtained from persons in various stages of convalescence. Strauss¹⁶ had similar negative results in a series of ten cases. Amoss and Taylor¹⁷ later showed that the normal nasopharyngeal secretions are able to neutralize the virus of infantile paralysis, and this may account for the negative results. However, Flexner and Amoss¹⁸ find that the protective power of the nasal mucosa is not in itself adequate to prevent infection with the virus placed on it, since slight injury to such independent structures as the meningeal-choroid plexus favors the passage of the virus to the central nervous system.

Flexner and Amoss¹⁹ consider that the virus is regularly present in the nasopharynx in cases of infantile paralysis in the first days of illness and especially in fatal cases; that it diminishes relatively quickly as the disease progresses, except in rare instances; and that it is *unusual* for the carrier state to develop. Hence, the period of greatest infectivity would be early in the disease.

There are epidemiological features of infantile paralysis that cannot be explained on the theory of contact infection, such as the seasonal prevalence, rural incidence, the lack of tendency to spread in families and the disinclination to attack congested centers or to spread in hospitals, schools, institutions and other crowded places.

The Insect-borne Theory.—Infantile paralysis has a seasonal prevalence corresponding to that of the insect-borne diseases and just opposite to the seasonal prevalence of diseases spread by contact through secretions of the mouth and nose. Many epidemiological features suggest the possibility of an insect vector. Studies²⁰ gradually focused attention upon the stable fly (*Stomoxys calcitrans*). Rosenau and Brues²¹ demonstrated that the virus may be transmitted from monkey to monkey through the bite of the stable fly. These results were soon confirmed by Anderson and Frost.²² Subsequent work, however, has given negative results. Flexner and Clark found the virus to survive forty-eight hours in house flies. Even though infantile paralysis may occasionally be transmitted experimentally by insects biting monkeys in a cage, the question remains whether this is the way the infection is spread from man to man.

Milk-borne Infection.—During the great epidemic of 1916, Dingman²³ reported a group of eight cases which were the only ones to develop at the

¹⁵ *Boston M. & S. J.*, 1911, 164: 743.

¹⁶ *J. Am. M. Ass.*, 1911, 56: 1192.

¹⁷ *J. Exper. M.*, 1917, 25: 507.

¹⁸ *J. Exper. M.*, 1920, 31: 123.

¹⁹ *J. Exper. M.*, 1919, 29: 379.

²⁰ *Month. Bull. Bd. Health Mass.*, 1912, 7: 308.

²¹ *Month. Bull. Bd. Health Mass.*, 1912, 7: 314, also Brues and Sheppard, *J. Econom. Entomol.*, 1912, 5: 305.

²² *U. S. Pub. Health Rep.*, 1912, 28: 1733.

²³ *N. York State J. M.*, 1916, 16: 589.

time in and about Spring Valley, New York, all of which had their onset between July 20 and 22. All the cases used raw milk from the same two-cow dairy. Three families who used this milk but boiled it escaped. There was a case, a four-year-old child, at the dairy who became ill with the disease on July 4.

Another outbreak attributable to milk occurred at Cortland, New York (population 15,000), December 14 to 25, 1925.²⁴ There was a paralytic case of the disease on the dairy farm, and this individual milked the cows and handled the milk for a period of four days (December 7 to 11) while in the acute stage of the disease. This milk constituted only 4 per cent of the total supply of Cortland, yet all the cases (8 in number) occurred among users of it. They came down between December 14 and December 25, and all of them had typical clinical pictures with paralysis. The milk was not pasteurized and no case occurred among those who first boiled it.

It is now clear that the virus of infantile paralysis may be taken in food or drink. It is also plain that while milk may be a vehicle of transmission, it is not the usual way in which the disease is spread.

Other Theories.—It has been suggested that the virus may be air-borne in the sense that it is carried in the dust. Neustaedter and Thro²⁵ claim to have infected monkeys from dust collected from sick rooms.

It is suspected, but not proven, that some healthy animal serves as a reservoir for the virus. Richardson believes the infection comes from rats. Brues finds a similarity between the epidemiology of infantile paralysis and plague.

Transmission through wounds and other means have not been ruled out of consideration.

The favorite theory is to regard infantile paralysis as very communicable, like measles, and much more widespread than indicated by the paralytic cases. According to this theory, most cases are mild, escape notice, but leave protection. Furthermore, it is assumed that only the occasional severe cases with paralysis come to clinical diagnosis, the disease being spread mainly by missed cases and carriers. We know that in practically all epidemic diseases the infection is much more widespread than indicated by the clinical cases, and it is assumed that in infantile paralysis the virus is very much more widely broadcast; in other words, it is a very common infection which is always present in endemic regions, but which in recent years has gained world-wide distribution and an increased virulence.

Multiple Modes of Spread.—From our meager knowledge of the disease the conclusion is forced upon us that infantile paralysis is one of the diseases like typhoid fever that is spread in more than one way. This conclusion is emphasized by the epidemiologic vagaries of the disease and the evidence of animal experimentation.

²⁴ *J. Am. M. Ass.*, 1926, 87: 635.

²⁵ *N. York M. J.*, 1911, 94: 13.

Prevention.—No definite or effective system of prevention can be formulated until we are sure of the mode of transmission. Meanwhile health authorities are entirely justified in requiring cases to be reported, isolated, and analogous lines of preventive measures applied, such as disinfection, screening, and guarding against insects, allaying unnecessary dust, the pasteurization of milk, etc. Until the modes of transmission of the disease are established, however, we can have no confidence in our prophylactic measures, which most resemble the old "shotgun" prescription.

The following measures have been suggested: The patient should be isolated as completely as possible in a clean, bare room, well screened to keep out insects. This is a good practice despite the fact that the disease shows no tendency to spread in children's asylums, hospitals, and other institutions. The same statement, however, was made of typhoid fever not many years ago. Visiting should be interdicted and only the necessary attendant should be allowed to come in contact with the patient. All discharges, including sputum, nasal secretions, urine, and feces, should be thoroughly disinfected, and special care should be taken that cups, spoons, remnants of food, etc., which may have become contaminated by the patient, are burned, scalded, or otherwise purified.

Towels, bed linen, and other fabrics should be boiled or dipped into a germicidal solution strong enough to destroy the typhoid bacillus. The nurse and physician should observe the same precautions regarding their hands and clothing as are recommended in attending a case of scarlet fever.

The period during which the isolation should be maintained cannot even be guessed at. Children are usually not permitted to return to school for at least three weeks, but, if chronic carriers play the important rôle suspected, this time would be far too short in many instances.

Since the virus can be killed experimentally by a 1 per cent solution of peroxid of hydrogen, antiseptic gargles, sprays, and nose washes of this solution have been advocated for the patients, the nurse, and physician, and other members of the family. Germicidal chemicals, however, applied to the mucous membranes on which the virus has been deposited have no prophylactic action and are of doubtful value; in fact, such substances may affect unfavorably the protective properties of the mucosa. They may even be objectionable by injuring the delicate mucous membrane and thus favoring infection; in fact, nose washes or sprays, unless judiciously used, may force pathogenic bacteria up the eustachian tube or into the sinuses.

Normal mucous membranes are protective against this as against other infections of the upper respiratory tract. According to Flexner and Amoss, the normal nasal mucosa is an invaluable defense against infection with the virus of poliomyelitis, and the number of healthy and chronic carriers of the virus is probably determined and kept down through the protective activities of this membrane.

In the presence of an epidemic, street and house dust should be kept down by sprinkling, oiling, and the other means employed for this purpose. Dust

should be allayed whether there is an epidemic of infantile paralysis or not. During epidemics children should be kept away from public gatherings, prohibited from using public drinking cups, and special attention given to the diet to prevent gastro-intestinal disorders, for many a case of infantile paralysis starts with a digestive upset.

The possibility of milk-borne infection emphasizes the importance of the prophylactic value of pasteurized milk.

The summer prevalence and the rural distribution of the disease are disturbing factors with regard to summer vacations. The only safe rule is to avoid places when localized outbreaks are in progress. The fact, however, that infantile paralysis does not usually strike the same place in epidemic form in two succeeding seasons makes such places reasonably safe the year following an epidemic.

The prevention of deformities in paralyzed children is important. Massage, exercise and all active measures are contra-indicated during the acute stage and as long as there is tenderness. Special attention must be given to maintain a normal position of paralyzed limbs so as to prevent deformities which develop quickly. As soon as acute symptoms have subsided, muscle training should be instituted.

EPIDEMIC ENCEPHALITIS

Epidemic encephalitis, also called encephalitis lethargica, infectious ophthalmoplegia, acute encephalitis, and nona, is an acute infection due to a specific virus which is assumed to enter the system through the nasopharynx, and like infantile paralysis has a special affinity for the central nervous system, although for different areas and elements. The disease is characterized by progressive lethargy or stupor, which gives to it the popular term "sleeping-sickness." The lesions are in or about the nuclei of the third pair of cranial nerves; ophthalmoplegia occurs in 75 per cent of the cases; there is usually irregular fever.

Epidemic encephalitis has the characteristics of a communicable infection. Its mortality is high; the incidence is low, but growing. The disease shows a predilection for the later winter months. It is rare in young children, most common in adults, and affects both sexes. The incubation period is uncertain; the onset may be sudden or gradual. The symptoms vary greatly in severity from mild and abortive forms to rapidly malignant types. Almost no other infection is so irregular and has so many different forms. This is due to the fact that various parts of the brain, cord and meninges are involved.

The only epidemic appearance of any similar disease in the past has been in connection with epidemics of influenza. Following the pandemic of 1889-90, the mysterious nona appeared in northern Italy, and then in Hungary, and spread to Germany, France and Italy. Camerarius described a grip epidemic in Tübingen in 1718 and mentioned a sleeping-sickness (*Schlafkrankheit*) in connection with it. In 1768, Lepecq de la Cloture described a "coma somno-

lentum" after the grip, and Ozanann mentioned epidemics of "catarrhal fever" and "soporosite" as having occurred in Germany in 1745, in Lyons in 1800, and in Milan in 1802. The disease was not especially reported following the influenza epidemic which swept this country in 1889-90, but was observed after the 1918-19 outbreak. Von Economo described cases occurring in Vienna during the winter of 1916-17, and shortly afterward cases occurred in England, the United States, and France. It reached the United States in 1918 and has spread to the four quarters of the globe. It is becoming a problem of serious magnitude.

Flexner ²⁶ points out that lethargic encephalitis is a communicable disease, imperfectly understood. Amoss ²⁷ states that lethargic encephalitis is an epidemic disease, the main manifestations of which relate to injury inflicted upon the central nervous system, in particular the basal ganglia of the brain. It is distinguished from infantile paralysis, which affects in particular the gray matter of the spinal cord and medulla oblongata. Furthermore, infantile paralysis prevails especially in warm weather, whereas lethargic encephalitis is reported mainly in the winter season, although recently cases have arisen in the midsummer months. Infantile paralysis is readily transmitted to monkeys, whereas this is doubtful with lethargic encephalitis. There are also difference in age incidence and other factors, which make it plain that we are dealing with two separate infections.

The cause of the disease is not known. Loewe and Strauss claim to have reproduced it in rabbits and monkeys by the inoculation of brain material and nasopharyngeal washings; also by means of cultures.²⁸ This work was extended in part by Levaditi and Havier, but these results lack confirmation. Rosenow ²⁹ has isolated a peculiar streptococcus constantly from infected tonsils, teeth or nasopharynx during life, and from the brain after death.

Increase of Infections of the Central Nervous System.—It is disconcerting to note the growing prevalence of various kinds of infections which expend their energy on the central nervous system. As the common communicable diseases are being controlled, infections of the brain, spinal cord and nerves are growing. This increase is probably real, for it is not likely that these diseases are simply being uncovered. Certainly there has been a great increase in the amount of infantile paralysis; disseminated sclerosis is prevalent. The spread in the amount of encephalitis is alarming. Meningitis of varying forms rose during the World War and as a result of the influenza epidemic.

There are evidently several forms of encephalitis in addition to the epidemic form described above, about which little is known. We recently had an epidemic of the Australian X disease, and large outbreaks in Japan of a peculiar disease with cerebral and spinal symptoms. There is a suspicion that cowpox sometimes attacks the brain and meninges. This may be pro-

²⁶ *J. Am. M. Ass.*, 1920, 74: 865; also L. Van Boeckel, A. Bessemans and C. Nelis, *L'Encephalite*, Nossent and Co., Brussels, 1923.

²⁷ *J. Exper. M.*, 1921, 33: 187.

²⁸ *J. Infect. Dis.*, 1920, 32.

²⁹ *J. Am. M. Ass.*, 1924, 82: 1648.

duced experimentally in rabbits by placing some of the vaccine on the dura mata or by vaccinating rabbits on the cornea. The relationship of this disease to herpes and even to chickenpox is being studied. This is a fertile field for investigation and a subject concerning which little is known.

CHICKENPOX

(*Varicella*)

Chickenpox (*varicella*) is usually regarded as one of the minor communicable diseases in that the mortality is low, complications and sequelæ not frequent. Chickenpox is not always a harmless disease; when it runs through an institution with many small children it occasionally develops malignancy. It may leave disfiguring scars; sepsis or erysipelas sometimes originate in the pustules; complications, such as pneumonia, nephritis, and gangrene of the skin also occur.

The period of incubation is probably from fourteen to sixteen days; the maximum for public health purposes is twenty-one days. One attack produces a definite immunity. No age is exempt. The maximum incidence occurs at five to six years. By the time adult age is reached, about 52 per cent have had the disease. Chickenpox is more prevalent among girls than boys; among native than foreign-born children; and among white than colored.

The disease is peculiar to man; animal inoculations have so far proved negative, although the virus is now being studied in rabbits.³⁰ Rivers³¹ has demonstrated nuclear inclusions in varicella lesions by injecting human material in the testicles of monkeys (vervets) which resemble those deemed characteristic of a certain well-known group of filtrable viruses. There is a curious and apparently intimate relationship between chickenpox and herpes.

Chickenpox is very readily communicable and spreads through families or institutions, and occurs in more or less widespread epidemics. The cause of the disease is not known and its modes of transmission not certain. The virus is contained in the content of the vesicle. The vesicles appear early on the mucous membranes and rupture at once, rendering the disease communicable early, even before the exanthem is in evidence. The mode of transmission is probably directly from person to person; indirectly through articles freshly soiled by discharges from an infected person.

Differential Diagnosis from Smallpox.—Health officers should require cases of chickenpox to be reported, if for no other reason than that it is often mistaken for smallpox. The differential diagnosis between chickenpox and smallpox is often an important and difficult public health matter. In smallpox the vesicle is multilocular, in chickenpox unilocular. The smallpox eruption comes out as one crop, the chickenpox eruption in several crops, and therefore the several stages appear on the skin at the same time. In smallpox the extremities are usually involved, but are spared in chickenpox.

³⁰ T. M. Rivers, *J. Exper. M.*, 1923, 38: 673.

³¹ *J. Exper. M.*, 1926, 43: 275.

There are other clinical differences, but the diseases are not always true to textbook type.

The differential diagnosis may be made in doubtful cases by a histological examination of the pock, or by inoculating the contents of the vesicle upon the cornea of rabbits. Vaccine bodies are found in sections of the skin lesion in smallpox, not in chickenpox; the vesicle of the former is multilocular, the latter unilocular. The vesicle upon the cornea of rabbits produced by smallpox is distinct and contains the vaccine bodies; the lesion resulting from chickenpox is trifling and does not contain the vaccine bodies. See the Paul reaction, page 30.

Force and Beckwith³² state that rabbits previously vaccinated with vaccine virus will give a marked intradermal reaction with smallpox vesicle contents in from twenty-four to forty-eight hours, but will not give such a reaction with varicella vesicle contents. I have not been able to obtain clear-cut results with this test. A similar test has been proposed by introducing some of the contents into the skin of a well-vaccinated person. If smallpox, an "early" reaction results; if chickenpox, no reaction. This test also is uncertain on account of the associated microorganisms which confuse the skin reaction. Monkeys are not susceptible to chickenpox, but may be given smallpox.

Prevention.—The prevention of chickenpox depends upon isolation and disinfection at the bedside. Children with chickenpox should not be permitted to go to school.

Kling³³ favors vaccination with chickenpox virus, in the face of an epidemic in an institution. The response to such vaccination is slight and local. The virus is taken from a fresh, clear vesicle and introduced into the skin. Eight days later red papules appear at the site of "vaccination," which next day develop into typical vesicles of chickenpox, with a slight reddened areola. There are no general symptoms. Previous vaccination with smallpox does not prevent a positive reaction to vaccination with chickenpox, thus emphasizing the essential difference between the two diseases. An epidemic of chickenpox at the Stockholm Children's Hospital in August, 1914, was cut short by vaccinating all the well children with the virus from a chicken pock. Steinert³⁴ also obtained successful results. Michael's³⁵ results are less convincing.

Hess and Unger³⁶ obtained an immunity to varicella by means of an intravenous injection of the contents of the vesicles. In thirty-eight instances it failed to protect in only one case.

GLANDERS

Glanders or farcy is a widespread communicable disease of horses, mules, asses, and other animals, and is readily communicated to man. Cats may become infected by eating the flesh of glandered horses. Goats also have the

³² *J. Am. M. Ass.*, 1915, 65: 588.

³⁴ *Ztschr. f. Kinderh.*, July 28, 1920, 94.

³⁶ *Am. J. Dis. Child.*, 1918, 16: 34.

³³ *Berl. klin. Wchnschr.*, 1913, 50: 2083.

³⁵ *Arch. Pediat.*, 34: 702.

disease. Cattle are immune. Guinea-pigs and field mice are very susceptible by experimental methods; white mice have a natural immunity. In both man and horses it is remarkable for its fatality. The disease is characterized by the formation of inflammatory nodules either in the mucous membrane of the nose (glanders) or in the skin (farcy). The nodules break down, leaving crater-like ulcers. On the skin the farcy buttons break down and discharge an oily material. The mortality is about 50 per cent. Glanders occurs both as an acute and chronic disease.

The infection may be introduced into the system either through the skin or mucous membrane, and is usually communicated directly from the horse to man by contact with the infected discharges. The disease is sometimes communicated from man to man. Washerwomen have become infected from the clothes of a patient.

The bacillus of glanders, *B. mallei*, does not have a spore. It is comparatively frail and readily destroyed by the usual physical and chemical germicidal agencies used against spore-free bacteria. Its resistance corresponds to that of the tubercle bacillus, which it closely resembles in several particulars. The bacillus, however, is frequently protected by albuminous matter or buried in the dirt of stables, water troughs, harnesses, and other objects. While the naked germs of glanders are readily destroyed, they are frequently hard to get at; penetration and cleanliness are, therefore, imperative.

The prevention of glanders in man depends primarily upon the suppression of the disease in horses. The only difficulty in controlling the disease in horses lies in the early diagnosis and recognition of mild or missed cases, which are very common. Horses affected with occult or latent glanders are important factors in the propagation of the infection, especially in the crowded parts of cities. The clinical diagnosis in the frank cases usually is made without difficulty from the characteristic symptoms and the lesions, but laboratory aid is necessary to discover the mild and atypical cases.

Diagnosis.—The diagnosis of glanders may be made by: (1) the mallein test, both intradermo-palpebral and ophthalmic; (2) the agglutination test; (3) the complement fixation test; (4) the Strauss reaction; (5) the isolation of *B. mallei* in pure culture. Each of these tests serves a definite purpose. The mallein test is the simplest and most used of all the tests. The isolation of the glanders bacillus in pure culture is definite and final, but time consuming; it is least commonly used. Of the two serological tests the agglutination test is more sensitive in picking out beginning infections, whereas the complement fixation test is to be preferred for the detection of chronic glanders whether active or inactive.

The Mallein Test.—Mallein is a product of the glanders bacillus corresponding to tuberculin. The injection of mallein into glanderous animals causes a definite rise in temperature and a local swelling about the site of injection, whereas the injection of mallein into normal animals produces no local reaction and very slight if any rise in temperature. The injection of mallein into the subcutaneous tissues (subcutaneous mallein test) has ceased

to be used as a practical method for the field diagnosis of glanders, having been supplanted by the ophthalmic and intradermo-palpebral tests. With the mallein test latent and occult cases of glanders can be diagnosed, but the test must be made and interpreted by an experienced veterinarian else the results may be unreliable.

The *ophthalmic test* has an advantage over other tests on account of its very simple application. It is only necessary to drop into one of the eyes of the animal three drops of concentrated mallein or to dip a camel's hair brush into mallein and introduce this into the conjunctival sac. The reaction usually commences in five or six hours after the introduction of the mallein and lasts from twenty-four to thirty-six hours. A positive reaction is manifested by swelling of the eyelids and a purulent secretion from the tested eye. Irritation of the conjunctiva due to cold weather, dust or other irritating influences must not be confused with a positive reaction.

The *intradermo-palpebral test* is performed by injecting 0.1 c.c. of 1:4 concentrated mallein (one part concentrated mallein plus three parts 0.5 per cent solution phenol in water) into the dermis of the lower eyelid, about one-third of an inch from the edge and midway between the inner and outer canthi. When the injection is well made it produces a small, distinctly outlined swelling about the size of a lentil. In animals affected with glanders the specific reaction becomes marked after about ten to twelve hours, reaching the maximum in twenty-four to thirty-six hours. In a typical reaction there is an extensive edema, hot and extremely sensitive, involving the upper and lower lids and surrounding tissues so that the eye is almost closed. The conjunctiva is markedly congested and from the inner canthus there flows a mucopurulent secretion which collects as a yellowish deposit on the hair below the eye. Sometimes the lower lid only may be involved, again the swelling may extend to below the zygoma, in which instances the sublingual gland is more or less sensitive.

The Agglutination Test.—The agglutination test is of value in all cases of recent infection, the blood-serum possessing a very high agglutinating power—1:1,000 and higher. In chronic glanders the agglutinating power of the blood may be very low—1:400 or less; in some cases even lower than that of normal blood-serum—which may be 1:800 and even higher. It is, therefore, plain that the agglutination test alone does not constitute an entirely satisfactory diagnostic method for glanders. It may be used as an adjunct to other tests.

The Strauss Reaction.—The Strauss³⁷ reaction for the diagnosis of glanders consists in inoculating material containing virulent *B. mallei* into the peritoneal cavity of male guinea-pigs, which causes an enlargement of the testicles, involving the scrotum; the testes become glued to their sheaths. A positive reaction associated with organisms resembling those of glanders, and typical cultures obtained from the lesions, are unfailing evidence of the pres-

³⁷ *Compt. rend. Acad. d. sc.*, 1889, 108: 530.

ence of the specific virus. Failure to obtain the reaction is not proof that a suspected specimen may not have come from a horse or animal with glanders. Arms³⁸ recommends that it is better to use more than one guinea-pig in testing suspected material, and that, before inoculating, it is well to make a microscopic examination as a guide to the dosage. A culture made from the swab often aids in the early diagnosis. Guinea-pigs should be kept under observation for a month, and if a lesion of any kind is present an autopsy should be made and cultures taken.

The Isolation of Bacillus Mallei in Pure Culture.—The bacillus of glanders may be isolated by introducing some of the suspected material subcutaneously and also intraperitoneally into male guinea-pigs. In this way pure cultures may be obtained from the pus or necrotic foci in the spleen, which follow subcutaneous inoculation; or from the characteristic enlargement of the testicle which is observed in animals inoculated intraperitoneally. The organism isolated must be studied for cultural, morphological, and biological characters. The isolation of the bacillus in pure culture gives positive information of unquestioned character in any critical case. The method is not generally applicable to the diagnosis of glanders because it requires too much time and may occasionally fail to discover the bacillus. One of the chief difficulties is that the material is usually grossly contaminated with other pathogens.

Complement Fixation.—In 1909 Schütz and Schubert³⁹ published the results of their important work on the application of the method of complement fixation for the diagnosis of glanders. The splendid results obtained constitute, without doubt, the most reliable method for the diagnosis of glanders which we have at our command at the present time. The complement fixation test is, in fact, one of the most specific of the biological tests in immunity. It is readily applicable to the case of glanders. In testing the blood of mules slightly different treatment is given the serum, because of the presence of inhibiting properties present in mule serum that are not found in horse serum. Previous to testing, horse serum is inactivated at 58° C. for thirty-five minutes, while mule serum is heated to 62° or 64° C. for a like period. The essential elements used in test are as follows:

The *hemolytic* mixture consists of the washed red blood-corpuscles of a sheep and the blood-serum of a rabbit which has been injected with the washed red blood-corpuscles of a sheep.

Complement.—The complement is contained in the fresh blood-serum of a healthy guinea-pig.

Antigen.—This is an extract obtained by shaking glanders bacilli in distilled water. The bacillus is grown in pure culture on 2 per cent acid, 6 per cent glycerin agar. A luxuriant growth upon the surface of the medium is usually obtained in 48 hours. This is suspended in distilled water to which 0.5 per cent phenol has been added, heated to 60° C. for two hours in order

³⁸ *J. Am. M. Ass.*, 1910, 55: 591.

³⁹ *Archiv für wissenschaftliche und praktische Tierheilkunde*, 1909, Bd. 35, Heft 1 and 2, pp. 44-83.

to kill the bacilli. After heating, the suspension of dead bacilli is shaken in a special apparatus for eight to twelve hours, or better yet allowed to remain in the ice box for ten to fourteen days with occasional agitations by hand. The bacilli are separated in the centrifuge and the clear supernatant liquid which is drawn off constitutes the antigen. The strength and specific quality of each extract must be determined by suitable methods of titration.

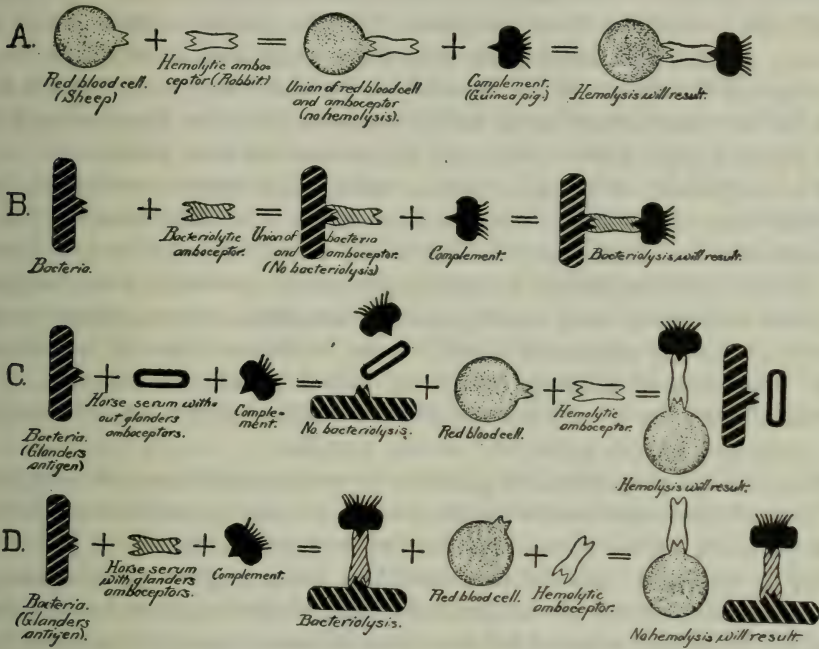


FIG. 44.—DIAGRAMMATIC REPRESENTATION OF COMPLEMENT FIXATION. (Mohler and Eichhorn, U. S. Dept. Agric. Bureau animal indust. Bull. 136.)

A, Hemolytic system; B, Bacteriolytic system; C, Negative reaction with normal horse serum; D, Positive reaction with glandered horse serum.

Technic.—The test is carried out by adding together, in proper proportions, the following: (1) The blood-serum of the horse to be tested; (2) the antigen (extract of glanders bacilli); (3) complement (fresh guinea-pig serum); and (4) the hemolytic system. If the blood-serum of the horse to be tested contains the specific amboceptors, these will unite with the antigen, fix the complement, and thus prevent hemolysis. If the blood-serum of the horse to be tested does not contain these specific amboceptors, this fixation of the complement cannot take place and hemolysis results. Therefore, the absence of hemolysis means the presence of glanders, and vice versa. The tests must always be carried out with controls and carefully conducted as to the amount of each substance used, the temperature and time.⁴⁰ The technic

⁴⁰ A complete description of the diagnosis of glanders by complement fixation, giving in full all the details, will be found in *Bulletin 136*, Bureau of Animal Industry, Apr. 7, 1911, by Mohler and Eichhorn.

and interpretation is precisely that of the Wassermann reaction except that the antigen is an extract of the glanders bacilli.

Prevention.—When glanders is discovered or suspected among horses in a stable, the horses in the infected stable should be tested in the manner above described. All animals with glanders should be destroyed without further consideration. After these animals have been killed and properly disposed of, the stable should be thoroughly cleansed and disinfected. All other horses which have in any way been associated with the infected animals should be carefully watched and tested again after three weeks, and, should there be no indication of the disease in the second test, the stable may be considered free from the infection; otherwise the infected animals should be destroyed and the tests repeated every three weeks until the disease has been eliminated.

The eradication of glanders from a stable often means considerable loss and sometimes a sacrifice of valuable animals, but it is only through vigorous measures that the disease may be controlled. In the disinfection and cleansing, special attention should be paid to the stalls, harnesses, water troughs, bits, food containers, curry combs, sponges, and other objects exposed to the infection, which is eliminated mostly in the secretions from the mouth and nose. The common drinking trough for horses spreads the infection. The bacillus of glanders is very susceptible to bleaching powder, and it therefore is a cheap and reliable germicide for this purpose.

The personal prophylaxis of glanders in man depends upon the education and care of those who have to handle horses. In working with horses known to be infected, rubber gloves, disinfection, and other methods of protection should be employed. Special care should be taken to prevent the spread of the disease through the discharges or by infected fomites from human cases. Fatal accidents have occurred in laboratories in research workers handling pure cultures of *B. mallei*.

ANTHRAX

(*Splenic Fever; Charbon*)

Anthrax has figured prominently in the history of bacteriology and immunology. Anthrax was the first pathogenic bacillus to be seen under the microscope, by Pollender in 1849. Anthrax was the first communicable infection to be experimentally transferred, when Davaine and Rayer,⁴¹ in 1850, communicated splenic fever by the direct inoculation of blood containing these "infusoria" to susceptible animals. This established the etiological significance of the organism which they found in the blood of the sheep which they infected. Pasteur studied the disease and observed the multiplication of the bacilli under the microscope. He also grew the organism on coagulated blood derived from infected animals. Anthrax was the first bacterium to be grown in pure culture by Koch, in 1875.⁴² This was not only the first, but a convincing

⁴¹ *Mém. Sec. de biol.*, 1850, 141.

⁴² *Cohn's beit. zur biol. de pflazen*, 1876, 2: 277.

demonstration of the use of solid medium and the plate method for the isolation of pure cultures. Anthrax was the first dramatic demonstration by Pasteur,⁴³ in 1881, of the prophylactic value of an attenuated virus. Pasteur's achievements in protecting sheep against anthrax closely followed similar laboratory experiments upon fowl-cholera. The disease itself has long been known and has been recognized from early times. One of the best descriptions of an early epidemic of anthrax was by Athanasius Kircher, a Jesuit, who, in 1658, described an outbreak among cattle, occurring in 1617, which later spread to man, claiming about 60,000 victims.

Anthrax belongs to that group of diseases which occurs primarily in the lower animals and secondarily in man. The infection is found especially in cattle, but also in horses, sheep, and other cloven-hoofed animals, and may be transmitted experimentally to mice, guinea-pigs, rats, and rabbits. Cold-blooded animals and birds, as well as dogs, are refractory. Zoölogically, anthrax is one of the most widespread of infectious diseases. It is found in most parts of the world and in a large range of climates. In Siberia it has caused fearful destruction, where it is still known as the Siberian "boil plague." Continental Europe has suffered much from its ravages, and it prevails in South America, Asia and Africa. It has been introduced into the United States with hides and hair. With us, it is a rare disease except in certain well-known infected areas.

In man the infection may enter the skin (*malignant pustule*) or the lungs (*woolsorters' disease*), or may infect the digestive tract and produce intestinal lesions. The infection sometimes localizes itself in the brain and meninges. Human anthrax is mostly an industrial disease contracted through the handling of skins, hair or the animals themselves. Those especially exposed to the hazard are laboratory workers, veterinarians, meat inspectors, farmers, cattlemen, butchers, but especially workers with hides, hair, bristles, wool, etc. Of about one thousand men exposed to the danger in Pennsylvania tanneries, 123 in all contracted anthrax in the course of twelve years, or more than 11 per cent of the number of directly exposed tanners. Seventy-three of these cases were due to the handling of cattle hides and fifty to the handling of goat hides. One-fifth of the cases died.⁴⁴

In anthrax of the skin the infection usually enters through slight abrasions, scratches, or small wounds, especially on the forearm, hand, neck, or face. In butchers or persons who handle infected carcasses or hides, it is apt to occur on the neck and shoulders.

Many cases of anthrax in the United States and in England have been traced to shaving brushes made of horsehair. Vincent⁴⁵ found anthrax spores adhering to a shaving mug after the removal of the brush.

The spores have been carried to the skin by flies; Schuberg and Kuhn⁴⁶

⁴³ *Compt. rend. d. Acad. de sc.*, 1881, 92: 427.

⁴⁴ Smyth and Bricker, *J. Indus. Hyg.*, 1922, 4: 53.

⁴⁵ *J. Infect. Dis.*, 1919, 31: 499.

⁴⁶ *Arbeitem. g. d. kaiserl. Ges.-Amt.*, Bd. 40, Heft 2, 1912.

have shown that anthrax may be transferred from animal to animal through the bite of the stable fly (*Stomoxys calcitrans*). Mitzmain obtained positive results with the stable fly and also with *Tabanus striatus*.⁴⁷

Woolsorters' disease, or anthrax of the lungs, appears to be due to the inhalation of anthrax spores. It is observed only among persons who handle skins or who work with horsehair, wool, or other raw materials from animals afflicted with anthrax. The symptoms are like those of pneumonia; this form is frequently fatal.

The mode of transmission in intestinal anthrax may be through infected meat and other food. The usual heat of cooking or even canning does not necessarily kill anthrax spores. Intestinal anthrax is rare, but when it does occur is rapidly fatal.

Resistance.—The anthrax spore is exceedingly resistant to heat and external influences, such as dryness and sunlight, and also to germicidal agents. Its resistance may be compared to the tetanus spore, page 94.

Prevention.—A number of species of animals have a natural immunity to anthrax, and an artificially acquired immunity may be induced in cattle or sheep through the injection of attenuated cultures, in accordance with the classical method of Pasteur. These procedures are not applicable to man. Besredka⁴⁸ found that infection of guinea-pigs by means of *B. anthracis* can be prevented most efficiently by immunizing the skin rather than the body as a whole. The prevention of the disease in man must first be directed to a suppression of the infection in animals. The sick animals should be isolated, or, better, killed, and the carcasses burned or buried with lime at least three feet deep. The carcasses may be "tanked," that is, subjected to a prolonged exposure to steam under pressure. Tanks for this purpose are found in all the larger slaughter houses. It is important in handling the body of an animal dead of anthrax not to open it or shed blood, for the bacillus does not produce its spore except in the presence of oxygen, that is, the bacilli are mainly in the blood and internal organs and will not sporulate as long as access to the air is prevented.

The neglect of precautions in disposing of anthrax carcasses favors the spread of the infection through the activity of carrion feeders. Morris⁴⁹ has shown that buzzards may carry infection for long distances and contaminate clean ground or water through contamination on their feet and beaks. Dogs discharge anthrax spores in their feces 114 hours after feeding upon an anthrax carcass.

Anthrax spores may live months, even years in the soil. Hastings⁵⁰ found anthrax spores in a pond in a pasture in which the disease had occurred eighteen and one-half years before.

Gegenbauer points out that animals may harbor anthrax organisms in

⁴⁷ *U. S. Pub. Health Rep.*, 1914, 29: 75.

⁴⁸ *Ann. de l'Inst. Pasteur*, 1921, 35: 422.

⁴⁹ *Louisiana Bull.* No. 136, Agricultural Experiment Station, Nov., 1912.

⁵⁰ *J. Infect. Dis.*, 1923, 33: 526.

their hair without themselves being infected.⁵¹ It is stated that the bacilli may grow in the skin.

The proper prevention of anthrax consists in the veterinary control of the disease among animals. The chief preventive measure so far as man is concerned is the disinfection of all raw material in those trades in which horse hair, hides, wool, and other substances liable to harbor the anthrax spore are handled. Veterinary surgeons who conduct autopsies upon anthrax animals should exercise special precautions, and, if practicable, wear rubber gloves.

Workers in leather tanneries, hair and wool factories can be protected by rubber gloves and rubber aprons. Protection is also afforded by an effective quarantine against all hides, wool, and hair from infected and suspected areas, and areas about which information is lacking (see also page 1272).

Proper ventilation should be provided to carry off the dust where hair and wool are handled, especially about carding machines. The refuse from tanneries and woolen mills should be properly disposed of; otherwise they may infect streams or fields through fertilizers.

A serum made by injecting horses with virulent anthrax bacilli is a useful therapeutic agent. Normal bovine serum also has curative value; neither of these sera is practical as a prophylactic.

Disinfection.—Disinfection for anthrax is difficult on account of the extraordinary resistance of the anthrax spores. It is further complicated by the fact that dependable methods are apt to injure the hides.

Steam disinfection is practicable for hair, but not for wool or hides. The wool fiber and hides are seriously damaged by the action of the steam. Wool may be disinfected by formaldehyd used as given below. The method preferred for hides is the hydrochloric acid-salt bath of Schattenfroh. Manufacturers object to the disinfection of hides with bichlorid of mercury and formic acid, according to the Seymour-Jones method, on account of the apparent injury of such treatment.

All hides, wool and hair from anthrax-infected and suspected areas, or areas about which information is lacking, should be disinfected. This disinfection should be done in the country where the hides, wool and hair are collected, preferably at the market center, where the work could be done under skilled supervision.

Hair for Shaving Brushes.—The disinfection of the hair used for shaving and lather brushes, in accordance with the requirements of the United States Government,⁵² is accomplished by one of the following methods: (a) by boiling the hair or bristles for not less than three hours; (b) by exposing the hair or bristles to steam under not less than fifteen pounds gauge pressure for not less than thirty minutes with a preliminary vacuum of not less than ten inches before turning on the steam; (c) by exposure to streaming steam for not less than six hours; (d) by dry heat, 200° F., for twenty-four hours.

Shaving Brushes.—Any brushes found in the market which do not bear

⁵¹ Arch. f. Hyg., 1920, 80: 202.

⁵² Interstate Quarantine Regulations, Treasury Dept., Washington, July 30, 1918.

the name or the trade mark of the manufacturer should be regarded with suspicion, and should be returned to the source from which they were secured, or should be disinfected. Shaving brushes will stand boiling or steaming. The following procedure is believed to be effective:

The brush should be soaked for four hours in a 10 per cent formaldehyd solution. The solution should be kept at a temperature of 110° F. and the brush so agitated as to bring the solution into contact with all hair or bristles.⁵³

Wool.—The Departmental Committee on Anthrax⁵⁴ recommends the following for the disinfection of wool, goat hair and camel hair: A preliminary agitation with an alkaline solution of soap in water at a temperature of 102° to 110° F.; exposure for twenty minutes in a 2 to 2½ per cent solution of formaldehyd in water at a temperature of 102° to 105° F. The hair is then dried in a current of air at 160° F., and allowed to stand a short time in order that the formaldehyd may complete its germicidal action.

Hides and Skins.—1. The Seymour-Jones method: To one pound of perchlorid of mercury add five hundred gallons of water, and to this mixture add five gallons of formic acid (commercial 50 per cent strength). In this bath steep the material for twenty-four hours.

2. The Schattenfroh method: In a 2 per cent hydrochloric acid solution to which 10 per cent of common salt has been added steep the material for forty hours at a temperature of 60°-70° C. A quicker method can be used by substituting a 1 per cent solution of hydrochloric acid and 8 per cent of salt, provided the temperature of the solution is maintained at 40° C. (104° F.) for a period of six hours.⁵⁵ The effectiveness of this method was confirmed by Smyth.⁵⁶

Hair, Bristles and Pigs' Wool.—1. By letting a current of steam act on the material for not less than one-half hour at a pressure of seventeen pounds (0.15 above atmospheric pressure);

2. By boiling for at least one-quarter of an hour in a solution containing 2 per cent of permanganate of potassium, and subsequent bleaching with a 3 or 4 per cent solution of sulphurous acid;

3. By boiling in water for not less than two hours.

FOOT-AND-MOUTH DISEASE

Foot-and-mouth disease is also known as aphthous fever, epizootic catarrh, and eczema contagiosa. It is an acute and highly communicable disease, generally confined to cloven-footed animals, and characterized by an eruption of vesicles on the mucous membrane of the mouth and on the skin between the toes and above the hoofs; sometimes on the udder and other parts of the

⁵³ Report of the Departmental Commission on Anthrax, London, 1918, Vol. II.

⁵⁴ Pub. Health Bur. Cir. Letter, No. 136, July 31, 1918.

⁵⁵ Ind. Bull. No. 6, Commonwealth of Massachusetts, State Board of Labor and Industry, p. 7.

⁵⁶ Am. J. Hyg., 1921, 50: 541.

body. The vesicles rupture, leaving superficial erosions which sometimes develop into ulcers. Other symptoms are: salivation, tenderness of the affected parts, loss of appetite, lameness, emaciation, and diminution in the quantity of milk secreted.

Foot-and-mouth disease is primarily a disease of cattle and secondarily of man. It also affects hogs, sheep, goats, deer, buffalo, American bison, camel, chamois, llama, giraffe, antelope, and even dogs and cats are said to occasionally become infected. Horses and fowl are not susceptible.

The disease prevails in European countries, especially Russia, also South America, Asia and Africa, and occasions great economic loss. The mortality is low; the serious losses depend chiefly upon the diminution of the milk secretion and the loss of flesh in the affected animals as well as the disturbances of quarantine. It occurs as widespread epizootics, especially in the warm season.

Foot-and-mouth disease has appeared in the United States only on eight different occasions: in 1870 in the eastern states; 1880 and 1884, Massachusetts; 1902, New England; 1908, Detroit; 1914, Chicago; 1924, California; and 1925, Texas. Every outbreak on American soil has thus far been followed by its complete suppression through the application of well-known preventive measures, such as isolation, destruction and burial of the affected herds, disinfection, restriction of the movements of cattle, and a systematic inspection of all farms in the infected area to detect cases of the disease. It pays in the end to use prompt and aggressive measures.

Animals may be infected directly, as by licking, and in calves by sucking, or indirectly by fomites such as infected manure, hay, utensils, drinking troughs, railway cars, animal markets, barnyards, and pastures. The spread of the disease is due largely to carrying of the infection on the hands of persons who examine, milk, or otherwise come in contact with diseased animals.

Veterinary inspectors must take unusual precautions not to spread the infection in their efforts to control it.

Löffler and Frosch⁵⁷ in 1898 showed that the virus will pass the finest porcelain filters. This was the first "ultramicroscopic" virus of animals discovered. The first filtrable virus was discovered by Iwanowski⁵⁸ in 1892 as the cause of the mosaic disease of tobacco. In 1898, Beijerinck⁵⁹ independently arrived at similar conclusions. In the same year Löffler and Frosch⁶⁰ published their epoch-making studies on the etiology of foot-and-mouth disease. Frosch continued to work on the subject, and twenty-six years later announced⁶¹ that the cause was a tiny bacillus, which he called *Loeffleria nevermanni*. A British and also a German commission failed to substantiate his results.

The specific principle is contained in the serum of the vesicles; in the

⁵⁷ *Deutsche med. Wchnschr.*, 1898, 24: 80. ⁵⁸ *Beihefte. botan. Centralbl.*, 1893, 3: 296.

⁵⁹ *Centralbl. f. Bakteriol.*, 1899, 5: 27. ⁶⁰ *Centralbl. f. Bakteriol.*, 1898, 23: 371.

⁶¹ *Lancet*, 1923, 26: 962.

saliva, tears, milk, and various other secretions and excretions; also in the blood until the eruption comes out, then it disappears.

No definite immunity is rendered by an attack. The period of incubation is variable, usually from two to six days or longer, exceptional instances being prolonged to fifteen or even eighteen days.

The disease in man is a direct counterpart of that in cattle. The infection is transmitted to man through the ingestion of raw milk, buttermilk, butter, cheese, and whey from animals suffering with foot-and-mouth disease. It may also, though more rarely, be transmitted directly from the salivary secretions or other infected material which gains entrance through the mucous membrane of the mouth. It is doubtful whether the disease can be transmitted to man by cutaneous or subcutaneous inoculations, though it is probable that the infection may be communicated if the virus enters the blood directly through wounds of any kind. Children are most frequently infected by drinking unboiled milk during the time in which the disease is prevalent in the neighborhood; while persons in charge of diseased animals may become infected through contact with the affected parts or by milking, slaughtering, or caring for the animals. The disease is usually mild in man; death practically never results, except in weakened children, and then from secondary complications.

The original experiments of Löffler and Frosch, as well as experiments which have been made in Denmark and Germany, indicate that the virus is destroyed comparatively readily by heat or the usual germicides, except phenols and cresols, which are unreliable for this and other filtrable viruses. Milk pasteurized at a temperature of 60° C. for twenty minutes is safe.

Foot-and-mouth disease has a special interest on account of the fact that it may be associated with vaccinia. Vaccine virus has been known to contain the infection of foot-and-mouth disease.^{61a} Glycerin acts as a preservative for the virus of foot-and-mouth disease, so that it may remain viable in glycerin-ated vaccine virus a very long time. No instance of the transmission of foot-and-mouth disease to man through vaccine virus has been recorded, and it is doubtful, in view of the known facts, whether it is possible to reproduce the disease in man by the cutaneous inoculation commonly used in the process of vaccination. The prevention of foot-and-mouth infection in vaccine virus is assured through federal inspection and through special tests (see Vaccine Virus, page 19).

The prevention of foot-and-mouth disease consists (1) in a cattle quarantine, to keep it out of countries where it does not exist; (2) in the elimination of the disease in cattle through isolation and destruction of infected herds, destruction and burial of all the affected and exposed animals, and disinfection; (3) the disease in man may be avoided by care in the selection of the animals from which milk is taken or by pasteurization of the milk when foot-and-mouth disease is prevalent.

^{61a} "The Origin of the Recent Outbreak of Foot-and-Mouth Disease in the United States," by Mohler and Rosenau, *Cir. 147*, Bureau of Animal Industry, United States Dept. of Agriculture, 1909.

MALTA FEVER

Malta fever is a general infection not unlike other specific bacteriemias, such as typhoid fever. The cause was discovered by Bruce in 1887, during the earlier days of bacteriology, and called by him *Micrococcus melitensis*.⁶² Clinically the disease is characterized by profuse perspiration, constipation, frequent relapses often accompanied by pains of a rheumatic or neuralgic character, and sometimes swelling of the joints or orchitis. Malta fever is further remarkable for its low mortality and long-drawn-out and indefinite duration. It prevails especially about the Mediterranean basin. It is also endemic on the Rio Grande and the southwestern section of the United States. Malta fever is a disease primarily of goats and occasionally other animals, secondarily of man.

Gentry and Ferenbaugh⁶³ in 1911 found a nest of Malta fever throughout the older goat-raising sections of Texas. This endemic center embraces an area approximately three hundred miles along the Rio Grande extending ninety miles to the north. An outbreak which occurred in Arizona was described by Lake.⁶⁴ In the southwestern section of the United States, Malta fever is traceable to goats. The patients give histories of drinking unboiled goat's milk or being associated with the goat-raising industry. In recent years it has been found that Malta fever occurs occasionally in all sections of the United States.

Brucella melitensis.—*Brucella melitensis* is a small coccobacillus occurring singly and in pairs, sometimes in short chains; it has no spore and is Gram-negative. The first culture generation from blood or urine requires several days to develop. After about four days of incubation, minute dewdrop colonies appear. After having become established on ordinary media the cultures may be kept alive indefinitely by transplanting every six or eight weeks.

Sometimes high agglutinating power develops in persons suffering with Malta fever—titers of 1:5,000 are not uncommon. On the other hand, in some cases the agglutinating power is so low as to be misleading. A negative agglutination reaction does not always signify that the patient is not infected with *Brucella melitensis*. In cases with high agglutinating power, the proagglutinoid zone may appear, that is, the serum may refuse to agglutinate in low dilutions, such as 1:100, but agglutinate actively in higher dilutions, such as 1:1,000.

Brucella melitensis is readily destroyed by heat. I have shown that 60° C. for twenty minutes is sufficient to destroy this organism in milk and provides at the same time a liberal margin of safety. It is not destroyed at 55° for a short time, but succumbs in one hour; the majority die at 58°; at 60° all are

⁶² This organism is not a micrococcus. It goes by various generic names: Bacterium, Brucella and Alcaligenes. *Brucella melitensis* is the one most commonly used in current publications.

⁶³ *J. Am. M. Ass.*, 1911, 57: 889, 1045, 1127.

⁶⁴ *U. S. Pub. Health Rep.*, 1922, 37: 2895.

killed. Phenol, 1 per cent, destroys the coccus in fifteen minutes. While this micrococcus shows a comparatively feeble resistance against heat and the ordinary germicides, it shows a remarkable resistance to dryness, for it may remain alive in this state for months.

Relationship between Malta Fever and Contagious Abortion.—In 1918 Evans⁶⁵ called attention to the fact that by ordinary laboratory methods it is impossible to distinguish the causal organism of Malta fever from the so-called *Bacillus abortus* which causes contagious abortion in cattle and hogs and other domestic animals. These observations were confirmed and amplified by Meyer and Shaw⁶⁶ and by a number of investigators in foreign countries. Evans⁶⁷ has studied the subject farther and found that there are a number of related organisms which can be distinguished only by the agglutinin absorption test and which may be regarded as varieties of the *melitensis* species. It appears that any one of these varieties may infect any kind of domestic or laboratory animal, or man. Certain varieties, however, are characteristic for given host species. There are several varieties which commonly infect goats, and are readily transmitted to man. The *abortus* variety causes a widespread disease in cattle, and is commonly present in cow's milk. It also causes contagious abortion in hogs. On account of its frequent occurrence in cow's milk, and the rarity of the human disease in regions where cow's milk is used, the bovine variety must be regarded as of comparatively slight virulence for man. During recent years, however, at least fifteen human cases of *abortus* infection have been recognized in this country, and a considerable number of cases have been reported from Italy and from South Africa. A considerable percentage of cases which have occurred in this country have been in men who handled hog carcasses in slaughterhouses. This suggests that *abortus* strains of porcine origin are more virulent for man than strains of bovine origin. Human *abortus* may be more frequent than we now suspect, cases going unrecognized. It may be mild or as severe as when contracted from goats. One of the human cases of *abortus* infection in the United States was fatal.

Modes of Transmission.—From experimental evidence it would appear that the infection of Malta fever may be taken in through wounds, the mucous membranes, or by food and drink. The usual mode of infection is by drinking raw goats' milk. *Brucella melitensis* leaves the body in various secretions and excretions. Great numbers of the cocci in pure cultures may appear in the urine. The milk of goats also contains the virus. All the secretions from the body must be regarded as infectious until further knowledge on the subject is at hand. In man the coccus may be isolated from the feces, blood, urine, spleen, lymph glands, bone marrow, and mammary glands. In goats it first disappears from the blood, then the spleen, and, last of all, from the mammary glands.

Goats are susceptible to Malta fever and continue to discharge the infec-

⁶⁵ *J. Infect. Dis.*, 1918, 22: 580.

⁶⁶ *J. Infect. Dis.*, 1920, 27: 173.

⁶⁷ *U. S. Hyg. Lab. Bull.*, No. 143, 1925.

tion in the milk for a long time. The disease is usually contracted by drinking such infected milk. While this is the common mode of infection, occasional cases doubtless arise through other sources; thus one case which arose in England is supposed to have been conveyed from son to father by using a clinical thermometer in the mouth immediately after its use by the patient. Monkeys may readily be infected either by the inoculation of pure cultures or by feeding them. There are few diseases transmitted to laboratory workers so readily as Malta fever; in fact, most investigators of this disease contract it and are unable to trace their infection to any definite accident. Macfayden lost his life from a laboratory infection with *Brucella melitensis*. This micro-organism has, therefore, more than complied with all the requirements of Koch's laws.

There has long been a suspicion that Malta fever may be conveyed through the bite of an ectoparasite. In fact, Captain Kennedy was able experimentally to infect a monkey as a result of bites of mosquitoes (*Culex pipiens*) which had fed on patients suffering with Malta fever. This probably was an instance of mechanical transference of the infection, corresponding in all respects to a laboratory inoculation with fresh virulent material from a hypodermic syringe. This cannot be a frequent way by which the infection is transmitted in nature, for the specific organisms are found in small numbers in the peripheral blood of Malta fever patients. The British Commission found *Brucella melitensis* only four times from a total of 896 mosquitoes studied.

While the drinking of goats' milk is the usual way in which the disease is transmitted, the possibilities are varied and correspond somewhat to those of typhoid fever. The fact that the micrococcus may be successfully introduced either by ingestion, or by inoculation, or through the mucous membranes, makes it evident that occasionally the disease may be contracted by means of insect bites and other wounds, infected food, and the various modes of contact infection. Contact infection, however, probably plays a minor rôle, for there is evidence that the disease is not, as a rule, directly transmitted from the sick to the well.

There is also experimental evidence to show that monkeys can be infected by dry dust artificially contaminated with cultures of *Brucella melitensis*. The path of entrance may be through the nares, throat, respiratory passages, or alimentary canal. Dry dust contaminated with the urine of cases of Malta fever has given rise to infection in goats but not in monkeys. The experience gained during the work performed in Malta during 1904 and 1905 has convinced Horrocks that men are more susceptible than monkeys and goats. Shaw's work on ambulatory cases among Maltese has also shown that opportunities for the creation of infected dust were plentiful in Malta. Infected dry dust as a mode of transmission cannot, therefore, be discarded.

Goats' Milk and Malta Fever.—We are indebted to the six reports of the British Commission for the Investigation of Mediterranean Fever (1905-1907) for the fact that Malta fever is chiefly spread through goats' milk.

Before the researches of this commission the common mode of infection was not definitely known.

The usual source of milk in Malta was the goat. The udders, which are abnormally long, often touch the ground and are very liable to be soiled. It was first shown by Zammit in the report of 1905 that goats could be infected by feeding them with *Brucella melitensis*. In the same year Major Horrocks discovered *Brucella melitensis* in the milk of an apparently healthy goat. Further studies showed that one or more healthy goats in every herd were excreting *Brucella melitensis* in their milk and urine, and that about 50 per cent of the goats reacted positively when examined by serum agglutination tests. All the available evidence points to their food as the main vehicle of infection in goats. The young goats, of course, are infected through their mother's milk. Horrocks and Kennedy considered that 10 per cent of the goats supplying milk to various parts of Malta excreted *Brucella melitensis* in their milk. The excretion of the specific microorganisms may continue steadily for three months without any change occurring in the physical character or chemical composition of the milk and without the animal exhibiting any signs of ill health. On the other hand, the excretion of *Brucella melitensis* in the milk may be intermittent, appearing for a few days and then disappearing for a week or more.

Major Horrocks in *Report Number 5* of the British Commission shows a direct relation between the number of goats in Gibraltar and the number of cases of Malta fever. With the reduction in the number of goats in Gibraltar there was also a decrease in the number of cases, so that finally, when the number of goats had decreased to about two hundred, in 1905, Malta fever had practically disappeared.

The story of the steamship *Joshua Nicholson* is instructive in showing the relation between goats' milk and Malta fever in man. Sixty-one milch goats, all healthy in appearance and good milkers (many being prize animals), and four billygoats were shipped on board the cargo steamer *Joshua Nicholson* August 19, 1905, at Malta for passage to the United States via Antwerp. Many of the ship's company partook freely of the milk. The officers drank "mixed" milk collected in a large vessel; the members of the crew each obtained the "whole" milk from one goat in his own separate pannikin. Subsequent bacteriological examination resulted in the recovery of *Brucella melitensis* from the milk of several of the goats. Of twenty-three men on board the steamer who drank the goats' milk on one or more occasions, no evidence whatever is available as to thirteen, while of the remaining ten, nine suffered from febrile attacks, five of them yielding conclusive evidence of infection with *Brucella melitensis*.

Prevention.—Our knowledge of the cause and modes of transmission of Malta fever makes the prevention of this disease a comparatively simple problem. The infection must first be eliminated in the goats. Until this is done goats' milk should be pasteurized. The possibility of occasional infection from cows' milk also demands the protection of pasteurization. Patients

having the disease should be treated upon the same principles laid down for typhoid fever, in order to prevent the spread of the infection through food, fomites, and contact. Convalescents should not be released until the micrococcus has disappeared from the urine. General sanitary measures, such as the suppression of flies and mosquitoes, allaying dust, and the promotion of general cleanliness, should not be neglected.

YAWS

(*Frambæsia tropica*)

Yaws is a communicable infection caused by the *Treponema pertenue*, described by Castellani in 1905. It is not a "venereal" disease, but closely resembles syphilis. It is named from the raspberry-like (frambesiform) appearance of the eruption.

Yaws is essentially a tropical disease and exists in Africa, especially on the west coast; in Asia, it is common in the Malay Peninsula, Assam, Upper Burma, Siam, Java, Batavia and Ceylon. In America, it is common in the West Indies, in British Guiana, Venezuela, Colombia and Brazil. Cases have been reported from the southern United States, although it is rare in this country. Yaws also occurs in northern Australia and in many of the Pacific islands.⁶⁸ A disease in Guam known as gangosa is regarded as a tertiary form of yaws. One of the characteristics of yaws is that it prevails especially in country districts.

Treponema pertenue of yaws closely resembles *Treponema pallidum* of syphilis. They are distinguished only by animal tests; thus, monkeys immunized with *pertenue* do not become immune for *pallidum*. The parasite is found constantly in the primary lesion and in the unbroken papules of the general eruption. It may also be found in the spleen, lymph glands and bone marrow.

Paulet in 1848 inoculated fourteen Negroes with the secretions from frambetic granulomata. All of them developed yaws in from twelve to twenty days. Syphilitic patients may contract yaws naturally and experimentally; yaws patients may likewise contract syphilis. The two diseases are therefore distinct. However, in persons who have had yaws a positive Wassermann reaction seems to be given in a higher percentage than is true for syphilis. Yaws is a disease of the rural population, while syphilis prevails more particularly in cities. Yaws is a much less serious disease than syphilis, as it does not attack the central nervous system or viscera and is not transmitted hereditarily. The primary lesion does not occur on the genitalia. Furthermore, it is much more easily eradicated by arsphenamin.

Yaws is usually conveyed by direct contact. It appears, however, that

⁶⁸ It has been suggested by Hume, Adams and others that yaws was the disease which afflicted the Israelites during their emigration from Egypt, and that therefore the term "saraat" in the thirteenth chapter of Leviticus does not mean leprosy as usually translated.

there must be some abraded surface or small wound, for the parasite probably cannot enter through the normal skin. Women are frequently infected by their children, the primary lesion appearing on the mammae. Castellani believes that the virus may also be transmitted by insects, especially flies.

Arsphenamin and similar spirocheticides are efficacious. Bismuth and sodium and potassium tartrate give good results. Mercury is useless. Prevention consists therefore in the treatment of all active cases so as to destroy the foci of infection. Segregation may be practicable. Personal prophylaxis in endemic centers consists in avoiding native contact and promptly treating the slightest abrasion of the skin with an active germicide. Patients should be isolated until cured. The skin lesions should be dressed and other measures used to guard against infection through the agency of flies and other insects. Traveling clinics in endemic centers for the administration of salvarsan, organized after the manner of the hookworm work, could control yaws. Work of this kind has been demonstrated in Santo Domingo.⁶⁹ Such work must be followed up to control the disease.

LEPROSY

Leprosy is a chronic specific infection, characterized by tubercular nodules in the skin and mucous membranes (tubercular leprosy) or by changes in the nerves (anesthetic leprosy). These two forms may be separate, but ultimately are combined. Acute exacerbations and remissions occur. Nodular cases live eight to ten years on the average; anesthetic cases live on indefinitely. Leprosy is contagious, but with difficulty and only under special conditions. The fear of leprosy is almost without parallel, due to biblical influence. Among many people, however, especially where the disease is most prevalent, a leper is regarded with no greater concern than is a syphilitic with us.

Incubation.—It is difficult to place the period of incubation with any degree of certainty. It is usually stated as from six to eight years, but may vary from a few months to twenty years. The disease both in the individual and in the community develops very slowly. Leprosy commonly exists several years before it is recognized.

Occurrence, Geographic Distribution, History.—According to Leonard Rogers ⁷⁰ there are 1,370,277 known lepers in the world. There must be many more, for accurate figures are not available because leprosy prevails most in countries that give least heed to vital statistics. It is estimated that in China alone there are about 1,000,000 lepers; in Africa probably 500,000, where the disease is still spreading; the last figures from India count 102,000.

The figures for the countries chiefly afflicted are given in the following table: ⁷¹

⁶⁹ W. L. Moss, G. H. Bigelow, *Johns Hopkins Hosp. Bull.*, 1922, 33: 43.

⁷⁰ *Leprosy*, J. Wright & Sons, Ltd., Bristol, 1925.

⁷¹ Condensed from Table I, *Leprosy*, by Sir Leonard Rogers and Ernest Muir, John Wright & Sons, Ltd., Bristol, 1925.

Country	Authority	Year	Number of Known Lepers	Ratio per 1,000
Norway	Hansen	1856	2,833	1.91
Norway	Lie	1919	180	0.03
Korea	Cochrane	1923	30,000	1.70
Japan	Papellier	1912	102,585	1.94
China	Van Vioten	1916	1,000,000	2.50
Egypt	United States Report	1915	6,513	0.58
South Africa	Colonial Report	1920	2,248	0.31
Argentina	Mission Report	1920	1,000	0.12
French Guinea	Joyeux	1912	8,687	5.00
North Nigeria	Dalziel	1914	5.20
Ubangi-Shari-Chad	Vallet, Jumot	1922	1.70
Upper Senegal Niger	Vallet	1913	4,000	10.00
Cameroons	Zieman	1902	20.30
Gabon (French)	Ringerbach and Guvomerch	1915	16.60
Middle Congo	Ringerbach and Guvomerch	1915	10.60
Middle Congo	Daniel	20,000	40.00
East Belgian Congo	Coronisio	1909	200.00
Tanganyika Territory	Colonial Report	1924	11,480	2.8
Madagascar	Suddey	1919	6,373	2.02
South Rhodesia	Colonial Report	1921	1,000	1.11
India	Census Report	1921	102,513	0.32
Philippines	Wade	1922	5,232	0.69
Indo-China	Jeanseime	1900	15,000	0.67
New Caledonia	Leboeuf	1914	949	21.10
Loyalty Island	Ortholon	1912	219	19.50
Marquesas Islands	Grosfillex	1906	3,480	33.05
Hawaii	Hoffman	1919	611	2.32*
Cuba	Matias Duque	1907	1,500	0.73
Dutch Guiana	Boes van Dort	1907	2,000	25.00
Brazil	Vernet	1923	15,000	0.72
Colombia	Urila	1922	6,568	1.31

* This rate is based on cases in the Molokai Settlement alone (see text).

The whole story of the spread of leprosy over the globe is one long record of affected persons carrying it to countries previously free. As a rule, it increases very slowly and insidiously at first, taking several decades before it attracts attention. The only countries from which it has disappeared entirely are the temperate zone areas of Europe, due probably to improvement in the standards of living.

Every country now having a high leprosy rate (above 5 per 1,000) is tropical, with high annual rainfall and a hot, damp climate. It is endemic in some cold countries, as Norway, Iceland and the Baltic states. On the other hand, one of the striking features of leprosy is that it shows little tendency to spread in certain localities.

In Europe.—Leprosy prevailed in epidemic form in Europe in the middle ages, but the disease has disappeared from central Europe, remaining only upon the fringe of the Continent, in Norway, Sweden, Spain, Portugal, Italy, Greece, Turkey, Russia, and Finland. There are a considerable number of cases of local origin in Bosnia and Herzegovina, and a few in Memel, France, and Bulgaria.

Five or six hundred years ago, leprosy was epidemic in Europe; with

one-tenth of the population of to-day, it is estimated to have had 19,000 leprosaria. In the thirteenth century, there were said to be two thousand leper houses in France alone.

Leprosy spread to Europe from the Orient. It was unknown in Italy until the return of Pompey's soldiers from the East in 62 B.C. Galen wrote of the disease in Germany in A.D. 180. Four centuries later it became so common and widespread that according to Virchow there were then 636 leper houses in Italy. In the fifth and sixth centuries Spain was infected by Roman troops, and leprosy became common by the tenth century, and from there spread to France. Newman states that the first known leper hospital in England was established in Nottingham in A.D. 625; in Ireland in 868. Simpson showed that leprosy had reached Wales by 950. The disease was prevalent in Great Britain from the eleventh to the fourteenth century, reaching its height in the twelfth.

Leprosy appears to have died down in European countries much in the order in which they were attacked. Its rapid decline during the fourteenth and fifteenth centuries is perhaps the most remarkable feature in the long history of the disease, and regarding its cause there has been controversy.

In America.—Leprosy was brought to America (Cuba and Brazil) after its discovery by the Spaniards. A government commission in 1902⁷² took a census of the lepers in the United States and found 278. Of these 145 were born in the United States and 186 probably contracted the disease in the United States. Of the entire number 72 of the cases were isolated and 205 were at large. Brinckerhoff again studied the prevalence of leprosy in the United States in 1909 and found 139 cases. The official figures for 1912 were 146.⁷³ In 1925, the National Home for Lepers at Carville, Louisiana, had about 250 cases. Competent leprologists believe that the total number of cases is not far from five hundred. Denney places the number at approximately twelve hundred.

It is evident that the disease is not markedly on the increase in our country and that, while it may be contracted here, it is "contagious" with great difficulty. There are three foci of leprosy in the United States: one among the Scandinavians in the region of the Great Lakes, made up almost exclusively of imported cases; another among the Orientals and Mexicans on the Pacific Coast, likewise chiefly of imported cases, and the third on the Gulf Coast, particularly in Louisiana, Texas and Florida, where most of the cases are native born. According to the most recent figures available the number of cases in isolation in our insular possessions is as follows: Hawaii (1924),

⁷² White, Vaughan, and Rosenau, *Document No. 269*, 57th Congress, 1st Session, 1902.

⁷³ Complete returns from only eighteen states. The incompleteness of the official returns is indicated by the fact that New York State acknowledged only five cases, whereas at a medical meeting more than that number of cases were shown in New York City alone. The laws concerning leprosy vary in the different states. New York, for example, has no stringent laws and there are forty or fifty cases at large in New York City. Pennsylvania and Massachusetts, on the other hand, enforce strict segregation.

624; Porto Rico (1922), 44; Philippine Islands (1922), 5,522; the Canal Zone (1923), 81. The lepers in Guam, eighteen in number, were transferred to the Philippine leper colony at Culion during 1913. It is known, however, that everywhere that the disease prevails many cases escape tabulation in the official returns.

In Oceanica.—During the latter part of the nineteenth century, outbreaks of a startling nature took place in certain islands in the Pacific, especially in the Hawaiian Islands, New Caledonia and the French Marquesas Isles. The greatest incidence is found among the natives of the Hawaiian Islands, where one in every forty or fifty has the disease. In forty years close to five thousand cases were reported.

In the Hawaiian Islands.—The disease is believed to have been brought to Hawaii by Chinese following the finding of gold in California in 1848. The first report is that of Hillebrand in 1863, who found it then to be rapidly spreading. He states that it was introduced by the Chinese in 1848, the first local case being seen five years later, and at the end of ten years it had spread considerably in the immediate neighborhood of the first case. This is the common story. By 1864 it had spread to the other islands of the group. It is said that the Chinese likewise introduced leprosy into New Caledonia and the Loyalty Islands. A leprosy hospital was established at Molokai, but the conditions were deplorable and it served small purpose until the improvements brought about by Father Damien's devoted labors from 1873 to 1889. The precise prevalence of the disease cannot be known because so many cases were hidden until a late stage. The cases in the Molokai Settlement alone numbered from 8.67 to 11.88 per thousand from 1870 to 1894, after which a steady fall took place coincident with more efficient segregation under American supervision. The rates declined to 2.3 per thousand in 1915, where they remained. The latest figures are 624 under detention in 1924.

Etiology.—Hansen ⁷⁴ in 1879, after six years of careful study, described the bacillus of leprosy, *B. (microbacterium) lepræ*. This was confirmed the same year by Neisser ^{74a} and subsequently by many other students of the disease. The bacilli are acid-fast and in other respects resemble the tubercle bacillus, except that they lie chiefly within the cells, grow with difficulty if at all on artificial culture media, and are not pathogenic for the lower animals. They are present in greatest numbers in the tubercular nodules of the skin and mucous membranes, but are widely disseminated throughout the body and even occur in the blood during the febrile stage which precedes the breaking out of fresh tubercles. The bacilli of leprosy seem to grow within certain cells, for they are found crowded in great numbers within the peculiar round and oval cells of the granulation tissue. In the anesthetic form they occur most frequently in the nerves and less frequently in the skin. They have

⁷⁴ *Virchow's Arch.*, 1879, 79: 32.

^{74a} *Breslauer Artzl. Ztschr.*, 1897, 20.

been encountered in the spleen, liver, and testicles; in fact, in all the lesions of the disease. Acid-fast bacilli resembling leprosy bacilli also occasionally appear in the feces and urine. They may occur in the expectoration.

From the standpoint of prevention, the location and number of the bacilli are important. They leave the body from any of the lesions that are broken down. They are discharged in enormous numbers from the degenerated nodules of the skin and mucous membranes. If we may depend upon microchemical evidence, it appears that many of these bacilli are probably dead. They occur in the discharges from the mouth and nose, for lesions in the throat and nose are common. The bacilli that remain locked up in the nerves in the anesthetic cases present no public health hazard.

Hansen's bacillus is accepted as the cause of leprosy because it is invariably associated with the disease and often in great numbers, but Koch's laws are far from being complied with. We really know little about this bacillus. For years it evaded all attempts at cultivation. Clegg,⁷⁵ in 1909, succeeded in cultivating an acid-fast bacillus, probably *B. lepræ*, in symbiosis with amebæ and cholera vibrios on agar. These results have been confirmed by Currie, Brinckerhoff, Holmann and McCoy in Hawaii and by Duval in New Orleans. The work up to 1914 is summed up by Wolbach and Honeij.⁷⁶ The cultures obtained are either diphtheroids or acid-fast bacilli, some of which are chromogenic and grow luxuriantly on artificial culture media, suggesting saprophytism. There is no satisfactory evidence that any of the cultures isolated will reproduce the disease in experimental animals.

Immunity.—Leprosy occurs spontaneously in man only. There is no racial immunity to leprosy. The white race suffered severely during the middle ages. Malays, Mongols and Negroes now appear most liable to the infection, perhaps on account of their mode of life. The disease is remarkable for its prolonged period of incubation and its chronic course. The lesions may not be noticed until twenty years after infection. The disease is not usually recognized until several years have elapsed after the first manifestations. These facts indicate that the body must possess a high degree of resistance to this infection. Leprosy even shows a tendency to self-healing. Immunity seems to be gradually produced in some cases.⁷⁷

Children over five years of age and young adults, particularly around the age of puberty, are especially susceptible; while over the age of thirty years the liability to contract the disease, even when exposed to contact with lepers, rapidly decreases, although no age period is entirely exempt. Fifty per cent of cases occur before twenty years of age, and 66 per cent before twenty-five years; few cases start after thirty years. In this regard leprosy is somewhat like tuberculosis. These well-established facts are of the utmost importance in relation to prophylaxis.

⁷⁵ *Philippine J. Sc.*, 1909, B, 4: 77.

⁷⁶ *J. Med. Research*, 1914, 29: 367.

⁷⁷ E. Muir, *Lancet*, 1924, 1: 277.

There are twice as many male lepers as females. This may be due to relative susceptibility of the sexes, or to modes and opportunities of infection.

There are interesting immunological relationships between leprosy, syphilis and tuberculosis. All three diseases may coexist. Many lepers become tuberculous, especially in the late stage; in fact, leprosy is one of a number of infections that predispose to tuberculosis. Tuberculosis and leprosy have many points of resemblance. There is special susceptibility in childhood and early adolescence; in both diseases resistance develops with maturity. Lepers react to tuberculin both generally and locally, although somewhat less than the tuberculous. Analogy with tuberculosis suggests that in endemic foci many become infected with lepra bacilli but have sufficient resistance to prevent the disease reaching the threshold of clinical manifestations. Rogers believes that at least 50 per cent of persons infected do not develop leprosy.

Many lepers are also syphilitic. Syphilis is believed to lower resistance to leprosy, and thus act as a predisposing cause. A positive Wassermann is found in from 15 to 95 per cent of lepers. Rogers reports 62 per cent of positive Wassermann reactions among leprosy children, with only 17 per cent among non-leprosy children. Kölmer and Denney,⁷⁸ however, claim that by their new method of carrying out the Wassermann test, a positive is never given by lepers unless accompanied by syphilis. They found clinical or serological evidence of syphilis in twenty-seven, or 17 per cent, of a series of 159 cases of leprosy.

The Experimental Disease.—Leprous material has been inoculated into human beings by Danielson, Profeta, Cagnina and Bargilli with negative results. These experiments demonstrate the resistance of the body to the virus. However, most of these experimental attempts were done on adults who are resistant to this infection. In Arning's well-known case of the convict Keanu, who was pardoned on condition that he allow himself to be inoculated with leprosy, the disease did develop, but the experiment is somewhat spoiled by the fact that the man lived in a leprosy focus and had lepers among his relatives. In this case a piece of leprosy material was planted into the subcutaneous tissue of the left arm. One month after the inoculation, pain appeared in the arm and shoulder and four and one-half months later a typical leprosy nodule was found. Four years after the inoculation, the patient was a typical leper.

Many unsuccessful attempts have been made to transmit leprosy to the lower animals. Lesions simulating leprosy have been reported in the guinea-pig (Clegg); in the Japanese dancing mouse (Sugai); in rats (Marchoux); in the monkey (Nicolle, Duval). It is questionable, however, whether the disease has been reproduced in the lower animals. Rat leprosy needs separate mention.

Rat Leprosy.—There is a disease among rats which is a close counterpart of leprosy in man. It occurs naturally in *Rattus norvegicus* and may be transferred by inoculation to the more tractable laboratory white rat. The

⁷⁸ *Arch. Dermatol. & Syphilol.*, 1923, n.s., 8: 63.

disease was first observed by Stenfansky in 1903 in Odessa. In the same year Rabinowitsch found the disease among the rats of Berlin, and Dean in 1903 discovered it independently in London, and in a later publication (1905) reported success in transferring the infection by artificial inoculation. Since then rat leprosy has been found by Tidswell in the rats of Sydney, Australia; by Kitasato in Japan; Marchoux and Lebœuf in Paris, and the English Plague Commission observed the disease among the rats in India. Wherry and McCoy found a number of cases among the rats caught in San Francisco, California.

The proportion of rats infected with rat leprosy in different localities varies greatly; thus in Odessa and Paris from 4 to 5 per cent, in San Francisco 0.2 per cent, and in Sydney only 0.001 per cent. Currie failed to find leprosy among the rats of Honolulu. The fact that the infection is absent among the rats of Honolulu and present among the rats in Berlin suggests that it plays no part in the epidemiology of the human disease.

Leprous rats in a late stage of the disease are usually recognized by the presence of patchy alopecia associated with cutaneous and subcutaneous nodules which may be the site of open ulcers; only in advanced cases are the internal organs affected. The diagnosis is readily confirmed by microscopic examination of a smear from an ulcer or a nodule, which will show the acid- and alcohol-fast bacillus of the disease in enormous numbers, and mostly in the cells.

Currie has shown that rats may infect each other by contact, also that bacilli of rat leprosy may often be demonstrated in the heart's blood of infected rats. Currie and also Marchoux and Sorel had no difficulty in demonstrating the presence of acid-fast bacilli in mites taken from the bodies of rats when the latter's heart's blood contained the microorganisms. Uchida⁷⁹ found that rat fleas on the body of a leprosy rat carry many acid-fast bacilli. The fact that these insects so frequently contain the bacilli naturally leads to the suspicion that they may be one of the means of transmitting the infection from rat to rat, but up to the present time no positive evidence has been adduced that such is the case.

Marchoux⁸⁰ inoculated rats with the spleen tissue from a man with a leprosy-like condition and produced a disease in rats indistinguishable from their spontaneous leprosy.

Mezincescu, using rat leprosy antigen, obtained complete complement fixation with human leprosy serum; this confirmed Slatineau's work. Numerous other observers have, with different antigens and serum, obtained positive reactions. This, however, does not clearly tie together the two diseases, for the immunological reactions in the diphtheroids and group of acid-fast bacilli are comparatively feeble and not very specific. The bacillus has been cultivated by Dean, Hollman, Chapin, and others.

⁷⁹ *Japan. Med. World*, 1922, 2: 4.

⁸⁰ *Paris méd.*, 1923, 49: 313.

In this leprosy-like disease of rats we have an infection which closely resembles leprosy in man. The fact that the infection may be propagated in a laboratory animal permits of its investigation, and it is hoped that further studies upon rat leprosy will throw light upon the modes of transmission and control of the human disease.

Contagiousness.—Leprosy is communicable but with difficulty. Ordinarily, it requires close and prolonged association with a leper to contract the disease. It is a common story for lepers to give a history of long and intimate contact with a case, such as sleeping in the same bed and wearing the clothes of a leper. Even then only 3 to 5 per cent of those living in houses with lepers contract the disease.

While the disease is transmitted with difficulty, doctors, nurses, sisters of charity, ward tenders, and others directly exposed in leprosaria sometimes become infected. Notable examples have been Father Damien at Molokai, Hawaii; Father Bogliolo in New Orleans; Sir George Turner in Pretoria; and Mary Reed in India. About 5 per cent of the healthy consorts of lepers become infected at the Hawaiian settlement.

The conditions which favor the spread of leprosy are low stages of civilization and hygiene, defective and overcrowded houses, promiscuity, geographic location and absence of fear of the disease. Contrariwise, leprosy shows little tendency to spread in any of the more highly civilized nations practicing personal cleanliness and enjoying the benefits of modern sanitation. The tubercular form is much more infective than the anesthetic cases.

Modes of Transmission.—Leprosy is evidently a contact disease, but just how it is caught we do not know. The prevailing notion is that the bacilli invade the body through the skin or mucous membranes. The bacilli may leave the body in any of its secretions or excretions. They are contained in enormous numbers in the matter from broken-down nodules of the skin or mucous membranes. As the nodules often affect the throat and nose, these discharges may be infectious. Even the feces and urine contain acid-fast bacilli.

In view of the doubt concerning the way the leprosy bacillus enters the body, the different possibilities deserve consideration. The site of the lesions, the course of the disease and the results of experiments make it probable that the bacilli usually enter through the skin. It is also possible for them to enter through the mucous membranes of the nose or throat, or through the digestive tract, or even during coitus. Food has been accused; insects are suspected.

It may be definitely stated that leprosy is not due to the eating of any particular food. Jonathan Hutchinson stoutly maintained that leprosy was due to a fish diet, but there is no satisfactory evidence in favor of the fish theory, and much against it. One thing is plain, and that is, leprosy is not contracted from any of the lower animals, but is an infection which passes somehow rather directly from man to man.

The suspicion that parasitic insects may play some rôle in the transmission

of leprosy has existed for some time. The evidence is reviewed by Nuttall,⁸¹ who says: "It appears that Linnæus and Rolander considered that *Chlorops (musca) lepræ* was able to cause leprosy by its bite." Blanchard and Corrodor tell of flies in connection with leprosy. Flies frequently gather in great numbers on the leprosy ulcers and then visit and bite other persons. An observation by Boeck of the presence of *Sarcoptes scabiei* in a case of cutaneous leprosy led Joly to conclude that these parasites might at times serve as carriers of the infection. Pediculi are usually present among the poor classes in Algeria, which furnish the greater number of lepers. Sommer of Buenos Aires expresses the belief that mosquitoes act as active agents in the spread of leprosy in warm countries. Carrasquillo of Bogota found the bacillus of Hansen in the intestinal contents of flies. The British Leprosy Commission investigated the possible rôle played by insects with entirely negative results. Wherry studied the occurrence of lepra-like bacilli in certain flies and their larvæ. He found that the fly *Chlorops vomitoria* took up enormous numbers of lepra bacilli from the carcass of a leper rat and deposited them with their feces, but the bacilli apparently do not multiply in the flies, as the latter are clear of bacilli in less than forty-eight hours. Larvæ of *Chlorops vomitoria* hatched out in the carcass of a leper rat become heavily infested with lepra bacilli. If such larvæ are removed and fed on uninfected meat they soon rid themselves of most of the lepra bacilli. A fly, *Musca domestica*, caught on the face of a human leper was found to be infested with lepra-like bacilli. The horrid sight of flies swarming about leprosy lesions and the nostrils of leprosy beggars is well known to travelers in eastern countries. Lepra-like bacilli have been found in bedbugs and these insects have long been associated with the spread of the disease.

The evidence bearing on the possible rôle of insects in the transmission of leprosy is based upon analogy and is largely presumptive. Further, not all acid-fast bacilli are leprosy bacilli. The final verdict will depend upon further studies.

A great majority of lepers at some time in the disease have lepra bacilli⁸² in their nasal secretions. The importance of the nose in leprosy was brought into prominence at the First International Leper Conference in 1897 by the work of Sticker, who made sweeping statements concerning the nose as the site of the primary lesion and the danger of nasal secretions in transmitting the disease. Jeanselme and Laurans (1895), Gerber (1901), Werner (1902), Sheroux (1903), and others have shown the frequency with which the bacilli of leprosy appear in the nasal secretions and the importance of the nose as a site of leprosy lesions. Brinckerhoff and Moore, however, who made a careful study of this question in Honolulu, point out that most of the studies upon the importance of the nose in leprosy have been made upon relatively advanced

⁸¹ Johns Hopkins Hospital Reports, 1900, 8: 1.

⁸² Acid-fast bacilli, resembling the bacillus of Hansen, are called "lepra bacilli." There are many acid-fast bacilli, and we have no clear criterion to differentiate the bacillus of leprosy.

cases. They found the nose frequently the seat of infection when the disease is well developed, but practically never as a primary or incipient lesion. If there be a primary lesion of leprosy it has not been discovered. Stricker's view that nasal lesions are frequently primary has not been confirmed. If the nose were the usual seat of the primary lesion in leprosy, it might indicate that the infection is carried there upon the finger.

Jeanselme⁸³ reports observations supporting the Chinese and Japanese belief that leprosy spreads largely by sexual contact. Jeanselme found "leprosy" urethritis set up by lepromata which invaded the navicular fossa. He further states that myriads of acid-fast bacilli may be found in a drop of pus which may be squeezed from the meatus under this condition. The fact that more males than females (about 2 to 1) have leprosy bears on this mode of transmission.

There is no evidence that leprosy is inherited. Children born of leprosy parents very rarely develop the disease if removed at once. Leprosy, like tuberculosis, runs in families as a result of contact infection. Children and young adolescents run a special risk. The danger of infection from leprosy persons is, of course, greater when there is a discharge from the lesions of the skin and mucous membranes.

It is sufficient for practical purposes to know that the disease is spread mainly by direct contact with lepers, especially under conditions of personal and domestic uncleanness and overcrowding, particularly where there is close and protracted association between the "clean" and the "unclean."

Prevention.—Despite the great gaps in our knowledge of the disease, leprosy may be prevented and controlled by isolation and hygiene. The disease automatically disappears with social betterment, especially with better housing to avoid crowding, personal and community cleanliness and higher standards of living.

Personal prophylaxis depends upon avoiding the infection and applying the benefits of personal hygiene, as in tuberculosis.

For the control of leprosy the most important administrative measure is to segregate the lepers in settlements or institutions. Segregation also entails proper treatment and humane care. The leprosarium must be both hospital and home. Compulsory notification and facilities for early diagnosis are essential. The leprosaria should be inviting and should contain all modern improvements for the care and treatment of the disease. Leprosy is by no means invariably fatal. In the United States, where there are only a few hundred lepers, the Government has provided for the establishment of a national leprosarium which is located at Carville, Louisiana. To require each state to provide suitable accommodations to segregate its few lepers is economically wasteful.

It is claimed that the decrease in leprosy in Europe during the middle ages was due in large part to the segregation of the lepers in leprosaria,

⁸³ *Bull. Soc. de méd. exotique*, 1914, 7: 557.

which at one time were numbered by thousands. On the other hand, the value of segregation is disputed because most cases were segregated after the disease became well established and many lepers were at large; however, they were not allowed in churches or market places, and were branded as "unclean" by a distinctive dress and were further required to make their presence known by a bell or clapper.

There is difference of opinion concerning the value of segregation. Sir Leonard Rogers believes that it is effective even when imperfectly carried out; Newman, Hutchinson and others hold that the disease disappeared in Europe despite segregation. The effect of segregation varies at different times and in different places. Thus, leprosy is declining in Japan, while it is increasing in other places. In some of the British colonies in South Africa, where only about half of the leper population is segregated, the rate of admission has remained constant for the last ten years. Segregation in the Hawaiian Islands and the Philippines probably so far has had no conspicuous effect upon the prevalence of the disease; the results, however, are the subject of controversy. There are factors in the control of leprosy not yet understood.

There can be little objection in a country such as ours, where leprosy shows slight tendency to spread, to give a clean leper his freedom, except in the endemic foci in the Gulf States and Great Lakes regions. There is no more danger from a leprosy patient with clean personal habits, who exercises care concerning the discharges from the lesions, than there is from an open case of tuberculosis of the glands of the neck. The purely nerve cases, particularly if there are no ulcerations, may properly be given a greater degree of liberty than those with nodular manifestations. The parole system, begun in 1912 in the Hawaiian Islands, is sensible and successful. Those paroled are required to report for examination from time to time.

The national quarantine regulations forbid the landing of an alien leper. The law requires that such person be deported on the same vessel that brought him. A citizen of the United States having leprosy cannot be debarred. Such individuals are admitted and then come under the health laws of the state or port of entry (see page 535).

The methods for the control of leprosy may be summed up as follows:

1. Compulsory notification and early recognition.
2. Facilities for skilled bacteriological diagnosis.
3. Immediate isolation, preferably in a leprosarium.
4. Home isolation should be allowed only when patients have no discharge of lepra bacilli from their skins and nasal mucous membranes, and provided no children or adolescents live in the house.
5. Separation of the sexes and prohibition of marriage with healthy persons, unless sterile or sterilized.
6. Separation of all healthy children of lepers from their parents from birth.
7. An examination every six months up to five years of all house

contacts and close associates of a leper to detect new infections at the earliest moment.

8. Immigrants arriving with leprosy or developing the disease within five years should be repatriated.

Specific Prophylaxis.—There is no specific preventive for leprosy. The disease, however, responds to chaulmoogra oil, which has been used for years, the benefits of which, however, were not demonstrated until chemical extracts of the oil were prepared. Sir Leonard Rogers, in India, has recently reported encouraging results from the subcutaneous and intravenous use of gynecardate of sodium, a derivative of chaulmoogra oil. McDonald and Dean⁸⁴ and others have reported good results from ethyl esters of chaulmoogra oil. The best results are obtained when the treatment is started early. Improvement also can be obtained from good food, fresh air, cleanliness and the general principles applicable to the modern treatment and prevention of tuberculosis.

In appraising the influence of treatment it must be borne in mind that leprosy is prone to prolonged periods of quiescence and that marked improvement often occurs spontaneously, and indeed that this may go on to apparent cure.

COLLATERAL READING

- BERTARELLI, E. *Centralbl. f. Bakteriöl.*, etc. 1 Abt. Ref., 1911, Bd. 49, No. 3, 65 [good review and long list of references].
- HANSEN. *Virchow's Archiv*, 1882, Bd. XC, p. 542 [original description of the bacillus].
- KEDROWSKI, W. J. *Centralbl. f. Bakteriöl.*, etc. 1 Abt. Ref., 1911, No. 50, 143 [Diphtheroid bacillus].
- Leprosy.* A journal containing everything upon the subject. Published since March, 1900.
- MARCHOUX and SOREL. (Rat Leprosy): *Ann. de l'Inst. Pasteur*, 1912, 26: Nos. 9 and 10.
- Public Health Bulletins.* Studies on Leprosy, U. S. Public Health Service, Washington, D. C.
- ROGERS, LEONARD, and MUIR, ERNEST. *Leprosy.* John Wright & Sons Ltd., Bristol (England), 1925.

CANCER

Cancer⁸⁵ is a general term used to designate all malignant tumors, including carcinoma derived from epithelial and sarcoma from connective tissue. Cancer causes upward of 90,000 deaths a year in the United States and has become one of the chief causes of death.

Increase of Cancer.—According to reported death rates, cancer is more common in civilized than among primitive peoples. The figures indicate a relentless increase during the past seventy years. Thus, in Massachusetts the death rate per 100,000 was 24.5 in 1856 and 116.1 in 1920. In England, the

⁸⁴ U. S. Pub. Health Rep., 1920, 35: 1959.

⁸⁵ Jacob Wolff, *Die Lehre von der Krebskrankheit*, Jena. 1909, 1911, 1913, 3 vols.: a monumental compilation with complete bibliography to date.

death rate which in 1860 to 1866 was 49.8 rose to 105.5 by 1913. These are crude rates, but significant, because when corrected for age, diagnosis, and other factors there is still an increase. Older states, such as Vermont, New Hampshire and Massachusetts, have higher rates than younger states, such as Oklahoma and Montana. This apparent difference is due to the higher proportion of people of the cancer age in the older states and also to differences of sex distribution. Thus, in Massachusetts in 1920, 30.8 per cent of the population was over forty years; in Oklahoma, only 21.3 per cent. Montana had the low cancer rate of 50.6 in 1920, which in part is accounted for by the high proportion of males to females (6 to 5), as well as a smaller proportion of the population of cancer age. Massachusetts has more females than males (19.6 to 18.9).

There are indications that cancer has been on the increase for many years. In England and Wales the proportions of the cancer deaths to total deaths were as follows:

<i>Deaths per 1,000</i>		<i>Deaths per 1,000</i>	
<i>Year</i>	<i>Total Deaths</i>	<i>Year</i>	<i>Total Deaths</i>
1837	7.5	1910	71.6
1850	13.5	1920	93.7
1875	20.9	1923	109.4
1900	45.5		

The statistical studies of Hoffman⁸⁶ indicate that cancer is on the increase. Schereschewsky's convincing analysis⁸⁷ shows that this increase is real and not apparent.

Age.—Cancer is a disease of adult life. About 90 per cent of all cancer deaths occur after the age of forty years; 98 per cent after thirty years. It should not be forgotten, however, that cancer, especially sarcoma, is not unknown in the first two decades of life.

Sex.—The disease is much more common in females than males, about 60 per cent of all cancer deaths being in females. This excess is due to the frequency of cancer of the breast and female generative organs. Women suffer less than men from cancer of the buccal cavity (1:1.3) and cancer of the skin (1:1.74). On the other hand, women have higher rates from cancer of the peritoneum, intestines and rectum.

Most cancer deaths occur in women at about the age of fifty-five and in men at about sixty-five. The earlier incidence in women is due to the fact that cancer of the uterus is more frequent in the younger age decade. At the present time, one woman in eight over the age of forty-five dies of cancer, and one man in fourteen.

Geographic Distribution and Race.—Cancer of the skin manifests an especial predilection for the country, where the rates are about twice as high

⁸⁶ *The Mortality from Cancer throughout the World*, Prudential Press, Newark, 1915.

⁸⁷ *U. S. Pub. Health Bull.*, No. 155, June, 1925.

as in the city. This may possibly be due to the irritating effects of light on skin which has lost its local pigment protection. Cancers of other organs have about the same distribution in city and rural districts.

Cancer seems to be more a disease of civilization than of racial stocks. The evidence here, however, is somewhat conflicting. The cancer death-rates are consistently higher in northern and western than in southern and eastern Europe. Some of this discrepancy may be more apparent than real, and due to differences in statistical methods. In the United States the reported death-rates from cancer are higher in whites than in colored, but the rates seem to be advancing in Negroes. In Maryland in 1920, the death-rate from cancer among whites was 99, among colored 70.

So far as climate is concerned, the southern and warmer countries have lower cancer rates than northern regions, both in Europe and the United States.

Economic Conditions.—There are no satisfactory studies correlating cancer with economic conditions, yet one cannot escape the impression that there is some connection between modern conditions of living and increase in cancer. Finally, cancer may be on the increase as a result of a combination of hereditary and environmental causes, and there is reason to believe that the disease will increase until it reaches an equilibrium, unless some effective measure for prevention or cure is discovered.

Diet.—Sir Arbuthnot Lane and others believe that cancer is one of the expressions of complete revolution in the dietary habits of civilization. Advocates of this hypothesis, for which there is no proof, urge a simple, balanced ration of natural foods, resembling the diet of primitive people. Experimental evidence is suggestive that avitaminosis, particularly a deficiency of vitamin A, may be a responsible factor.

Experimental Cancer.—Cancerous growths occur spontaneously in domestic animals and are occasionally found in wild animals. It is comparatively easy to inoculate cancer from one animal to another by putting a small bit of the tumor under the skin. However, there is a sharp species specificity. Thus, certain strains of mice may be very susceptible and other strains quite resistant to a given cancer. The different kinds of cancer remain true to type when transferred in experimental animals. They may be continued indefinitely. Human cancer has not been successfully transplanted into experimental animals.

Because cancer may be inoculated in laboratory animals is not proof that the disease is contagious or infectious in the sense that it is communicable under natural conditions from person to person. The supposed "cancer houses," "villages," or "streets" do not bear the light of critical examination.

Fibiger⁸⁸ has shown that rats may develop a cancerous-like growth in their stomachs as the result of eating roaches infested with a round worm. The cancerous growth is apparently due to the irritating presence of these worms.

⁸⁸ *Berl. klin. Wchnschr.*, 1913, 50: 289.

The **cause** of cancer is not known. The parasite seems to be the cancer cell itself, and the cancer cell comes from a normal preëxisting tissue cell. Some of the factors that influence the normal cell to take on unrestricted multiplication and growth have been studied with success.

According to Burroughs and others, the problem of cancer is a question of growth control. There is a factor in the body that promotes and another that retards growth. When these two influences are balanced, cell multiplication is normal. Tumors will result when there is an increase in the growth excitant or a decrease in growth inhibition, or both, acting locally. This attractive theory looks to the control of cancer by studies along these lines.

Chronic Irritation.—It seems clearly established that certain chronic irritating agencies may induce cancer, at least in susceptible persons. The irritation may be actinic, thermal, chemical, or mechanical, and perhaps bacterial. Thus we have Roentgen ray epitheliomas, Kangri skin cancer of India, the epitheliomas on the arms of paraffin makers, chimney-sweep's cancer of England, and also the brand cancer of cattle. Each of these presents definite and distinct lesions; each has a constant etiology, clinical course, and pathology. To this same group also belongs the buyo cheek cancer of the Philippine Islands caused by chewing buyo, the irritating agent of which appears to be lime.

A good illustration of the effects of irritation is Kangri cancer, which is an anomalous cancer of the skin of the abdomen and thighs, occurring among the natives of Kashmir. It is caused by the so-called Kangri basket, a small earthenware vessel surrounded by basket-work which contains burning charcoal, and which the natives carry in cold weather suspended next to the skin in order to warm themselves. Oft repeated burns of the abdomen and thighs lead to cancerous developments in that part of the body which, as a rule, is not a cancer site.

Paraffin, tar and other substances have been shown to induce cancer in experimental animals, thus confirming clinical observations.

The Virus of Cancer.—An infective agent has long been suspected and various microorganisms have been described from time to time. None, however, has been confirmed. Peyton Rous⁸⁹ in 1911 transmitted a chicken sarcoma by Berkefeld filtrates. Gye⁹⁰ recently extended these studies, and his researches have led him "to look upon cancer—using the term in its widest sense—as a specific disease caused by a virus (or group of viruses). Under experimental conditions the virus alone is ineffective; a second specific factor, obtained from tumor extracts, ruptures the cell defenses and enables the virus to infect. Under natural conditions continued 'irritation' of tissues sets up a state under which infection can occur. The connection between the specific factor of a tumor and an irritant remains to be investigated. Some

⁸⁹ *J. Exper. Med.*, 1911, 13: 397.

⁹⁰ *Lancet*, 1925, 209: 109.

of the relatively unimportant 'irritants' are known, such as coal-tar, paraffin-oils, etc. The virus probably lives and multiplies in the cell and provokes the cell to continued multiplication." Gye's novel contribution is the growth of the non-specific virus in culture media and the fact that there is a tissue extract, a specific factor, which can break down the defenses of the cell. Barnard⁹¹ has studied Gye's virus by special microscopic methods and describes small spheroid bodies which may be seen and photographed. Others have not been able to confirm Gye's interesting observations.

It is possible that various microorganisms may be the cancer "virus," in that they are capable of irritating cells to abnormal proliferation. In this sense, there may be no specific cancer virus, which most authorities consider to be the cancer cell itself.

Heredity.—Animal experiments clearly indicate that heredity is one of the important underlying factors in predisposing to cancerous growths. A susceptibility to cancer is transmitted as a recessive character and gives Mendelian expectancy in experimental animals, but whether this applies to man, although likely, has not been demonstrated. This is discussed in detail on page 588.

Prevention.—We do not boast of preventing a disease that claims over 90,000 victims a year in the United States alone. Nevertheless, many cases are controlled by early radiation or excision. Cancerous tendencies may perhaps be bred out of the human race, and the eugenic teaching in this regard deserves more attention than is given it. The avoidance of chronic irritation and its multiple causes are clearly indicated. Other facts dealing with the prevention are found above.

The prevailing misconception that cancer is a hopeless and incurable disease is not entirely correct. Cancer at first appears to be a local disease, and therefore curable if detected in time and removed. The literature contains instances of spontaneous reversion and even recovery. Sarcoma is sometimes favorably influenced by an attack of erysipelas.

The Commission on Cancer of the Medical Society of the State of Pennsylvania found that 39 per cent of the superficial cancers and 46 per cent of the deep-seated cancers are preceded by a precancerous condition or a chronic irritation. In other words, in almost one-half of the patients that are sent to the surgeon with a fully developed cancer there has been a previous condition which might have been removed and cancer might not have developed. Superficial cancers frequently exist for a year and a half before they come to the surgeon.

Women over forty should not neglect the first indication of trouble in the breast. A vaginal discharge, especially if bloody, should at once receive skilled medical attention.

The ultimate control of cancer must await the fruition of scientific studies now being made by ardent research workers.

⁹¹ *Lancet*, 1925, 209: 117.

FLUKES ⁹²*(Distomatosis or Distomiasis)*

Flukes are responsible for a group of diseases. These infestations, like hookworm, are important because they are particularly injurious in causing loss of efficiency, diminished vitality and lowered resistance. Flukes are exceedingly common in warm countries, especially the Orient and Africa, where, in parts, more than half the population harbor these parasites. They also occasionally are found in the United States and in other favored countries.

The flukes which infest man are divided into four groups: the blood flukes, the lung flukes, the liver flukes and the intestinal flukes. The diseases which they cause are known as distomatosis or distomiasis, and the symptoms vary, depending on the species and number of flukes. Altogether, about twenty different species have been found in man.

The Parasites and Their Life Cycle.—Flukes are trematodes or flat worms. They are generally leaf-like in outline, but vary markedly in size and shape. They are animals of a very low order of development and have a strange and complex life history. All of them are parasitic in the adult stage, when they attach themselves either internally or externally to their hosts by means of suckers, sometimes aided by hooks. Their life cycle is extraordinary, some of them passing through four and even five distinct phases of existence. During some of these phases they are free-living and during others are parasitic successively in two or even three different hosts.

The known types of life histories of flukes are graphically shown in the following table from Leiper:

Host	Transition	Intermediate Host (Snail)			Transition	Host
Egg	Miracidium (or ciliated embryo)	Sporocyst	Daughter cyst	Sporo-	Cercariæ	Adult
		Sporocyst	Rediæ			
		Sporocyst	Rediæ, Daughter			
			Rediæ			
					Encysted in mollusc in crustacean in insect in fish on vegetation Free-swimming	

The eggs of the fluke, containing a ciliated embryo (miracidium) pass out in the feces, urine, or sputum, depending on the fluke and its location. In water, the embryos hatch out and swim about until they reach a suitable mollusc. In all known cases, fresh-water snails act as intermediate hosts for the flukes which infest man and animals. In the snail, the miracidia develop to form the sporocyst. Within the sporocyst groups of cells appear which in some species develop directly into larval flukes or cercariæ, but in others form rediæ within which develop a brood of cercariæ. The cercariæ are free-living forms in the water and may infect man through the skin or by

⁹² Flukes and the diseases they cause are introduced at this point because they are all transmitted by fresh-water snails.

the mouth; or they may pass to blades of grass to be eaten by sheep; or they may enter some other host, as mollusc, insect or fish, and it is by eating such animals that man becomes infected. In the lung fluke, a certain crab, when eaten raw, conveys the infection; in the liver fluke, infection is brought about by eating fish. The life cycles of many species have not been worked out in all their varied and remarkable details.

Blood Flukes.—The most important of the human flukes are found in the large blood-vessels of the abdominal cavity. Blood flukes are exceedingly common in Egypt and in certain areas of the Orient, and are prevalent in the West Indies and the east coast of Africa. The disease is called schistosomiasis, formerly bilharziasis after Bilharz, who in 1851 first associated the parasite with the disease. There are three species of blood flukes, *Schistosoma hæmatobium*, *S. mansoni* and *S. japonicum*. Each has a different geographic distribution:

Except in severe infestations no serious symptoms appear, but when numerous the worms cause much pain and give rise to a variety of abnormal conditions.

The adult worms of all three flukes live in the abdominal veins, especially the portal vein and its branches. The eggs of *S. hæmatobium* are carried to the small vessels of the urinary bladder, and by means of a sharp spine penetrate the wall of the bladder and are passed in the urine. This results in bloody urine and the disease caused by infestation with *S. hæmatobium* is often called "parasitic hæmaturia." The eggs of *S. mansoni* and *S. japonicum* are laid in the small blood-vessels of the intestinal wall, which they penetrate, appearing in the lumen, and are passed in the feces.

The cercariæ are able to penetrate either the mucous membrane or the sound skin, migrating through the body until they reach their destination in the abdominal veins. Infestation almost always occurs as with hookworms through the skin. In schistosomiasis the danger is from contact with water containing the cercariæ. This commonly occurs in bathing, wading or working in water.

The intermediate snail hosts of the blood flukes are as follows: *S. japonicum*, *Oncomelania nosophora* and *Oncomelania hupensis*; *S. hæmatobium*, *Bullinus* spp.; *S. mansoni*, *Planorbis* spp.

Christopher has shown that intravenous injection of tartar emetic has a specific action on both the worms and their eggs. The chemically pure salt is injected intravenously in doses up to 2 grains (for adults), every second day, until a maximum of 20 to 30 grains is given. The treatment is stopped if symptoms of poisoning appear.

The prevention of schistosomiasis is a problem of great magnitude. The measures vary in different regions, owing to differences in the parasite itself, but especially on account of marked differences in the molluscan hosts. Just as in malaria, the problem becomes a local one, and success depends upon a knowledge of the habits and life history of the responsible snail.

Prophylaxis depends upon the prevention of contamination of water by

infected feces and urine. The disease may also be prevented by attacking the weakest link in the life cycle of the worm, namely, its free-swimming infective stage. Fortunately, this is frail and may be destroyed by filtering or impounding the water. The cercariae die in forty-eight hours. The disease may also be controlled by destroying the fresh-water snails, which are obligate hosts, by the use of copper sulphate or lime (see page 432). Some of the snails which act as intermediate hosts for schistosoma soon die when dried; some are amphibious. Hence, an intermittent flow in irrigating canals may be effective. Infected water can be rendered safe by the addition of one part of cresol in 10,000 parts of water.

Faust and Meleney⁹³ recommended the following prophylactic measures for China: (a) the control of feces disposal; (b) the avoidance of wading in canals, ponds, small lakes and irrigation ditches, and the protection of the skin by workers in rice nursery beds; (c) extensive specific treatment of the disease in outpatient clinics by all qualified hospitals in endemic regions. Immediate steps should be taken to start prophylactic measures against this disease. The activities which will contribute most to the progress of prophylaxis are: (1) more accurate determination of the distribution of the disease and of its intermediate hosts; (2) intensive study of, and experimentation in prophylaxis in small areas; (3) demonstrations of specific treatment; (4) educational propaganda throughout all known and suspected endemic regions.

Lung Flukes.—In certain parts of Japan and Formosa it is estimated that as many as 10 per cent of the inhabitants harbor *Paragonimus ringeri* (*Distoma ringeri*, *P. westermanni*). This parasite is also common in China and recently many cases have been reported in the Philippines. It produces chronic cough and expectoration of a rusty-brown sputum and is therefore popularly known as endemic hemoptysis.

These, in common with other flukes, pass part of their life cycle in snails and also in crabs. There are two ways in which man may become infected, namely by eating infected crabs which are not thoroughly cooked, and by drinking water containing cysts passed from infected crabs. Prevention of this fluke must focus attention upon the snail, the crab and drinking water.

Liver Flukes.—*Clonorchis endemicus* (*Opisthorchis sinensis*) and *C. sinensis* are the most important of the human liver flukes.

In some parts of Japan about 60 per cent of the population have these worms in their livers, sometimes in hundreds and even thousands. They are also common in China and the Philippines. The commonest is *C. sinensis*, the Chinese fluke, which is found in all southern Asia from India to Korea.

Liver flukes live chiefly in the gall-bladder and bile-ducts, where they often cause mechanical obstruction on account of their large numbers. Severe infections, such as occur in countries like Japan where raw fish is eaten, cause symptoms of a serious nature, such as enlargement of the liver accompanied by more or less bloody diarrhea, pain and jaundice. The patient becomes

⁹³ *Am. J. Hyg.*, Mono. Ser. No. 3, Mar., 1924.

anemic, emaciated and weak, and is a ready prey for other diseases. There is no specific treatment.

Prevention depends upon our knowledge of the life cycle of the parasite. The most important personal prophylaxis is to avoid the eating of uncooked fish in regions where the disease is endemic. The larvæ are readily killed by heat, but are not destroyed by refrigeration or by exposure to vinegar for five hours. Radical measures would consist in preventing contamination of water in which fish live. The almost universal use of human feces for fertilizer in Oriental countries keeps the infestation alive.

Fasciola hepatica (*Distomum hepaticum*) is of enormous economic importance by reason of destruction of sheep, but has been reported only twenty-eight times in man and in these instances does not seem to have occasioned marked symptoms and may be regarded as an accidental parasite.

Intestinal Flukes.—A number of species of flukes are commonly found in the human intestine, especially in Oriental countries where other human flukes abound. The following have been reported from man: *Watsonius watsoni* (*Amphistomum watsoni*), *Gasirodiscus hominis* (*Amphistomum hominis*), *Heterophyes heterophyes* (*Cotylogonimus herterophyes*), *Heterophyes nocens*, *Echinostoma ilocanum* (*Fascioletta ilocana*), *Metagonimus yokogawai* and *Fasciolopsis buski* (*Distomum crassum*). These parasites may occur in great numbers, producing anemia, emaciation and general debility.

The full life history of few of the intestinal flukes is known. Barlow⁹⁴ has just described the life cycle of the *Fasciolopsis buski*. They do not do enough damage to cause more than slight intestinal irritation. They are susceptible to most of the drugs used for expelling tapeworms and roundworms.

Prevention.—The prevention of infestation with flukes varies with each species, depending upon its life history and the sanitary habits of the people. The disease is attacked in one or more links of the chain; sometimes the snail, sometimes the crab, sometimes the free-living stages in water, and always the prevention of pollution by proper disposal of urine and feces.

Personal prophylaxis rests on the avoidance of infested water and food. The parasites may enter the body by way of the mouth, or they may penetrate through the sound skin. The water used for drinking and bathing purposes should therefore be clean or purified by storage, filtration or disinfection. People should not eat improperly cooked meat of crabs and fish in endemic regions.

Since fresh-water snails in all cases act as intermediate hosts, these molluscs must be destroyed to break the chain in the life cycle of the fluke. Care in the disposal of feces and urine is important for the same reason. The general custom of using human excreta for manure in Oriental countries makes this problem a difficult one.

Chandler⁹⁵ has shown that copper salts have a powerful toxic effect on

⁹⁴ Published by *Am. J. Hyg.*, Baltimore, 1925.

⁹⁵ *J. Agric. Research*, 1920, 20: 193.

snails. Dilutions of copper sulphate in proportions of one part to from 500,000 to 2,000,000 parts of water destroy snails of all species within forty-eight hours. The eggs of the snails are not destroyed by copper salts. Lime has also proved useful.

Suspected water can be rendered safe by storage for a period (*i.e.*, forty-eight hours) longer than the life of the free-swimming cercariæ; by mechanical filtration through suitable filters; by heat; by chemical agencies, *e.g.*, for drinking water the acid sulphate of soda tablets in use for bacteriological sterilization, and for washing and bathing the addition of creolin, cresol and similar coal tar derivatives (1:10,000), which, however, render the water unpotable. The bilharzia cercariæ are very resistant to chlorin, and water successfully sterilized from the bacteriological standpoint with chlorinated lime is not to be considered as free from risk if taken from bilharzia-infested sources (Leiper).

The reduction of snails can also be accomplished by domestic ducks which feed upon them. Non-operculate snails which convey *S. hæmatobium* and *S. mansoni* are rapidly killed by drying. The operculate snails which transmit *S. japonicum* withstand desiccation.

Tartar emetic is useful in the treatment of bilharziasis or infection with the blood fluke *Schistosoma*. There is no specific treatment for the other human flukes.

SECTION II

MENTAL HYGIENE

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Evolution of Institutional Psychiatry.—Up to a very recent period, psychiatry confined its studies and activities to the inmates of institutions for the insane, or, as they were then called, the lunatic asylums. Designations sometimes measure progress; the lunatic asylum became the insane hospital, and that, in its turn, became the hospital for mental diseases. This change in designation runs parallel with an important evolution. The lunatic asylum was a grim Bastille, isolated, with padded cells and strait jackets, with a high death rate from tuberculosis and dysentery, and where the inmates lived a life alienated from normal occupation and efficient medical care. The modern hospital is a real medical institution, easily accessible, with only a remnant of physical restraint, with occupational and physical therapy to make the lives of the patients conform to normal standards, and whose attitude toward the mentally sick as sick human beings is reflected not only in dining rooms, shops, wards and laboratories, but in a creditably low death rate from tuberculosis and dysentery.

Application of Psychiatry to Social Problems.—But far more significant and of greater importance for the future than the increased humaneness and efficiency of treatment given to the inmates of the hospitals for mental diseases is what may be called the penetration of psychiatry into society. Psychopathic hospitals, designed for service in great urban centers, have been established in several places throughout this country. These hospitals receive for temporary care and diagnosis the acute cases of mental disease; they co-operate with the courts in the study of people accused of crime; they study the subjects of social aid brought as problem clients—by the social agencies. Most significant of all, they have outpatient departments where any one may come for advice in his own mental difficulties, or where one may bring some one whose mental problem is of social or individual importance. These outpatient departments, which in Massachusetts, New York, and a few other states are also part of the equipment of the custodial state hospitals, reach yearly an immense number of people who would otherwise be inadequately understood and advised. The social importance of these outpatient depart-

ments is a ground-breaking one, since in the interplay between the psychiatrist and those who consult him there ensues a mutual profit. As the psychiatrist comes in contact with the borderline cases, as he sees the mental case in its beginnings rather than as an end-product of the asylum stage, the knowledge he gains increases his value to his own times and augurs well for the future.

Crime.—Not only through the psychopathic hospitals and the outpatient department has psychiatry penetrated society. With the realization that crime is, in some part, a problem of abnormal mentalities, enlightened communities have engaged psychiatrists to examine, in a routine way, people incarcerated in jails, houses of correction, and state prisons, with a view to understanding the individual delinquent, and to adjusting his care on this basis rather than on the crime committed. Though there has been a great deal of exaggeration in respect to the percentage of mental disease found among delinquents, yet it is true that wherever abnormal mentality is associated with crime, the abnormal mentality should receive first consideration, the individual placed not only where he can do society no harm, but also where he can be understood and treated. For example, Massachusetts is at present conducting a research into the nature of the minor delinquent by a complete social and psychiatric study. And throughout the country the juvenile delinquent is being studied in clinics whose effort is to understand his nature, his environment, and that reaction between the two which constitutes conduct. Wherever these clinics are once established, they become permanent fixtures, a clear proof of their value.

Education.—The penetration of psychiatry into education has been less extensive but is rapidly gaining impetus. The physical examination of the school child has been made into an elaborate and efficient routine, but the mental examination has just begun to filter into the school system. The early recognition of feeble-mindedness saves the time of the teacher and of the normal children, which is a great social advantage, while the gain for the defective child himself comes from intensive special effort applied to his education, with the goal in mind of developing to its utmost whatever capacity he has of fitting him into the social scheme in a useful way, and of preventing his drifting into a career of delinquency. In certain isolated instances the high school and the college are utilizing psychiatric knowledge for the purpose of understanding and properly directing the student who, though not defective, has emotional and other mental difficulties. Since the troubles of the adolescent student are greater than those of the child, it is necessary that this phase of mental study receive a far greater amount of study and attention than it receives at present.

Industry.—In all the phases of social life, with the interaction of human beings as their essential basis, psychiatric situations arise, using that term to imply difficulties which come from morbid mentality of one type or another. So in industry there has been a growing conviction that though economic disorders and conflicts do not by any means necessarily arise from diseased or morbid mentality, a good deal of it does so arise, and that the worker

must be viewed as a human being seeking satisfaction for his emotional and instinctive needs, as well as a "hand" capable of so much production, to be used or exploited for the advantage of the owners and managers of industry. Recognition of this fact is rapidly growing, and though it has only here and there borne tangible fruit in a technic of study and treatment, its indirect results are manifested by a wider interest in the life and welfare of the worker, which is wholesome and promises great benefits.

The Mental Hygiene Movement.—A factor which has profoundly influenced the development of psychiatry into a great branch of social medicine, and which has given expression to its aims, is the mental hygiene movement. Started by Clifford W. Beers as a reaction to his own experience as a patient in a state hospital, the movement has become one of the most significant factors in preventive medicine in America. It has passed beyond popular education, into social education. The mental hygiene program utilizes the woman's organizations, the churches, the labor union meetings, the speakers' table in the great men's organizations, and, in addition to articles in the press, has a high-grade journal of its own. The record of the work of the National Committee for Mental Hygiene is an imposing one, and with the efforts of its state branches, this society has contributed greatly to the cause of mental health. Though the main program has been along the lines of education, state surveys have been carried on to estimate the feeble-minded population, traveling clinics for the study of delinquents have been operated in the big cities, promising young men have been given fellowships in psychiatry, assistance and direction have been given to the training of social workers, and in many ways the National Committee has organized and stimulated work in mental hygiene.

PREVENTION OF MENTAL DISEASES

We come to a study of the means by which the prevention of the mental diseases and the production of normal mental health may be definitely arrived at through a practical program. As a starting point, it may be said that the first need is research, since only a few of the major mental diseases are understood as to etiology and pathology. It is idle to talk of preventing dementia præcox or manic depressive psychosis, to name two of the most common of the psychoses, when we are completely in the dark as to their cause, their pathology, and their cure. At the present time, there is a striking dearth of pathologists at work in the state hospitals, as there is a lack of psychiatric investigations in general. I venture to say that if part of the money which now goes for popular education and what might be called propaganda were used to subsidize able young men and women for a term of years in psychiatric research much more knowledge about cause and prevention would be gained in the next generation than has ever been gained before. Psychiatric research has not been sufficiently emphasized or supported by the states or the private agencies at work in this field.

Syphilis.—We know that somewhere from 10 per cent to 15 per cent of the male admissions to the hospitals for mental diseases of this country is directly due to syphilis in the form of general paresis, and that 2 per cent to 3 per cent of the female cases spring from the same source. Thus, stated as a simple medical fact, we approach a series of social problems of unending complexity. For syphilis is bound up with late marriage, prostitution, urban life, commerce, and the social results of our increased means of transportation in a unique way. That syphilis can be reduced in incidence seems certain from the experience of the military services in the late War, but whether our present-day society with its point of view on morals can be brought to the point of establishing a really effective technic is to be doubted (see page 70).

Alcohol.—Alcohol as a direct cause of certain mental diseases plays a varying rôle in increasing the population of the state hospitals. The figures in the Massachusetts State Hospitals, at any rate, show a very interesting set of trends before and after prohibition:

<i>Total First Admissions Due to Alcoholic Psychoses</i>		
<i>Year</i>		<i>Per Cent</i>
1912		11.33
1913		11.81
1914		10.42
1915		9.5
1916		9.8

At this point the admission rate is complicated by the fact that for three years, 1917, 1918, 1919, the so-called temporary care cases are included in the total, that is, alcoholic psychoses which were sent in for a ten-day period and not necessarily committed afterward. As the alcoholic psychoses are short in comparison with the others, this raises the percentage of alcoholic psychoses at once:

<i>Total First Admissions Due to Alcoholic Psychoses</i>		
<i>Year</i>		<i>Per Cent</i>
1917		12.28
1918		8.07
1919		7.75

Thus, comparing 1916 and 1919, it will be evident that there was a distinct drop, even though the temporary care cases are included in 1919 and not in 1916. That is to say, popular education and the War, prosperity and high wages, were attended by comparative sobriety in this period, with a decreasing rate of the alcoholic psychoses. January 16, 1920, the prohibition amendment went into effect. In 1920, the temporary care cases were removed from

consideration, and the figures from now on deal only with the alcoholic commitments:

*Total First Admissions
Due to Alcoholic Psychoses*

<i>Year</i>	<i>Per Cent</i>
1920	3.34
1921	4.81
1922	6.41
1923	7.82
1924	8.08

Figures for 1925 are not completely certain, but a tentative computation shows 8.06, with the feeling in the Department of Mental Diseases that when the figures are completed it will be somewhat higher.

The analysis of such figures, at first blush, tends to show that popular education, prosperity and the psychological factors of the War were reducing alcoholism, and therefore alcoholic mental diseases, very markedly. Following the prohibition amendment, there was an immediate drop which has been ascribed by many to the fact that those engaged in supplying alcohol to the community had not evolved a technic for evading the law, and also that the first reaction of the community was to obey the law. Then, in the course of time, the bootlegging industry became efficient, alcohol again flowed in, and the community, reacting to the supply, or perhaps increasing its demands, became again subject to alcoholism in a steadily rising proportion. It is very likely that equilibrium will again be established, and that the reaction against the prohibition amendment will reach its height, and that there will again ensue a drop in the alcoholic admissions to the hospitals. Certainly the effects of the prohibition amendment in so far as alcoholic psychoses are concerned have not justified the expectations of its proponents. From the standpoint of mental hygiene, it seems evident that popular education, the pressure of the great industrial organizations for sobriety, and an altered social attitude toward alcohol are, on the whole, fairly efficient ways of dealing with alcoholism. The alternative would be an autocratic exercise of power, relentlessly crushing the alcoholic habit. So long, however, as the judges, juries, district attorneys, and the other officials connected with enforcement of the law, as members of the community, have the prevailing attitude toward alcohol and drink it in their homes, no real enforcement on a popular basis is possible, since these men express in their attitude toward offenders their own secret psychological mechanisms.

The alcoholic psychoses are of relatively short duration, in the main recoverable, and thus, from the standpoint of state hospital mental hygiene, alcohol does not present the serious problem that syphilis does. It is very likely, however, that the use of alcohol is probably a more important social disorganizer than syphilis, in that its relationship to unemployment, poverty and crime is more direct than the rôle of its great rival.

In times past alcohol has been held as the cause of feeble-mindedness and of degenerative mental conditions in the descendants of those who drink. There is no proof whatsoever of this statement. The alcoholic races have no higher incidence of feeble-mindedness, epilepsy, and degenerative states than the non-alcoholic races. Without any doubt alcoholism represents a grave social problem. Its rôle in mental disorders is, on the whole, exaggerated. This by no means alters the fact that one of the great fields for mental hygiene is an effective effort to do away with the excessive use of alcohol in the community.

HEREDITY AND EUGENICS

A program of preventive mental hygiene must take into account the fact that at least certain mental diseases "run in families." We need not subscribe to the extreme position taken by many of the writers that a hereditary factor is the main cause of all the mental diseases, in order to emphasize a rational eugenical attitude. I have elsewhere maintained that, of all the mental diseases, the hereditary factor is of importance only in dementia præcox, manic depressive psychosis, and (to a lesser extent) in the involution psychoses. The hereditary factor in epilepsy is not proven, while in feeble-mindedness it is of great importance in a large, yet not exactly defined, percentage of cases. Cases of these conditions arise without known cause in so-called normal families and frequently in families whose heredity is otherwise indistinguishable from that of the great mass of mankind. It is because we do not know the etiology or pathology of these conditions that we cannot afford to be advocates of an extreme eugenical position and advise wholesale castration or sterilization.

Preventive medicine may, it seems to me, reasonably urge the adoption of eugenical standards which will not go beyond what is medically certain, and which will have at least a remote chance of entering into the thought of our times and, ultimately, into the only legislation which is capable of enforcement in America—that which flows from the urgent will of the people. Thus, the following measures are recommended:

1. Marriage should erect a barrier against syphilitic injury to the unborn and to the mate. The need for thorough examination of both contracting parties to marriage ought to require no argument, and no medical man who has seen wife and child develop syphilitic mental disease as a result of the husband and father's infection can fail to register his approval of a scheme of examination designed to prevent such tragedies. Yet no law involving pre-marriage examination has been effective in the United States, and it will require a long period of education before our public powers will really support any such measure.

2. (a). Sterilization of those feeble-minded who are discharged from a hospital for the feeble-minded back to the community.

- (b). Sterilization of those known feeble-minded in the community who

become socially inadequate or delinquent, especially if sexually immoral. It is obvious that these recommendations overlap somewhat, and it will be a long time before the second recommendation has much chance of reaching enforcement.

3. Sterilization of those insane of the childbearing age, discharged from state, or private hospitals, who are going through a remission of a chronic mental disease, such as dementia præcox, or who are recovering from an attack of manic depressive psychosis. As a corollary to this, it should be a cause for annulment of marriage if a sane person has concealed from his mate the fact of a previous psychosis.

A program of eugenics need not hesitate to stand for such measures of general social hygiene as the care of the pregnant woman, the destruction of the slums, better housing and feeding, etc., for the masses. Modern biology is fast doing away with the Herodian type of eugenics, which believed that the race grew better as the individual fared worse, and which separated the welfare of the germ-plasm from the welfare of the body plasm which housed it. Eugenics, which aims to advance the racial welfare, has no real opposition to eugenics, which aims at social amelioration.

Other causes of mental diseases, such as lead and other toxic substances, infections and toxins, nutritional deficiencies (as in pellagra), trauma, etc., furnish only a small portion of the sum total of mental diseases. Their prevention is considered under the corresponding topics of this book.

MENTAL HYGIENE FOR THE NORMAL

A program of mental hygiene for the normal cannot be laid down with anything like scientific precision, since character formation and personality growth, which are its aims, as well as the prevention of mental disease, are still matters concerning which theologian, lawmaker, educator, and industrial leader lay down dogmas from their own viewpoints, and for their own purposes. Certain fundamental principles of mental development merit exposition, from what may be called the viewpoint of the psychiatrist, as forming the basis for normal mental hygiene.

Influence of Body on Mental Health.—We must start with the assumption that the mental processes which we lump together under the word "mind" are functions of the organism, inseparably linked with its structure, as are the other functions, and inseparably linked up with those other functions. We need not pile instance upon instance to prove this, and indeed, we may assume it as a working hypothesis. We may regard the energy of the mental life as derived from the energy of the organism, and especially are we in the position to claim that the mood, the general feeling of well-being and energy, or the reverse, flows, at least in large part, from the condition of the viscera of the body—heart, lungs, intestinal tract, sex glands, endocrines, etc. Emotion is a great bodily event, working either for, or against, the welfare of the organism, increasing or decreasing the flow of thought and the output of

action. Will, purpose, and motive are altered—though, of course, in no regular manner—in fatigue, disease, indigestion, and other disturbances of the functions of the body. Thus, in any general program making for mental health, the foundation is that ancient adage, “A sound mind in a sound body.”

The Infant.—In the care of the child this is of primary importance. The infant under one year needs practically no other mental hygiene than that contained in regular habits of nursing, the proper diet, care of the bowels, plenty of sleep, freedom from excitement, and the fresh air and sunshine. The calm, placid mother and the quiet, intelligent home constitute factors making for mental health. Particularly is it true that a successful discipline, making for regular habits of eating, sleeping, and bowel evacuation, instituted with firmness and discretion, is all important. No teaching of any kind is essential, except, perhaps, that associated with the intestinal activities. A child that learns to walk or talk a month or so ahead of time because of the insistent pride of a mother anxious to see her child “smarter” than the child of her friend, is not going to be a better walker or talker later on, but may enter into a career of exhaustion leading to nervous manifestations.

The Preschool Child.—The growing child, up to the age of five or six, is to be regarded somewhat as the infant. Teaching has commenced, not in any formal way, but through the insistent pressure of the parents and the family—the teaching of habits, manners, and the like. But the mental life of the young child finds its greatest outlet in spontaneous play, at first undertaken alone and at home, and then outside with the neighboring children. Play for the child is a far different thing than for the adult. For the latter it is an escape from painful routine, from the pressure of convention and inhibition—play is thus an escape from repressing realities. But play for the child, though the prime source of pleasure, is his reality, his occupation, and his education. Through play, as an infant, he becomes acquainted with the world he lives in, “with the wetness of water and the soapiness of soap,” with power, push, pull, tension, earth, grass; with will, obstacle, and achievement. A little later, in the imaginative days of his two-year-old existence, he reconstructs the world according to his heart’s desire—this block is a house, that pebble is a man, and yonder blade of grass a meadow; his mind actively busies itself with reality, and he identifies himself with objects around him in a way that makes for the healthy-mindedness of extroversion. And then, in the next step, his play leads him into social intercourse, into the herd life which, from then on, will be the main arena for his efforts, the chief source of his satisfactions, his codes of conduct, and his ideas.

Social Adaptation.—Here we come face to face with one of the most important phases in the evolution of the personality of the child, and one fraught with much good and evil for his future. Watch two very young children, let us say of three years of age, at play. They play for a short time, and then a quarrel ensues over some claim to precedence or superiority on the part of one or the other. A fight, either physical or verbal, follows, and the children separate crying. Each goes home, let us say, to his mother, tells the story,

and in a very short time the quarrel is forgotten, providing the parents are intelligent enough not to take any part in it. The children come back, play again, quarrel again, then come tears, separation, and so on. In a short time you note that the period of play lasts longer than it did before, the quarrels, still numerous, are more often brought to a close by the children themselves, through some effort on their own part at conciliation, and in a relatively short time friendship arises which often becomes superior to egoism, and is not so easily severed by quarrels. The child has learned in this intercourse the art of getting along with his fellows. He has learned coöperation and competition, self-seeking and fellowship. He has learned how to scheme, how to coöperate, and how to abnegate his own self feelings for the sake of peace. He has intuitively acquired the arts of leadership or the arts of followership, both of which are essential to his life. In a word, he has learned to adapt himself to his neighbor. He has become a mixer, and one who finds that in this social relationship there is give and take. This lesson of social adaptability is learned easily, if early, and it is learned painfully, if later.

The feelings of superiority and inferiority mix and mingle in this relationship, and the child learns how to achieve superiority, and how to reconcile himself to inferiority, the latter being as fundamentally important as the former. Furthermore, in this intercourse, there is equality, such as children do not enjoy with the adults, where they are either indulged or tyrannized over, where codes are given them from above, and their own childish powers of criticism cannot come into play. As they mingle with each other, codes arise adapted to their own mental level, and they attack the problem of life in their own way, building up, of course, much that they must tear down later, but since this is a process that continues through life, it is best to start early. There is a flow of emotion, of ideas, and a conflict and a coöperation of wills, which has an all-important meaning for the future mental health of the child. *There is a morbidness in solitariness: a dangerous introspection arises of which ennui, boredom, and morbid attitudes toward reality are the fruits.* Early social life is as essential for the child as diet and play. It is better for any child to play with another who is dirty and uses poor language than to live alone, sapping his own energy by the lack of stimulation, or plunged into an abnormal, crafty, or precocious maturity by the society of adults.

The School Child.—When a child enters school and undergoes the discipline of formal instruction, we reach one of the critical problems of mental hygiene. There are those who would postpone the time of formal instruction until a much later period than is at present customary. In the large cities, the child enters the kindergarten at four or five, and the first grade at five or six. In the smaller town, he enters the first grade usually no later than six years of age. Whether this is too early or not has to be decided on the basis of the individual child. If going to school interferes with a child's appetite and nutrition, overfatigues him, physically and mentally, disturbs his play and his sleep, then that child should be taken from school. If, on the other hand, the child is eager for knowledge, and can take the discipline

and the burden of daily attendance and the regular habits incidental to school attendance, if he gains in weight, eats well, sleeps well, and plays with zest, then it is obvious that school attendance is a good thing for him. Particularly for the only child or the child quite widely separated in years from his next older brother and sister is school attendance a good thing emotionally, and from the standpoint of social adaptation. Many a child who is a problem to the mother at three or four becomes disciplined and easy to handle as he enters school. Subjected as he is to the pressure put upon him by his schoolmates, and to the contagion of discipline and adaptability imposed by the awe-inspiring teacher, he reaches into a civilization in which obedience and the acceptance of social purpose become paramount.

The school should make an effort to analyze the individual child, and to get some idea of his mental capacity and his special abilities and disabilities. The so-called intelligence tests, while not accepted as measures of innate capacity, are, nevertheless, useful measures for getting an insight into the intellectual make-up of the individual child. A child grading high by the intelligence tests, all other things being equal, can be taught accordingly, and no fear need be entertained of giving him a good program of work. On the other hand, if a child rates low by the intelligence tests, he may be either feeble-minded or deficient in some way or other, or else have some special disability which needs investigation and study. Such a child must not be pushed into competition with the brighter children, but should receive special attention, if it is apparent that his backwardness is a mere transitory phase, or he may need placement in a special class, if it becomes obvious that he is mentally deficient. It is kindness to the mentally deficient child to segregate him, and it makes for efficiency in the care of the other children, since it removes a burden from the energies of the teacher which lessens her capacity for teaching his normal mates.

The irritable, emotionally under-controlled child needs as much attention as the feeble-minded child. The special difficulty which is injuring the child may reach into his home, into some fault of discipline, some abnormal home influence. The teacher in an ideal scheme of things, yet realizable, should be able to confer with the parents on this matter, to take up the home influences and see wherein they may be remedied if they are operating badly.

Discipline.—The matter of discipline of young children has received a good deal of attention in the minds of most parents and teachers in recent years, and there has been quite a divergence of opinion on this matter. The modern swing is away from the idea that physical punishment and the infliction of pain are of value. In fact, it has come to be believed that corporal punishment and all measures which in one way or another inflict pain and instill fear, are cruel. With this opinion in moderate measure everyone must be in sympathy. To punish too frequently and without due regard for the undeveloped character of the child is not only cruel, but futile in that it does not correct faults, but merely brings either a crushed spirit, apprehensive and inhibited, or else it fosters a rebellion which alienates the child from the

home and the parent. It is nevertheless true that one of the cardinal methods of teaching is learning by experience, and that learning by experience is merely another way of saying learning by pain and through fear. The burned child dreads the fire and learns to keep away from it because he has experienced pain, and the state of fear has operated to make him careful. All kinds of moral precepts that cruelty to animals is bad will not operate half so efficiently as the realization that cats have claws, and that they will use them if their tails are pulled. One may preach politeness and consideration to a child without much effect on his character, but once he learns that the little boy next door will slap promptly if his toy is taken away from him, a lesson in self-control and equitable feeling is impressed deeply on his consciousness and his conscience. Green apples look good, but colic is bad, and the child learns through the experience of colic what some children can learn in no other way.

Praise and Blame, Reward and Punishment.—There come times in the history of every young child when corporal punishment becomes necessary and the infliction of punishment and pain are requisite measures to be used with firmness, calmness, and dispatch. The weapons of society in dealing with each of its members, and in bringing about a measure of conformity, are reward and punishment, praise and blame. As in the organism itself, where excitation and inhibition make for perfect nervous control, so in the handling of the child the adequate use of the two opposing sets of weapons, reward and praise on the one hand, pain and punishment on the other, lead the child to a satisfactory attitude toward life.

Praise and reward should not be given indiscriminately. They should be handed out with caution, to reward merit, and they should be earned. On the other hand, blame and punishment should be used perhaps with even greater discretion and more consideration, but they should be used, for there is a place for them. The child who is brought up without blame and punishment enters into a world, when away from home, in which blame and punishment are encountered on every side, and in which they are administered not so much for the punishment of faults but often through the spirit of revenge and hatred. It is better for this child that he should be initiated into their use by those who love him than by those who regard him oversternly and with no real affection.

The Habits of Children.—There is a growing emphasis upon the formation of habit as perhaps the most important question for a practical psychology. Psychologists as far apart as Dewey and Watson lay especial stress upon habit. The latter claims that we are even to seek the explanation for the differences in mentality and personality which we call innate, in differences in habit formation arising from different experiences. We cannot accept any such extreme position, since nothing is surer than the innate variability of the human being in every physical quality and, just as logically, in every mental quality. Accepting this, the emphasis laid upon habit formation is part of the technic of dealing with children. Habits of diet, exercise, study and work,

habitual attitudes towards others and towards oneself, are undoubtedly formed in childhood. In fact, the larger part of what has been emphasized in the previous part of this section may be brought into line with the term "habit formation."

While too little emphasis may be given to this subject, too much emphasis is being given lately. It is "too much emphasis" when it is stated that by the correction of the bad habits of childhood, we can prevent mental disease. Since in the main, we do not know the nature or the origin of most of the mental diseases, it is idle to talk of their prevention by the installation of good habits in childhood. Good habits in childhood need no other recommendation than the fact that they make life more comfortable for the child himself, make him more efficient and direct his energies in useful ways. We may be able to prevent dementia præcox or any other psychosis when we know something about their pathology and their genesis. Meanwhile, habit training needs no false claims. For example, it needs no argument that the child should be weaned away and trained away from fussiness in eating. Part of the training away from fussiness is to insure methods of giving him a good appetite; the training includes habits of exercise, play, and outdoors, and not too much stimulation. In part, he may be weaned away from faulty habits of eating by the example of his parents. The father or mother who picks at his food, who eats with lassitude, and is thrown into a tragic mental state if the chops are overdone, has no right to complain if his son or his daughter picks away at his meal. But a part of the mischief lies, too, on the overinsistence upon articles of food which individual children dislike. It sometimes happens that an individual child will not, and does not, eat spinach, and upon this a great domestic battle rages, renewed at every meal, and raised into a tragic issue by the mother. Many children grow up perfectly well who never eat spinach, and some respect must be paid to the individual idiosyncrasies of the child. He need not be badgered into loss of appetite and a meal-table neurasthenia by a mother who too literally follows the newer pediatric teaching. It would be wise for pediatricians in their dietetic teaching to keep in mind how completely they have revolutionized their own teachings in the past ten years, and how completely they may revolutionize them again in the next ten.

In this connection, it is also well to state that an overemphasis is placed upon table manners by the better-to-do mothers. The most important matter for the child at the table is the eating of food, and whether he breaks his bread into bits and butters each bit, or whether he butters the whole piece at once, is not really of importance. The too frequent repetition of the injunction, "Keep your elbow off the table," may make the table a place of torture rather than a place of pleasure. Children absorb good manners by good example, *in the due course of time*, some a little later, and some a little earlier. Overmuch preaching in this respect and in others to the young child is as inadvisable as it is unnecessary. If a child becomes too mannerly, he tends to become hyperæsthetic; trifles commence to mean too much to him. He sees life from too æsthetic an angle, and loses emotional robustness. A pro-

gram of mental hygiene has for one of its main ends the development of emotional robustness as a necessary preparation for life.

Though the effort is made for social conformity, since, on the whole, most people are better adapted to being conventional, even if that means Babbittry, than to be rebellious and outcasts, yet the individuality of the child should be respected. *All habits have not the same value for different individuals.* The day is past when uncompromising and implicit obedience must be exacted. Living in twentieth-century America, the child is no longer a fit subject for a domestic tyranny. The pressure of the parent must not be so heavy as to produce in the child a feeling of inferiority. The faults of the child should not be continually held up to him, so that if he is vigorous and strong, he becomes estranged from the parents and becomes rebellious, or, if weak and sensitive, he becomes a victim of the inferiority complex. The expanding ego of the child, seeking the respect of others and a high self-estimation, seeks those places where it has freedom to grow. If the home tends to crush that ego, then naturally the child seeks the street, transfers its loyalty elsewhere than to the parents, and when the time comes when it is large and robust enough, it throws off the yoke of the home. The home must be a place where freedom and discipline unite, where the child's energies receive consideration, but not suppression, where it can make noise, play, and be happy.

Endurance and Robustness.—The familiar adage that example is better than precept is as true as when it was first said. To ask of a child endurance and hardihood in a home where neurasthenia reigns is expecting too much. Often enough, we mistake for inheritance what is merely following a bad example. Thus, the parent who has a headache whenever difficulties arise, and who cannot stand the noise of the playing children, forfeits the respect of the child when it grows old enough to appreciate his or her weakness, or else subjects it to the tyranny of the vicious example. In too many homes when the child becomes ill, the emotional distress that the parents feel, and their overanxiety, communicate themselves to the child, and lay the foundation for a future hypochondriasis. A child should be taught fortitude and endurance by example as well as by precept.

Here enters a phase of child development which is important. Up to the time the child is nine or ten, it should be kept from undue fatigue and undue effort. After that age, if it is well, it should be taught endurance. It should be discouraged from quitting at the first signs of fatigue, whether the effort is physical or mental. No man ever became a great runner who stopped when first his breath became difficult and his legs became weary. He kept on running until there ensued the miracle of the second wind, when his body fed itself with energy from deep-lying sources. No man ever became a scholar who read only interesting books, and gave up when first he became sleepy at heavy reading. Persistence and patience are necessary conditions by which intelligence achieves good scholarship. No system of education based upon being merely interesting will ever turn out scholars. Adding the radio and movie to education will fail of bringing about real attainment, unless, at the same

time, the child is taught and expected to work on the non-interesting, and to apply himself without stint wherever necessary. Achievement does not come through the merely interesting, and he only is great who is willing to take pains for achievement. *Since achievement in some form or other is necessary for that maintenance of self-respect which is essential to mental health*, the teaching of endurance becomes a prime requisite in mental hygiene.

Parent and Child.—Much has been said of late years on the struggles of the child against the parent. The father complex and the mother complex and the various incest motives have received a vague popular diffusion from their intensive application in the Freudian scheme of things. My own viewpoint leads me to accept the belief that the *sexual side* of the parental-child relationship is not of any fundamental importance, but that the *social-human side* is of vast importance. The problem in the home is the problem of education, discipline, and the maintenance of loyalty. These things are in part obtained through the mere fact that there is a parent and there is a child, but in part they must be earned and worked for. The home may foster or may crush the individuality of the child; it may bring about mental hygiene or it may destroy mental health. The parent must learn to swing from his status as absolute monarch, which he has when the child is born, through an evolution in which he becomes constitutional king, and then merely duly elected leader, working toward that phase of things when the child will be entirely independent of him. In a word, he must learn to recede his own egoism (see page 80).

Puberty and Adolescence.—Puberty and adolescence bring specific problems. At this stage, the child shoots rapidly up in height, and with the evolution of his body toward the adult form come emotions and passions, to which he has hitherto been, in most cases, a comparative stranger. It is true, as has been pointed out by the Freudians, that sex does not spring up *de novo* with the growth of the beard and the appearance of menstruation. Children are sexual from their earliest days, but this sexuality in the normal instance is a diffuse background, leading in the most instances to no overt acts except, perhaps, the auto-erotic act. It gives color to life, but no great amount of form. At puberty, it tends to express itself in new relationships with the opposite sex, most of which must be governed, and in part frustrated, by society and by a self-imposed inhibition. That at this stage the emotionality of the child often becomes a serious problem, and that the energies of life seem to flag instead of expand with the bodily growth, are facts. At this period of life the major mental diseases tend to first make their appearance. On the whole, dementia præcox and manic depressive insanity rarely, if ever, appear earlier than the adolescent stage, and neurasthenia in childhood, although found early enough in some instances, becomes more serious at the stage we are considering. Delinquencies hitherto sporadic and manifested mainly by non-serious misdemeanors become a real problem as the child becomes freer to roam, more resentful of parental authority, and physically and mentally more capable of maintaining an independent existence. As we

take up these problems of adolescence, we shall be able to deal more effectively with a very complex subject (see page 80).

Sex.—Though undoubtedly overstressed by a good deal of the psychiatric thought of the times, it is undoubted that sexual problems become translated, in many cases, into mental problems. When the curiosity of the child becomes aroused by the fascinating gleams he gets, here and there, through the veil of his ignorance of the sexual life around him, he seeks information from his parents or from his companions. Usually he is misinformed, sometimes through the desire to shield him from the difficulties of sex, at other times to protect him from the immorality which the parent fears enlightenment will bring. The problem of sex enlightenment is a very real one in the minds of those who come in contact with children. On the whole, it may be stated that when sex curiosity becomes definitely manifested in the life of the child, that information should be given in harmony with the development of the child, that it be placed on an absolutely naturalistic basis, so far as this is possible. With puberty and the awakening into consciousness of more important feelings than mere curiosity, enlightenment should be rather thorough, with an emphasis on the dangers from venereal disease and on the growth in character which comes through self-control. The introspective child, to whom the sexual life is usually more difficult than to the extroverted child, should be directed into energetic physical activity and a social life which, including both sexes, will at least swing him out of his morbid curiosity into the more real and more controllable difficulties of frank desire. Masturbation, which often starts at an earlier period than puberty, tends to become acute at this time. Its dangers should not be overstressed, nor should fear be aroused that it produces mental disease and the grim catastrophes which are so vividly painted in the quack literature. While masturbation in excess injures the organism in a direct physical way, the main difficulty with it is the feeling of inferiority it produces. There comes a brooding sense of secret sin, which plunges the individual farther and farther into unreality. Wholesome social activity, wholesome physical activity, and the sustaining, tolerant counsel of a wise parent or a wise adult friend are of great value to those boys and girls who find the sex problems of the adolescent period trying ones (see page 86).

Introversion.—There are two main personality types, the introverted and extroverted personalities. The introverted type, the one whose main interest lies in his own emotions and his own mental and physical processes, and in whom the real world is in himself, may be and often is the very finest human type, contributing ethical and æsthetic values and often highly original. Unfortunately, though the occasional introvert will have great value to society, there are many others who drift into mental disorder or into unhappiness through their lack of interest in the outside world. It is a significant fact that all illness tends to make us introspective, that in moments of weakness and loss of energy, we turn away from our conquest of the outer realities to plunge ourselves into contemplation and occupation with the inner ones. On the whole, the mainstay of normality is objective interest with occasional

plunges into subjective self-analysis. Without attempting to alter in any radical way the nature of any introspective child, the dangers inherent in his type of personality should be recognized, and the effort made to interest him in the outer world of things, in the outer world of people, arousing ambitions of an objective kind and developing skilled objective aptitudes.

Extroversion.—While the extroverted personality is not so apt to run into mental disorder as is the introverted, he is perhaps more apt to have social difficulties. In the extreme development, the extroverted individual develops no insight into the character of others because he has no insight into himself, and our insight into others largely depends upon the capacity for self-understanding since it is by a process of projection and sympathy that we reach into the lives of others. The extroverted boy or girl is apt to be scattered in purpose, dissipate his or her energies in irrelevant ways, and often remains relatively immature and unsuccessful. The problem in the case of the extroverts is the consolidation of purpose, the building up of ideals and aims of a relevant kind, the harnessing of the energies of life so that results will come.

Delinquency.—The delinquency of the adolescent is fundamentally the beginning of all delinquency. Very few young children become legal problems in any serious way. It is when the spurt of growth takes place, when the boy or girl is plunged into adult desires and throws off the authority of his adult relatives, that serious delinquency begins. The study of criminals shows that by far the overwhelming majority who present the main legal problems of society began their career at from fifteen to eighteen years of age. While psychopathic trends of an ineradicable kind account for a small percentage of these cases, by far the majority of those who drift into delinquency are relatively normal persons who have not succeeded in disciplining their desires into social conformity. Often the home is unattractive, frequently through its own deficiencies; sometimes, however, a relatively good home is unable to control the adolescent. In other words, the home environment, while often responsible in that the lack of understanding and of pleasure within its walls makes the street and the gang more desirable, is exonerated in many other cases. The street corner and the street gang are, in the main, the locus and the society in which the average young male delinquent is bred. In its enticing freedom from discipline, its stimulating excitement, and the false emphasis laid upon toughness, the street-corner life becomes a great rival of organized society for the adolescent. Gang habits are formed which are inimical to organized industry and the kind of growth which, however slow, is on the whole sure. I am firmly of the opinion that society is largely to blame for its delinquency, and that each social group gets the kind of criminals it deserves. Boys' clubs, boys' organizations, places where the boy may go whose home is unattractive, and where he may be subjected to the influences of robust, athletic, and interested men; where his energies may expend themselves in directed games, social organizations and athletics; where toughness and idleness are met, not by wishy-washy preaching or by uplifters, but by a

red-blooded boy's life and directed toward social usefulness; such agencies would prevent the major part of juvenile delinquency. I have seen a Big Brother group greatly reduce the juvenile delinquency in a large city. The main weapon of this group is the genuine interest shown in the potential delinquent by a man whom he respects and by the stimulation of purpose and skill by that man. Individual attention rather than organized punishment is the only cure for delinquency, though it is more difficult to obtain and apply. Punishment has its place, but when it is not combined with individual attention and an effort at understanding, it merely results in the transformation of the delinquent into the criminal.

The Adult.—Concerning adult mental hygiene, there is mainly this to be said: the basis for a normal mental life has to be laid in childhood and adolescence rather than at the time when habits become fixed and character is formed. As in childhood and every other period of life, physical habits of health are of utmost importance. The adult's moods, his reaction to life, his capacity to think clearly and act normally, depend in very large measure upon the physical condition of his organism.

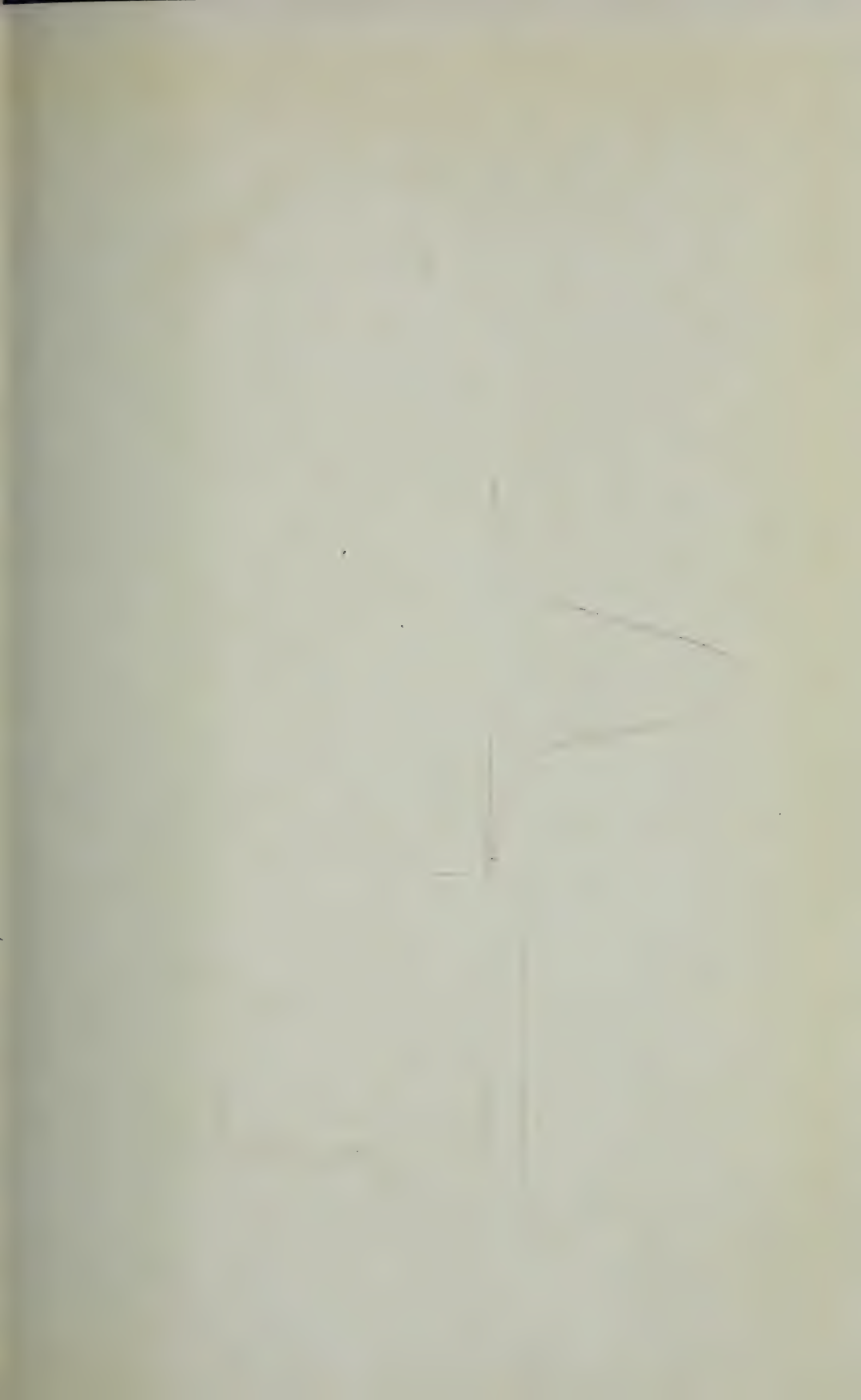
Marriage.—The marital relationship enters into life in the adult period as the source of a great part of the happiness of the individual and as a source of a very large part of his difficulties. The mental hygiene of married life has not as yet received thoroughgoing popular attention. This is because it is in large measure entangled with sex and the taboos of sex. Sexual incompatibility means marital incompatibility, and breeds the dissatisfactions and the disgusts which often lead both to the divorce court and to neurasthenia and hysteria. The sex life is left to chance and to whispered information and misinformation, whereas part of the preparation for marital life should be a thoroughgoing scheme of education given by those competent to give it. Many married women pass through life without real sexual satisfaction and are accused of frigidity. In most cases this frigidity is artificially created by the inconsideration or lack of technic on the part of the husband. Elsewhere I have discussed the neurosis of the housewife, its origin in the evolution of the home and the rising individuality of woman. Here it suffices to say that part of the home difficulties, and the mental results, arise from the economic side, but the larger part are psychological in origin and, in at least a large measure, preventable whenever the man and woman honestly try to understand each other.

Though the sex life of the married adult presents its difficulties, the unmarried are face to face with graver problems. The choice between unchastity and continence plunges the fully developed adult into difficulties of a peculiarly trying kind. The leader who will devise a social-sexual system by *which the social fabric may be preserved* while at the same time the sexual urge finds satisfaction will be numbered among the very wisest of the race. Meanwhile, through absorption in work, through religious faith and in the sublimation of the sexual energies into other channels, many of those balked of satisfaction by the moral laws find their way to a tolerable and even

contented existence. Sex has been overstressed by our civilization, and a step forward will have been made in mental hygiene when sexual differentiation will be less emphasized in clothes, manners, and training, when literature and art deal less with sexual subjects (see Sex Hygiene).

Desire and Satisfaction.—The laws of normal desire and satisfaction will some day constitute the basis of a program of mental hygiene. We know that civilization enormously complicates what we call our needs, so that the luxury of one time is the absolute necessity of another. The rise of æstheticism breeds hyperæstheticism, which really means that those so afflicted find many more things to disgust and dissatisfy than to please them. Refinement brings it about that one finds a subtle pleasure in the very nice adjustment of things, but also brings it about that little disharmonies are keenly felt, and the pin-pricks of life become tragedies. The mounting of desire and taste is a part of civilization, yet it is a part fraught with neurasthenia and an anhedonic reaction to life; that is, a reaction of lost desire and satisfaction. Simplicity of taste, robustness of satisfaction—to maintain these is to maintain happiness and mental health. And the formula for maintaining them is not easy to find and harder to follow in a competitive world where people measure the value of what they have by a comparison with the acquisitions of others. If we could get a real picture of the inner life of men and women of a striving, civilized community, we would find in it an appalling amount of heartache and dissatisfaction, bred by envy and jealousy of others. The struggle for existence, tense as it naturally is in our industrial civilization, is intensified a hundredfold by a struggle for superiority in things which are of no real value. This struggle breeds fatigue, disgust, and depression. Its only cure is a philosophy of life which has been preached at mankind from time immemorial, but which is difficult to follow though all men theoretically accept it. "Thou shalt not covet" is its essence, however amplified it may be.

When a man commences to find that his desires are finicky and his satisfactions vague, when appetite for food, sex, sleep, commence to disappear, when he is as fatigued in the morning as when he went to bed, when the pleasure of work and play become difficult to obtain, it is time for him to take stock of himself and his habits of life. Changes in mood and feeling are as important and need attention as much as shortness of breath and indigestion. The periodic physical examination has proved its value. The periodic mental examination, meaning by that no implication of mental disease, but a study of the mental life and the mental habits, should be established as a great factor making for continued mental health.



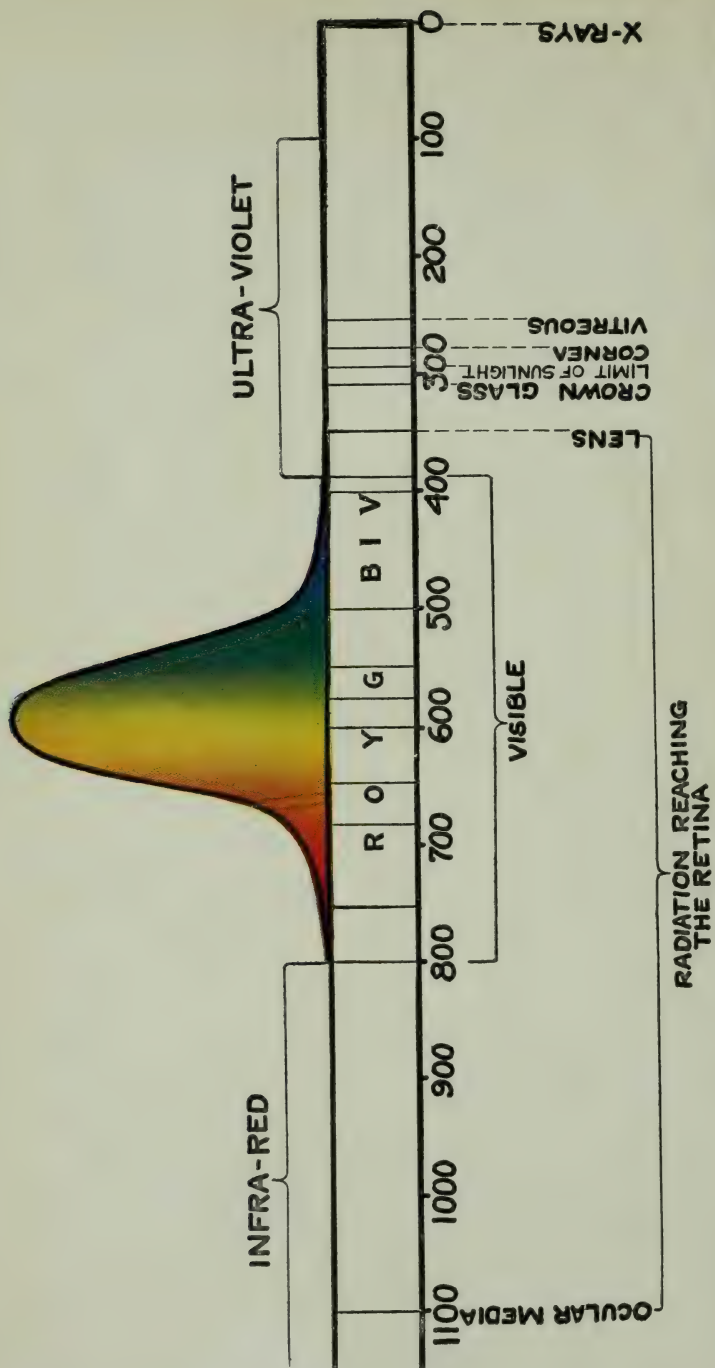


PLATE 1.—RADIANT ENERGY IN RELATION TO THE EYE, SHOWING THE LUMINOSITY CURVE. THE NUMBERS REPRESENT WAVE LENGTHS IN MILLIMICRONS.

SECTION III

CONSERVATION OF VISION AND OCULAR HYGIENE

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FUNDAMENTAL PRINCIPLES INVOLVED

A sound program for the prevention of eye damage and the conservation of vision must be based upon the fundamental principles involved. Therefore, a condensed summary of our present knowledge is given of the physics, physiology and psychology of vision.

Vision involves three factors: (1) light, the external stimulus, a problem in physics; (2) the conversion of the physical energy of light into a nerve impulse, a question in physiology; and (3) the conscious response to this impulse, a puzzle in psychology. Light, the external stimulus, constitutes that portion of radiant energy which falls within the visible spectrum. The conversion of light into a nerve impulse is accomplished at the retina. The conscious response to a visual impulse takes place in the brain. Since vision becomes impossible with the default of any one of these three factors, we must first know the essentials of each.

PHYSICS OF VISION

Light.—Radiant energy of any wave-length must be absorbed before it can be utilized by protoplasm. Protoplasm in general shows two absorption bands in the "electromagnetic spectrum," one in the infra-red region which produces heat, and the other in the ultraviolet region which causes chemical change. Unlike protoplasm in general, retinal protoplasm shows an additional absorption band between infra-red and the ultraviolet, approximately from $800\ \mu\mu^1$ to $400\ \mu\mu$, which induces sensations of light and color.

In terms of the effects produced, one is accustomed to think of the "electromagnetic spectrum" as consisting of heat rays, of visible rays, and of actinic rays. Light includes only the visible rays, which can be sensed through the eye.

Radiant Energy and the Eye.—Nature shields the retina with certain protective devices to prevent the damage which would follow excessive absorption of radiant energy. The air acts as a selective filter for the shorter ultra-

¹ $\mu\mu$ or millimicrons = one-millionth of a millimeter. An angstrom unit is one-tenth of a $\mu\mu$ or one ten-millionth of a millimeter.

violet radiations, and the ocular media act as selective filters for both infra-red and ultraviolet radiations. The atmospheric envelope that surrounds the earth removes at sea level all solar energy under the wave-length of $292\ \mu\mu$. The cornea absorbs all wave-lengths shorter than $295\ \mu\mu$, the lens all those shorter than $350\ \mu\mu$, and the vitreous all those shorter than $270\ \mu\mu$. On the other end of the spectrum the combined ocular media absorb all infra-red rays longer than $1100\ \mu\mu$. Consequently, the retina is exposed only to that band which is included between $350\ \mu\mu$ and $1100\ \mu\mu$, no matter what is the source of radiant energy. Absorption of excessive amounts of energy within this band, as when one views an eclipse, may give rise to damage of the retina through accumulated heat; in fact, all retinal damage caused by excessive exposure to light is believed to be of the nature of a burn.

Radiant energy is not uniformly absorbed by the retina. The maximum absorption in the light-adapted retina takes place with the wave-length of $589\ \mu\mu$, the yellow zone of the spectrum, where the maximum luminosity occurs. Luminosity falls with the decreasing absorption of wave lengths longer and shorter than this, and ceases at $800\ \mu\mu$ and $400\ \mu\mu$; it is not called forth by absorption of that energy beyond the visible spectrum which is transmitted to the retina. See luminosity curve, Plate I.

PHYSIOLOGY OF VISION

The Conversion of Light into a Nerve Impulse.—Few realize what a complicated process vision is. The manner in which light is focused and then converted into a nerve impulse must be considered in two phases: first

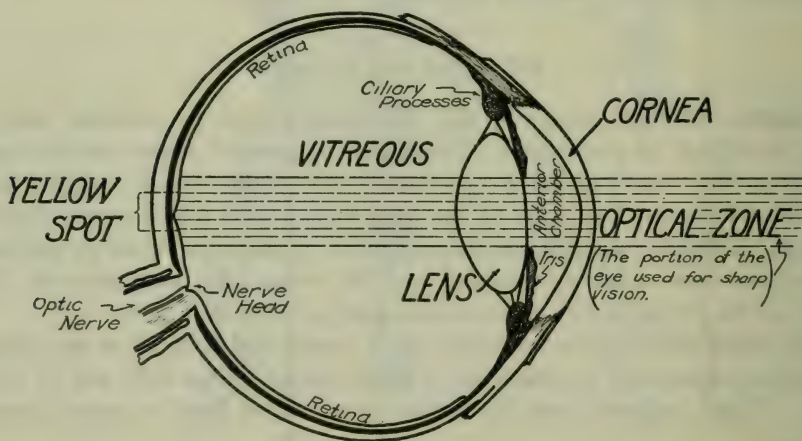


FIG. 45.—NORMAL EYE, SHOWING THE SMALL PORTION USED FOR SHARP VISION.

the mechanism for obtaining sharply defined images upon the retina, and second the photochemical process in the retina wherein physical energy becomes nerve energy.

For sharp images there are three essentials; namely, (1) a dark chamber

(the eyeball) with a diaphragm (iris and pupil), (2) a taut sphericity of the coats of the eyeball, and (3) a mechanism for focusing upon far or near objects. The iris and its continuation in the pigmented layer of the eyeball (uveal tract) and the retina provide pigment for excluding light from the interior, and the reflexly controlled pupil regulates the amount of light entering the eye. The ciliary processes "secrete" the aqueous under a normal pressure equivalent to 10 to 25 millimeters of mercury. This pressure keeps the coats of the eyeball tautly spherical, and at the same time it maintains a flow of the aqueous to nourish the lens and other non-vascular refractive media, thus keeping them transparent. Focusing upon far or near objects, called accommodation, is accomplished at will through changing the shape of the lens by action of the circular ciliary muscle.

The conversion of light into nerve energy is an obscure process which probably takes place in the rods and cones of the retina. With light stimulation, it has been observed in animals that the cones contract, that the retina becomes acid in reaction, that electric currents are generated, and that a photo-chemical change is induced. In darkness there is regenerated in the rods a photo-active substance, visual purple, which again is bleached quickly into a nearly colorless substance by light. It seems reasonable to assume that there must be a similar photo-active substance in the cones, although no such substance has ever been demonstrated there. No one has yet been able to assemble these fragments of knowledge into a complete explanation of the process by which light is transformed into nerve impulses.

PSYCHOLOGY OF VISION

The Conscious Response to a Nerve Impulse.—In the brain, nerve impulses from the retina excite sensations of light, color and form. The mechanism of the conscious response is entirely unknown, and its study is limited to subjective methods; it is a parcel of the realm of psychology.

In connection with the conscious response, it is interesting to note the relationship which exists between the rods and cones of the retina and the visual cortex of the brain. The left half of each retina is in communication with the left occipital cortex, and the right half of each retina with the right occipital cortex, through the semi-crossing of optic fibers at the chiasm, just before the visual fibers enter the brain. Thus each rod and each cone of either retina is connected with a cell group in the visual cortex, and image patterns upon the retina are thus projected upon the visual cortex. For form and color perception, the most sensitive area of the retina is located at its posterior pole in the so-called "macula lutea," the yellow spot where rods are entirely lacking within a 3° circle. Each cone here sends its impulse through a single nerve fiber (axon) toward the brain. For light perception, the most sensitive area of the retina is located about 10° peripheral to the center of the macula. Here rods reach their greatest aggregation, and here impulses from multiple rods pass toward the brain over a single axon. The

von Kries duplex theory assumes that the rods function for light sense, and the cones for form and color sense.

Since the human retina possesses one small spot, the yellow spot, highly sensitive for form and color perception, it follows that for sharp vision the eyeball must always be so turned that the image from the visualized object falls upon this spot. Hence, the important light rays traverse a relatively small thoroughfare in the ocular media, the so-called "optical zone" outlined by an imaginary cylinder passing through the pupil to the yellow spot. Opacities within this zone in the cornea, lens, or vitreous prevent clear definition of images upon the retina. Any disturbance within the yellow spot of the retina, or within its pathways to or its representation in the visual cortex, gives immediate notification to the patient in the form of reduced visual acuity. On the other hand, disturbances in the retina sparing the comparatively small yellow spot, even though they involve nearly the whole retina, do not remove the patient's ability to read with ease the finest print.

While light, form, and color can be sensed with either eye singly, depth can be sensed only when the two eyes are used together to obtain the stereoscopic effect of the third dimension. For example, when viewing a solid object with both eyes, the right eye sees more of the right side of the solid, and the left eye more of the left side of it. These two dissimilar images are fused in the brain into one composite image which possesses three dimensions. In normal people this "fusion is so complete that neither right or left image can be sensed as a separate image with both eyes open, nor can introspection detect in fusing any effort of memory, judgment or reason."

CONSERVATION OF VISION

Practical Importance of Sight.—The mind would remain blank, except for the sensory experience which is built up from impulses which come through the five special senses: sight, hearing, smell, taste, and touch. Sight, hearing and smell are "distance receptors," giving to the individual information of his environment far removed. The optic nerve is the only nerve in the body that is exposed and can actually be seen. Of all sensory nerve fibers from every source, in the human body, one-half are visual, and since these trunk lines convey detailed information about such a far-flung environment, it is easily understood why sight is the most informative and most effective route to the sensorium. The environment is constantly explored by sight before the individual moves into it, from birth until death. The education of the individual is largely accomplished through visual imagery; that is, through the ever-changing retinal pictures of the world about us; school education depends principally upon the printed word, illustrations, pictures, and visual impressions. Indeed, the effectiveness of the spoken language rests largely upon its appeal to the visual memory. One need only observe the handicap of blindness upon the locomotion and the education of a blind individual to

appreciate the importance of sight. A blind person could not possibly survive without aid.

The Problem: Conservation of Vision.—Viewed in this light, conservation of vision takes place second only to the conservation of life itself, and therefore stands out conspicuously as a vital public health problem. The magnitude of the problem cannot be shown because of the inadequacy of statistics. Statistics of blindness exist, but they are confused by inaccuracies of diagnosis and in the degree of blindness indicated. If one takes as a criterion the ability to count fingers held three feet away from the eyes, then there are well over 100,000 blind people in the United States. Of these, 15 per cent are blinded by industrial accident, 12 per cent by glaucoma, and 10 per cent each by atrophy of the optic nerve, high myopia and corneal opacities; about 3 per cent by trachoma and 2 per cent or more by ophthalmia neonatorum, and the remainder to miscellaneous causes.

Conservation of vision must take into account not only blindness itself, but also the much more extensive problem of partial blindness, which includes every degree of visual impairment up to the inability to count fingers held three feet away from the eyes. Statistics of partial blindness are fragmentary and unsatisfactory.

The lessening of blindness from industrial hazards and the checking of the damage due to ophthalmia neonatorum are noteworthy accomplishments brought about by the splendid work of the National Committee for the Prevention of Blindness, many progressive industries, and also certain hospitals and local agencies. It must be admitted, however, that the all too frequent presence of needless visual damage is a serious indictment against the present efforts to conserve vision. For example, half the total blindness and one-half of all visual damage now existing in the United States could have been prevented. Remembering that thirteen out of fourteen blind in the United States are partly or wholly dependent upon others for support (Best), and that blindness in both eyes is rated by the United States Department of Labor as 100 per cent disability and equal therefore to permanent total bodily disability, this problem becomes not one for national pity alone, but for the national pocketbook as well.

Any successful effort to conserve vision must take root in educated public opinion, and from that firm anchorage it must grow out into a virile campaign of accomplishment. For this purpose, the following pages will deal briefly with the major causes of preventable visual damage, and with the methods of control which experience has shown to be valuable. Details which have no bearing upon such control will be omitted.

GLAUCOMA

Glaucoma is a disease characterized by increased pressure within the eyeball, which brings about damage to the optic nerve head, causing gradual or sudden impairment of vision or even complete blindness. The common

form of glaucoma develops insidiously and frequently progresses so far before it is recognized that treatment cannot restore vision although it can check further loss.

Glaucoma is here given first place among serious disorders of vision, because no organized effort is being made for its control. It contributes one-eighth of all total blindness, and unknown amounts of partial blindness and blindness in one eye. It is mainly a disease of later life, causing one-third of all blindness arising after the fortieth year. Under competent medical direction, its presence can be recognized fairly early, and its serious damage can then be averted. If neglected, as it almost always is at present, its blight is stamped permanently upon its victims.

Two varieties are known: primary, when it appears out of a clear sky, and secondary, when it occurs as the sequel of preceding disease, especially inflammation of the iris, or as the consequence of injury.

Common to all types of glaucoma, there is an increased intra-ocular pressure, which kills the delicate optic nerve fibers where they emerge from the eyeball. If the pressure is very high, as in acute glaucoma, this destruction is very rapid. The use of atropin or any other mydriatic drug is exceedingly dangerous in any form of glaucoma, because a dilated pupil crowds the filtration angle, blocks the escape of aqueous, and thus heightens the intra-ocular pressure.

Primary non-congestive glaucoma is the most common type, and it is of very insidious onset. Since it always progresses painlessly, and spares the central vision until very late, the disease usually gives no warning of its presence until the useful sight of one eye is hopelessly lost. It is prone to occur in hyperopic eyes, usually in both eyes but commonly in one eye before the other; and frequently in the Jewish race. Careful questioning may elicit a difficulty in focusing, a frequent unavailing change in near glasses, a misty vision in the morning clearing by noon, a tardy retinal adaptation requiring more than ordinary illumination for reading, or occasional colored halos around bright lights after nightfall. In many cases of established glaucoma, these premonitory symptoms have passed entirely unnoticed. Later in the course of the malady, due to destruction of the peculiarly distributed nerve fibers in the retina, a defect arises on the nasal side of the visual field. On account of the nasal overlapping of the visual fields of the two eyes, the patient is rarely aware of such defect, unless by chance he may close his good eye. In brief, then, to the average victim of non-congestive glaucoma in its early stages, there appears to be no serious disorder because the eye feels normal, looks normal, and sees with the customary distinctness.

Just as the symptoms of early non-congestive glaucoma are difficult for the patient to sense, so are its signs difficult for the physician to establish. Only painstaking examination, and reëxamination will yield the necessary information. An insensitive cornea, a shallow anterior chamber, a vertically dilated sluggish pupil, and an elevation of the light threshold in the dark-adapted eye suggest the diagnosis. Increased tension measured by the tono-

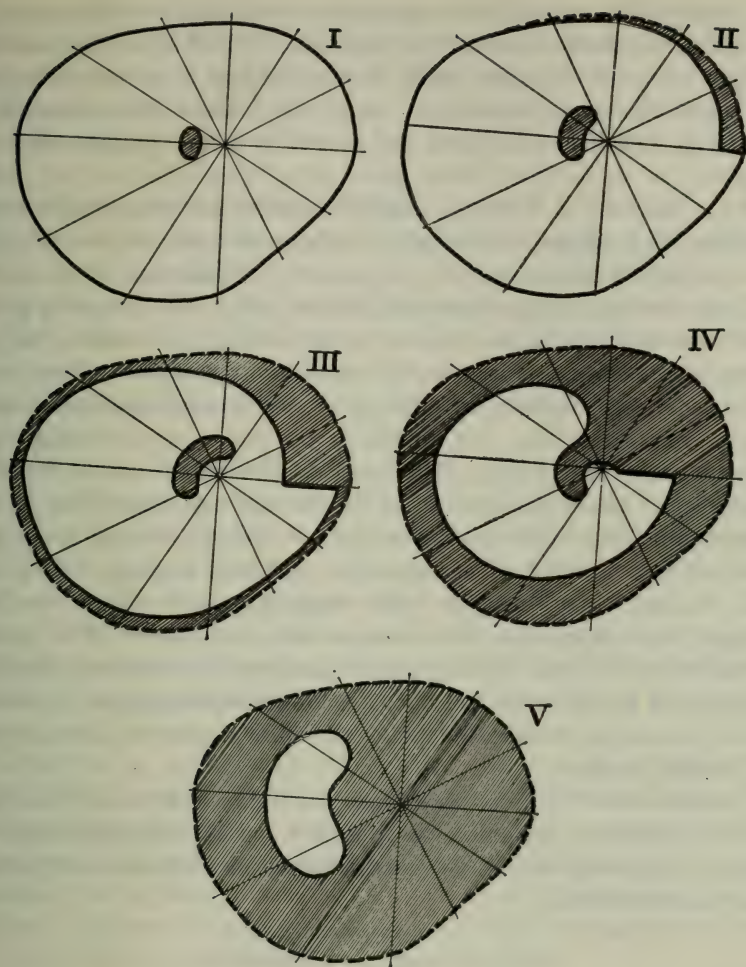


FIG. 46.—THE EVOLUTION OF GLAUCOMA BLINDNESS, AS SHOWN IN THE VISUAL FIELD.

- I. *The Normal Visual Field of the Left Eye.* Note: (1) The fixation point, the projection of the fovea. (2) The normal blind spot, the projection of the optic nerve head. (3) The peripheral limits.
- II. *The Earliest Visual Field Changes in Glaucoma.* Note: (1) Fixation point preserved. (2) Vertical enlargement of the normal blind spot. (3) Peripheral defect nasally, the "nasal step."
- III. *The Visual Field in Early Established Glaucoma.* Note: (1) Fixation point preserved. (2) Concentric enlargement of the normal blind spot. (3) Enlarged "nasal step."
- IV. *The Visual Field in Late Established Glaucoma.* Note: (1) Fixation point preserved. (2) Blind spot joined with the "nasal step," the so-called Bjerrum's sign.
- V. *The last stage, preceding total Glaucoma Blindness.* Note: Central vision completely lost, as is every other portion of the field, except a small area to the temporal side.

meter, characteristic cupping of the optic nerve head, and especially visual field defects typical of glaucoma clinch the diagnosis. Of all signs, the most trustworthy are defects in the visual field, showing first as a vertical enlargement of the normal blind-spot, then as a "nasal step," which later connects with the enlarging blind-spot to form an arcuate scotoma or Bjerrum's sign (Fig. 46).

Once the diagnosis is definitely established, there are two effective methods for checking the progress of the disease, both based upon the control of pressure: (1) by miotic drugs, and (2) by surgery. Pilocarpin nitrate in 1 per cent or 2 per cent strength, or eserine salicylate in $\frac{1}{10}$ per cent to $\frac{1}{2}$ per cent strength, hold the filtration angle open through strongly contracting the pupil, thus bringing about a decrease in the intra-ocular pressure. However, the eye slowly acquires a tolerance for either drug, which must be dropped into the eye several times every day, and eventually the higher strengths fail to hold the pressure within safe limits. The miotic method is the one of choice only in old people, in whom the expectation of life does not exceed the period of efficiency of the drug. The second method is through prompt surgery, the aim of which is to establish a permanent and sufficient drainage for the excess aqueous. Success is more likely to follow surgery applied early in the disease, than surgery done later after tissue degeneration is present. The operation must be suited to the needs of the eye, and may be either the simple iridectomy for glaucoma, or it may be an iridectomy combined with some one of the many devices for promoting drainage such as excision of a piece of sclera, adjacent to the filtration angle.

The whole secret of preventing damage from glaucoma is early recognition and prompt treatment to control the pressure and maintain drainage. The prevention of glaucoma itself is not satisfactory because the cause of the disease is not at all understood.

INDUSTRIAL ACCIDENTS

Two hundred thousand workmen in the United States each year meet with accidents to the eyes, which cause blindness in about 2,000 or 1 per cent of all such injuries. Of the total blindness in the United States, 15 per cent is attributed to industrial accidents. The greater proportion of these accidents nowadays occurs in the smaller shops or on the farm, where safety devices are not in use. They also occur on railroads, in construction work and in field and forest.

Of all accidents, the most hazardous are those from flying bits of metal, stone, or wood, because such bodies, when they penetrate, mutilate the tissues of the eye, and also because there is likelihood of sympathetic disease involving the uninjured eye. The most dangerous occupations, therefore, are chipping metal with a cold chisel, the use of small hand tools which "mushroom," the dressing of stone, the chopping of wood, etc. The United States Steel Corporation reports that 44 per cent of all ocular injuries in its plants accom-

pany the use of hand tools. Particles of steel or brass may carry infection into the eye, or if left in the eye, they may cause inflammation through the irritating action of their oxidation products. Glass is often tolerated in the eye for long periods without irritation. Chips of stone or splinters of wood in the eye are doubly bad, first because not being magnetic they are difficult of removal, and second because rarely being sterile they usually set up a purulent inflammation which may destroy the eye.

Injuries to the eyes from splashing liquids, especially from molten metal or from acids and alkalies, though less frequent, are hazardous to sight. Alkalies are particularly bad because they penetrate and spread, thus causing large and dense scars, which often involve the entire cornea. With any corneal injury, the deeper the destruction, the more opaque the scar upon the cornea. Hence, the hazard of a burn depends upon the depth and the location of the injury.

The abrasive wheel has hosts of victims, but fortunately only a few of these go on to the loss of the eye. The danger here is not from the particle itself, which is often red hot at the time it strikes, and therefore sterile, but rather in the attempted removal of the particle by a fellow workman with the aid of a toothpick, handkerchief, or other equally filthy article. In this way infection is introduced into the corneal tissues, already lacerated, which may lead to perforation of the cornea, or even to inflammation (panophthalmitis) and loss of the entire eyeball. Such foreign bodies should be removed only under aseptic precautions with a sterilized instrument.

Radiant energy generated in industry close to the eye is not filtered through a thick aerial envelope as is the solar energy. Therefore the eye may be exposed to unusually intense infra-red and ultraviolet radiations. Sufficient exposure to the ultraviolet of the electric arc, or to Roentgen rays, may set up a sharp conjunctivitis, which usually subsides without permanent defect. Prolonged exposure to intense heat in glass blowers frequently induces cataract, first in the eye nearest to the furnace.

Indirect damage to the eye is an industrial hazard in certain processes, through the absorption of special poisons which circulate in the blood, act upon the delicate optic nerve fibers, and cause atrophy of the optic nerve. Examples in this class are poisoning from lead or alcohol, especially wood alcohol, which are discussed more in detail under toxic amblyopia.

Prevention.—For the control of this industrial hazard, whether from flying particles, splashing liquids, or injurious radiation, the tendency of yesterday was to place the safety device at the eye in the form of goggles. Due to the unpopularity of goggles even when provided, the tendency of to-day is to place the safety device at the machine. Of equal importance is the education of the employees in the matter of safety. Both safety devices and education have received the greatest impetus from the enforcement of the Workmen's Compensation Acts in the various states. The problems of the small workshop, of the hand tool, and of the implement on the farm are still to be coped with, because there neither education nor safety devices

pertaining to ocular hazards have been adequately applied to these problems.

Adequate light is an important tool in industry and is receiving more and more deserved consideration. Much work done by illuminating engineers shows a definite relation between illumination and industrial efficiency. The aim is to have adequate illumination where needed and illumination without glare or flicker. The direction and quality of the light also deserve consideration. Adequate natural illumination requires a window area equal to one-quarter of the floor area. The intensity of illumination on the work should be from one to twenty foot-candles, the amount depending on the nature of the work, irrespective of whether the light comes from natural or artificial sources. The avoidance of glare is important for the reduction of ocular fatigue, and for the prevention of industrial accidents which may result from ocular fatigue. Insufficient illumination reduces efficiency and also promotes accidents.

The amount of illumination is measured by photometers, of which there are several varieties. Most of those in use depend upon comparing the illumination against a standard.

ACCIDENTS IN CHILDHOOD

A material proportion of blindness is caused by accidents to children at play; sometimes the eyeball is torn by a buttonhook, or pierced by a knife or awl; or a scissors blade, used to untie a knot, slips. Some eyes have been injured by the crack of a whip, by a shot from an air-gun or toy pistol. The toys of children should not include darts, arrows, spears and other warlike hazards. Accidents also occur to the eyes from fireworks, lime burns, curling tongs, a child's finger scratching the parent's cornea, or a doll's china head broken into the eyes of its tumbling "mother." Prevention of such accidents depends upon an intelligent choice of toys and watchful care.

MYOPIA

Myopia, or near-sightedness, is due to elongation of the eyeball. The focus of the myopic eye at rest is upon near objects, whereas the focus of the normal eye at rest is at infinity. It is only in the high degrees of myopia that one usually finds serious dangers to sight which spring from degenerated, overstretched tissue.

The cause of myopia is unknown. It is present in the cultured Caucasian races, and likewise in equal degree in uneducated races such as Bedouins and Egyptians. Practically every child is born somewhat far-sighted (hyperopic), and as the eyeball elongates with age, the hyperopia tends to change toward myopia. As slight a change as one millimeter in the length of the eyeball gives three diopters of change in the refraction. Myopia tends to run in families, and it practically always develops during the growing period, ceasing at about twenty-one years of age. Progressive myopia, however, does not

cease at this time, but continues in later life, often with serious damage to sight. Therefore, from the standpoint of the conservation of vision, the progressive myopes are the only ones that run the risk of partial blindness, because this is the only form of myopia in which serious complications ensue.

Of every hundred pupils leaving the primary schools of England, ten are myopic, and one is progressively myopic, according to Kerr. If this same proportion applies in the United States, then there are over one million progressive myopes here, all of whom are candidates for the sight-damaging complications of high myopia. One-tenth of all blindness in this country is ascribed to myopia. Myopia rarely causes total blindness, but frequently causes serious impairment of vision, usually in both eyes, which cannot be corrected with glasses.

Progressive Myopia.—There are two serious dangers, from which there is no recourse, which threaten the progressively myopic individual after his thirtieth year, occasionally earlier. The more common danger is the degeneration of the choroid and the retina in the macular area, which causes marked reduction in visual acuity without complete blindness. Accompanying these changes, there may be hemorrhages in the retina or in the vitreous, or the formation of slowly developing cataracts. The less common but more serious danger is detachment of the retina from the choroid with almost complete loss of sight. This catastrophe may be brought on by too violent exertion, or by slight trauma to the eye.

The known ways in which progressive myopia may be retarded and its serious complications minimized all find expression in the sight-saving classes. In London in 1908 Bishop Harman started the first sight-saving class, and five years later similar classes were formed in Boston. Nearly every alert city now has its sight-saving class. In brief, the purpose of these classes is to promote both the general health and the eye health of the pupils. With regard to general health, careful attention is paid to proper nutrition, to posture, to sufficient sleep, and to adequate ventilation. While outdoor exercise is encouraged, all violent exercise is rigidly avoided on account of the danger of separation of the retina.

With regard to eye health, the following practice is followed. Everything is done to avoid poor retinal images, and fatigue of the eyes. Of fundamental importance is a careful annual, or even semiannual, refraction with an alteration in the glasses to suit the needs of the constantly changing eyes. Reading is restricted, and what little reading is allowed must be done with daylight illumination equivalent to ten foot-candles on the page. Glare is excluded. The blackboard is matt-black to avoid specular reflection, and the characters written upon it are at least two inches high. Finally, in order to spare the eye, the ear is utilized as much as possible as a portal of education. Education by ear requires small classes and quiet rooms.

Such classes should be presided over by teachers specially trained in this work, and to these classes should be admitted all children who at the age of ten years have developed five or more diopters of myopia. In such children,

the myopia is likely to double or treble by the age of twenty-one. All youths who show a definite tendency toward progressive myopia should be strongly urged to avoid the constant exacting use of the eyes in trades such as watch-making, or the prolonged education necessary for the learned professions. However, the majority of myopes have a mental development well above the average, and they are quite loath if not positively unwilling to forsake the mental life which is so attractive to them (see also page 1290).

VISUAL DEFECTS FROM CORNEAL OPACITIES

Preventable visual defects may arise from opacities which occupy the central area of the otherwise clear cornea. The extent to which vision is cut down depends upon the size and the density of the opacity, and upon its location with reference to the pupil. Normally, the corneal tissues are transparent, and they contain no blood-vessels. With inflammation, there may be a proliferation of new blood-vessels into the clear cornea in the interest of healing, or with ulceration there may be a loss of the corneal substance which can be replaced only by scar tissue. Both new vessels and scar tissue persist, and they are both nearly opaque. The chief conditions which lead to preventable corneal blindness are corneal ulcers of all varieties, burns, interstitial keratitis, and the pannus of trachoma.

Corneal Ulcer.—Corneal ulcers may be grouped into two classes, (1) those which tend to perforate without heroic early treatment, and (2) those which rarely perforate but commonly recur. The chief factors in perforating ulcers are virulent organisms such as the gonococcus, the pneumococcus, or the liquefying diplobacillus of Petit; accessory factors are increasing age, lessened resistance of the patient, and the interference of free secretion and drainage of tears which serve to mechanically cleanse the conjunctival sac. Ulcers of this type are always serious, and even under expert care they practically always scar the cornea, and often go on to perforation. Consequently, every conjunctivitis should be studied bacteriologically, at least by the microscopic examination of a smear preparation for gonococci and pneumococci, and if a virulent organism is found, the patient should be placed at once under the care of a competent ophthalmologist.

The second class of corneal ulcers, which rarely perforate, are phlyctenular keratitis and rosacea keratitis. While these rarely expose the eye to the dire consequences of a perforated cornea, they may nevertheless with each recurrence deposit centrally upon the cornea opacities which interfere with clear vision.

Phlyctenular Keratitis.—Phlyctenular disease occurs in undernourished, anemic children in whom a tuberculous process can frequently be found elsewhere in the body, as in bones, lymph glands or in the nasopharynx. The disease is characterized by a nodule called a phlyctenule, which is red on the conjunctiva but gray on the cornea, and consists of a nodular collection of lymphocytes just beneath the epithelium; it is not a tubercle, and does not contain tubercle bacilli which can be demonstrated morphologically. The

disease is usually chronic, sluggish and recurrent. The phlyctenules are usually multiple, and may disappear completely by absorption in one or two weeks, but they often break down to form corneal ulcers. Secondary infection from the conjunctival secretions may deepen the ulcer, and thus render the scar tissue more opaque. The ulcer rarely perforates unless it is badly neglected.

The prevention of phlyctenular keratitis is part of the prevention of tuberculosis. The prevention of damage to sight from phlyctenular keratitis is also obtained through general hygienic measures. In other words, both the prevention and treatment of this condition must be directed to the whole body and not to the eye alone. Sufficient food and sleep, fresh air and sunshine, combine to minimize the incidence of this affection, and to prevent its recurrences. Work of this kind depends more for its success upon sympathetic home visits by a social worker than upon the physician.

Burns of the Cornea.—The scars left on the cornea from burns depend upon the depth of the destructive process. If the epithelium and Bowman's membrane only are destroyed, the gap is filled in with regenerated transparent epithelium. If the destruction reaches deeper into the stroma, there is no regeneration of transparent stroma, but only of opaque scar tissue. Burns from a curling iron, or from hot fat or scalding water usually look worse at first than they really are, because the destruction is usually superficial. Burns from acids or alkalis always grow to be worse than they first appear, because unless fully neutralized at once, the chemical tends to penetrate more and more deeply and spread in the corneal tissues. Alkali is worse than acid in this respect.

Interstitial Keratitis.—Interstitial keratitis, a deep inflammation of the cornea, is one of the manifestations of congenital syphilis, along with deafness of nerve origin, Hutchinsonian teeth, rhagades, "saddle" nose, thickening of the periosteum of long bones, and synovitis. It rarely follows acquired syphilis. Usually it occurs between the ages of five and fifteen years, and unusually as late as thirty years. The affection is bilateral, but there may be an interval of months or years between the involvements of the two eyes. The process may affect not only the cornea, but frequently involves also the anterior choroid, or the iris or ciliary body.

The corneal inflammation begins with an infiltration of cells, and later with an invasion of new blood-vessels from the anterior ciliary branches. The cornea gives the appearance of ground glass, or of "salmon patches" if vascularization is profuse. Ulceration of the cornea rarely takes place. Finally the cornea begins slowly to clear, first in the periphery, last in the center. The maximum clearing may require as long as twelve to eighteen months. But the new blood-vessels never entirely disappear, and they obscure clear vision depending upon their number.

Since specific treatment with arsphenamin, mercury, etc., does not abort or prevent an attack of interstitial keratitis in the unaffected eye of a congenital syphilitic, in whom there has been a similar process in the other eye, the only

known way to prevent the malady is through the elimination of syphilis from the human race.

OPHTHALMIA NEONATORUM

Ophthalmia neonatorum includes every type of purulent inflammation of the conjunctiva which occurs during the first two weeks of a baby's life. The cause in 75 per cent of such cases is the gonococcus. Ophthalmia neonatorum and gonorrhea, therefore, are usually synonymous. The offending organism may gain access to the conjunctival sac either during birth as the baby's head descends through the infected birth canal of its mother, or after birth through contact with contaminated fingers or fomites.

Ophthalmia neonatorum varies greatly in severity. Usually it is severe and serious, although occasionally it may be very mild with slow onset and spontaneous recovery. In a typical case both the ocular and palpebral conjunctivæ are red and very much swollen; the eyelids and surrounding tissues are infiltrated and there is a thick, creamy, abundant secretion. Blindness from ophthalmia neonatorum does not occur if the inflammation remains limited to the conjunctiva, but only if the process extends to the cornea, causing ulcers which leave centrally placed scars. Sometimes the ulcer perforates, causing infection of the interior and destruction of the eyeball.

One-twelfth of all blindness in the United States in 1924 has been attributed to ophthalmia neonatorum (De Schweinitz). Best, however, states that ophthalmia neonatorum was responsible for only about 2 per cent of the total blindness in the United States. Fortunately, the application of prophylactic measures at birth and of efficient treatment for conjunctivitis in infants has caused a marked reduction of the infection and also of the serious complications of ophthalmia neonatorum generally over the United States during recent years. The presence of corneal complications nowadays may be laid to the failure of the obstetrical attendant to use a prophylactic, or of the physician to institute early adequate treatment; in brief, they signify neglect. A gonococcus of unusual virulence in an undernourished infant may provide an exception to this generalization.

Prevention.—Credé's Method.—In 1881 Credé introduced as a prophylactic the use of a solution of silver nitrate in the baby's conjunctival sac immediately after birth. The good results of Credé's method are sufficiently convincing to justify criminal proceeding upon those who fail to apply it in every case where there is reason to suspect that the mother harbors the gonococcus. The proper application of this method of prophylaxis involves three steps: (1) The baby's eyelids should be cleansed externally of all secretions with the aid of a cotton sponge moistened in boric acid. (2) The eyelids should be separated, and the conjunctival sacs should be thoroughly irrigated with saturated boric acid solution from the soft nozzle of a flexible rubber ear syringe. (3) With eyelids separated, one drop of 1 per cent solution of silver nitrate should be dropped into the conjunctival sac and allowed to come in contact with the conjunctival surfaces for one minute.

It is of the utmost importance not to scratch the surface of the cornea with cotton sponge, finger nail, or syringe nozzle in applying this method. Other substances have been proposed and tried, but no substance is known to be as reliable as silver nitrate, which should be used in all cases, especially where there is any reason for believing that the mother is infected with the gonococcus.

Credé's method does not strike at the root of the evil. It would, of course, be much better to eradicate gonorrhea from men and women than to be compelled to drop silver nitrate into babies' eyes. Wrapped up with the question of ophthalmia neonatorum is the question of midwives, for to prevent blindness we must have intelligent and conscientious obstetrical attendants, especially for the poor and ignorant classes. Midwifery practice needs regulation, supervision, and elevation. Education is one of the bulwarks of prevention in this as well as other preventable infections.

The good results of Credé's method is one of the great triumphs of preventive medicine. One out of every ten babies (10.8 per cent) born in the Leipzig Lying-in Hospital before 1881 developed ophthalmia neonatorum. Credé at once reduced it to one case in a thousand. Since then, even this thousandth case is disappearing. A few years ago the Gardner wing of the Massachusetts Eye and Ear Infirmary was crowded with crying babies with ophthalmia neonatorum. It was a sorry sight. Now many of the beds and facilities are used for other purposes. During the year ending October 1, 1924, only forty-six cases of gonorrheal ophthalmia were treated, which represents most of those occurring in Massachusetts. Institutes for the blind are finding other uses for their space and philanthropy. The change is magical.

Treatment to Prevent Corneal Ulcers.—Every purulent conjunctivitis of the newborn should be treated as a gonorrheal infection until an examination of the stained smear from the secretion eliminates the gonococcus. Any other practice is malpractice in view of the prevalence and the seriousness of the gonococcus in ophthalmia neonatorum. In a word, once purulent inflammation is present, a search for the responsible organism by examination of stained smear is of crucial and urgent importance, equaled only by the inauguration of immediate adequate treatment.

Adequate treatment, consisting of hourly irrigations night and day of the affected conjunctival sac, can be obtained only in an eye hospital, or in the home with a day and a night nurse under the direction of a competent ophthalmologist. The baby should be wrapped in a blanket, and kept in a position so that the affected eye is next to the sheet. This prevents the gravitation of germ-laden pus into the unaffected eye, which should be constantly watched for signs of inflammation. Thorough hourly mechanical cleansing of the affected conjunctival sac is more effective than any other known form of treatment. It is done by separating the eyelids, and by playing a forcible stream of boric acid solution (saturated) from the nozzle of a soft rubber ear syringe into the upper and lower fornices of the conjunctival sac. The greatest care must be taken not to scratch or injure

the surface of the cornea, and this calls for skill and patience on the part of the nurse. A mother or an unskilled attendant will more often than not at some time abrade the cornea of the struggling, crying baby, and thus she will open up the avenues for serious complications. The additional use of one of the stronger antiseptics, such as 4 per cent protargol, 25 per cent argyrol, or 2 per cent mercurochrome, may aid in ridding the sac of its noxious organism. It is of the utmost importance also to maintain the nutrition of the baby at the highest possible level, preferably with the use of its mother's milk, so that it can better resist the infection.

Legislation.—Until that utopian day when gonorrhea itself can be eliminated, the great work of prophylaxis and of early treatment in ophthalmia neonatorum should be continued lest the public shall again lapse into a state of indifference favoring an increase in blindness from this cause. To this end, legislation should be enforced, requiring the prompt reporting of all births, the prompt notification of all cases of ophthalmia neonatorum, and the regulation and supervision of midwifery and medical practice. Maine, in 1891, was the first state to take legal steps to control ophthalmia neonatorum, and subsequently most of the other states have done likewise. In general, the purpose of the legislation is to secure early treatment through compulsory notification. No law is ever fully enforced unless it is supported by the weight of public opinion. To secure this all-important fundamental year after year, requires a persistent campaign of education year after year.

TRACHOMA

Trachoma is a chronic contagious form of follicular conjunctivitis, due to an unknown virus, which causes a specific destructive inflammation characterized by the formation of so-called trachoma granulations. These granulations ultimately form scar tissue. When trachoma affects the vision, it is due to the formation of an abnormal membrane-like vascularization of the cornea, known as pannus. When trachoma causes impairment of vision, it results from poor treatment or from the lack of early treatment. The development of pannus can nearly always be prevented by rubbing a smooth crystal of copper sulphate over the affected conjunctiva daily for prolonged periods.

Trachoma is a public health problem of magnitude in China, Japan, Egypt and Russia; it is prevalent among the American Indians and is also frequently met with in the whites in some of our southern and midwestern states. It is not an exotic disease with us, but is endemic in the poorer sections of all the large cities.

Trachoma is of such a serious nature that all immigrants arriving at our shores have their eyelids everted and conjunctivæ examined for evidence of this infection. An alien immigrant arriving with trachoma is deported, and the steamship is liable to a fine of one hundred dollars for bringing every case of trachoma where it can be shown that the disease might have been recognized at the port of departure.

The disease has been transmitted experimentally in human beings and possibly also in apes. Important factors in communicability are poor hygienic surroundings and lowered resistance from any cause, which accounts for the slow and uncertain manner in which the disease spreads in families. Trachoma may occasionally be confined to one eye. It is believed the infection is rubbed into the eye by common wash cloths and towels, handkerchiefs, fingers and in similar ways.

The filtrability of the virus of trachoma remains undetermined. Experimental evidence permits no more than the suspicion that the virus may be filtrable under some circumstances. In 1907 Prowaczek² described the so-called inclusion or trachoma bodies. The nature of these bodies and their possible etiologic relation to trachoma still remain undetermined. Noguchi and Cohen³ have reported the successful cultivation of these inclusion bodies, but since the cultivated bodies proved non-pathogenic, the question still remains an open one.

Trachoma flourishes best where sanitary conditions are worst; it occurs chiefly among the overcrowded, underfed and overworked. The diagnosis rests upon the history and the appearance of the lesions, especially scar tissue, papillary granulations, trachoma follicles and pannus. The first visible evidence of the disease is the presence of follicles beneath the conjunctiva of the upper and lower retrotarsal folds, and later follicles may be found on the palpebral and on the bulbar conjunctiva. Errors in diagnosis commonly include follicular conjunctivitis of children, and vernal catarrh. Follicular conjunctivitis occurs chiefly on the lower lid, rarely on the upper lid, and never on the bulbar conjunctiva of poorly nourished school children, and it never progresses to scarring of the lid or to pannus formation. Vernal catarrh is of seasonal occurrence, it involves the palpebral or bulbar conjunctiva, and it can be differentiated from trachoma and follicular conjunctivitis by the presence of numerous eosinophile leukocytes in the conjunctival secretion.

Trachoma is a progressive disease unless thoroughly treated. Scarring is a marked feature, and a contracting scar deforms the eyelids, often with in-turned eyelashes which are apt to scratch the cornea and cause ulcers.

The trachoma pannus may be considered as a direct inoculation of the virus into the cornea from the infected lid. At first there is a lymphoid infiltration between the epithelium and Bowman's membrane at the upper margin of the cornea. Into this infiltrate new vessels grow from the superficial vascular loops, and the new vascular membrane sweeps downward over the cornea, even covering the whole cornea. Finally, Bowman's membrane disappears, its place being taken by the thickened vascular pannus. Ulcers are prone to form at the edge of the advancing pannus, and they may leave behind scar tissue to assist in clouding the cornea.

Prevention consists not only in avoiding the infection, but in maintaining superior hygienic and sanitary conditions, for trachoma does not flourish in

² *Deutsche med. Wchnschr.*, 1907, 33: 1285; *Arb. a. d. k. Gsndhtsamtc.*, 1907, 26: 44.

³ *J. Exper. Med.*, 1913, 18: 572.

such soil. When the disease is once established, damage to vision can be prevented by the standard treatment applied in the early stages. This consists in the daily rubbing of a smooth crystal of copper sulphate over the conjunctiva of the everted lids. More drastic remedies are the application of x-ray, radium or carbon dioxid snow to the follicles, or by surgical expression or even excision of the follicle-bearing area. When the disease is neglected and the pannus becomes dense, there is no effective treatment, and the patient in this stage, with otherwise healthy eyes, is doomed to live behind the shadow of his opacity.

SQUINT OR STRABISMUS

It is not generally understood that squint or cross-eye, if not attended to early, will result in practical blindness of the crossed eye. While never a cause of total blindness, ordinary squint if neglected is a common cause of disabled vision in the squinting eye, and of undeveloped binocular single vision. Both of these disasters become permanent through disuse, and both of them can be minimized through early treatment.

Ordinary squint is so noticeable that it is one of the common eye conditions for which relief is sought. It shows a marked hereditary tendency, and practically always appears before the sixth year of life. In all probability there are various causes of squint, and different theories have been advanced to explain the cause of ordinary or non-paralytic squint, no one of which, however, suits all cases.

No more pernicious advice can be given to a parent than "the child will grow out of his squint." Even if the squint may improve as the child grows older, there is nearly always an irremediable loss of the sense of depth, and of the visual power in the squinting eye. Upon the appearance of squint, the child deserves immediate examination and treatment by a competent ophthalmologist. The application of proper glasses early will correct the squint and preserve the sight in about one-third of all cases. In the instances in which glasses fail, the error may be corrected by surgery. If delayed too long, surgery does not as a rule restore binocular single vision or normal acuity in the squinting eye.

OPTIC NERVE ATROPHY

Atrophy of the optic nerve can arise only after an injury, be it toxic, traumatic, or inflammatory. Following the established clinical custom, if atrophy follows inflammation it is termed secondary, while if it has no such outspoken antecedent it is termed primary. Apart from history and observed clinical course, the differentiating points are that primary atrophy presents a gray, sharply outlined nerve head with prominent lamina cribrosa and vessels of normal caliber, while secondary atrophy presents a white, blurred nerve head with newly formed connective tissue obscuring the lamina cribrosa and throttling the vessels.

Primary atrophy of the optic nerve occurs prominently in neurosyphilis,

multiple sclerosis, poisoning by wood alcohol, lead, quinin and atoxyl, and in Leber's hereditary optic atrophy. In tabes dorsalis, along with the other classical symptoms, primary optic atrophy has been reported in from 10 to 50 per cent of case series. The disease is frequently manifested as optic atrophy without ataxia, or the reverse. There is with optic atrophy a slow, painless loss of vision, contracting the visual field from the periphery. Rarely central vision is lost first. It is seldom possible with any known form of treatment to check the progress of the atrophy once it has begun. Hence, the prevention of tabetic optic atrophy must be directed towards the prevention of a syphilitic infection or by an early and thorough course of treatment before it has become entrenched.

The optic nerve consists of sensory fibers which have their cell bodies in the retina and their terminations in the basal ganglia (lateral geniculate body, etc.). Injury to a peripheral neurone causes degeneration in the portion separated from its cell body (Wallerian degeneration). Injury to any portion of the intercentral optic neurone brings about degeneration of the entire neurose.

Wood alcohol, Columbian spirits, methyl alcohol (CH_3OH), methylol, causes blindness through atrophy of the optic nerve. As small a quantity as 20-30 cc. at one dose has caused loss of vision. Blindness may even be caused by inhaling the vapor. Quantities as small as 0.2 per cent in the inspired air may accumulate in the body and cause toxic effects. Wood alcohol is used as an adulterant, especially in liquors and lotions, and also in industry as a solvent. It should be prohibited in any form of material used for internal or external use on the human body. Ample ventilation should be provided in all works where wood alcohol is made or used, as in brewery vats. Containers in which wood alcohol is marketed should have suitable display of labels of warning.

Secondary atrophy of the optic nerve follows intra-ocular inflammations, and papilledema from intracranial tumors. Long-standing papilledema injures the delicate nerve fibers, and ultimately sets up an inflammation in the nerve head, which if neglected terminates in optic atrophy and complete blindness. If the intracranial pressure is released by early cranial decompression, this tragedy is usually avoided. Strangely, vision often remains normal in the early stages of a marked papilledema.

TOXIC AMBLYOPIA

The dimming of vision caused by poisonous substances is called toxic amblyopia. Poisons affecting the visual mechanism have a selective and specific action. Some, like diphtheria toxin, act only on the motor side, and others, like tobacco, wood alcohol, carbon disulphid, cannabis indica, chloral, iodoform, stramonium, thyroid extract, and others, act only on the sensory side. They probably act by poisoning the ganglion cells of the retina, and all of them first attack that part of the retina which is the seat of highest visual acuity—the sensitive *macula lutea*. The lesion at first is not evident

to inspection, but when the atrophy reaches the optic nerve head, pallor is evident. Such persons invariably show in the early stages a central blind-spot (scotoma) for red and green, and later a central scotoma for white, at first relative and then becoming absolute as the injury grows. Individuals vary greatly in susceptibility.

Tobacco and Alcohol.—The use of tobacco becomes largely a habit. Few gain the pleasure and solace from it which they crave and expect; further tobacco is in no way a help to health. Under ordinary circumstances, smoking a few moderately strong cigars a day should suffice for habitués; at least, this amount is not likely to cause trouble with vision.

Tobacco smoke is a very complex mixture. It contains carbon monoxid, hydrocyanic acid, ammonia, nicotin, pyrocin and their derivatives; tar and resinous compounds, etc. Nicotin is carried in the smoke in a suspended state. There is as much as 0.57 per cent nicotin in the smoke of cigarettes, much of which is absorbed. There is a retention as high as 66.7 per cent in puffing, and 88.2 per cent in inhaling.

Diabetics are especially susceptible as are also pellagrous subjects. Neither grain alcohol (C_2H_5OH , ethyl alcohol) alone nor tobacco alone seem to be sufficient to impair sight, but both together in persons over 35 years of age may cause dimness of sight and loss of color sense in central vision. This has been the cause of some marine and railroad accidents.

The *prevention* of toxic amblyopia is manifestly the avoidance of the poison or its immediate disuse upon the approach of symptoms, with measures to promote elimination by bowel, kidney and sweat glands.

DISEASES OF THE UVEAL TRACT

The uveal tract comprises the iris, ciliary body and choroid, which together form the vascular pigmentary layer of the eye. Inflammation of these parts is known as iritis, cyclitis and choroiditis, which are always secondary to foci of infection or altered metabolism elsewhere in the body. The diseases which are especially apt to be complicated with inflammation of the uveal tract are syphilis, tuberculosis, rheumatism, gonorrhea, tonsillitis, abscesses about the roots of the teeth, sinus infections, etc.

Unless the primary focus or the underlying disease is removed, inflammations of the uveal tract are likely to recur, and with each recurrence come disastrous consequences. Recurrent iritis frequently causes sealing off of the pupil, with destructive secondary glaucoma. Cyclitis, too, is serious because it alters the character of the aqueous, promoting secondary glaucoma, complicated cataract and soft, degenerated eyeball. Choroiditis destroys the adjacent retina, and gives rise in this way to blindness in the visual field corresponding in size and location to the damaged retina.

CATARACT

Cataract, which is an opacity of the crystalline lens or its capsule, occurs in many persons with advancing age. This is the common form, and is known as senile cataract, the causes of which are not understood, and therefore prevention is unsatisfactory. Preventable cataracts are those due to diabetes and traumatism; also glass blowers' cataract, due to exposure to heat. Cataracts may complicate disease in the retina or uveal tract, in which the intra-ocular fluids contain toxic substances; also glaucoma, or interocular tumors, etc.

Hereditary cataract occurs in several forms (nuclear, anterior polar and posterior polar), which are often inclined to remain stationary.

OCULAR HYGIENE

Eye-Strain.—Eye-strain is a problem of fatigue. It is not a cause of organic disease, but may aggravate it. It is particularly apt to manifest itself in persons with derangement in the build of the eye or in the proper balance of the eye muscles. Eye-strain is usually found in persons who use their eyes many hours daily in close work, or in those weakened by protracted or severe illness. The symptoms are both local in the eyes themselves, or reflex and general. The most common symptom of eye-strain is headache. Many headaches are due to this cause. The eyes themselves may present nothing abnormal, or they may show conjunctival injection, congestion, styes, etc. Other symptoms are print blurring, restricted distant vision, occasional double vision; in fine, those symptoms which are usually described under the term asthenopia. Reflex symptoms in distant parts of the body, such as digestive disturbances, vertigo, car-sickness, choreic twitchings, faulty positions, spasmodic movements of the head, and a great variety of other manifestations, sometimes seem to be caused or at least magnified, by eye-strain, especially in persons with unstable nervous make-up.

The prevention of eye-strain requires proper correction of refraction and muscle imbalance, care to secure the right kind of illumination, and in some cases by systematic resting of the eyes. Reading and working in dim or flickering light and glare are to be avoided.

Lighting.—Better lighting, especially daylight, improves health and comfort, makes work and play more agreeable, increases production, and diminishes the chances of accidents. There is no light equal to daylight. The general rule is to have at least one square foot of window to every five square feet of floor space. This will vary with the location, size, shape and purpose of the room. In tropical countries buildings have large wall spaces and small window openings, while in high latitudes the converse has been found desirable. The importance and economy of plenty of daylight has caused a fundamental change in the architectural design of buildings through

the invention of the steel sash. Prismatic glass is useful in giving rooms more natural light. Skylights are practical and should be used more than they are. Sharp contrasts must be avoided. A light viewed against the bright sky in daytime is scarcely noticed, whereas the same light against a black background is unbearable. Glare from any source is to be avoided. A well-illuminated room will have both direct and indirect lighting. Direct light gives sharply defined shadows; diffused light gives indefinite and soft shadows, or none at all. The light should be of such a character as to relieve the background of gloom. The light must shine on the object of interest and not in the eyes of the observer. All shades, globes, reflectors, etc., should have this simple object constantly in view. The best direction of light for close work is from the rear and above, and in writing over the left shoulder, in order to avoid shadows in the field. A safe rule is that there should be enough diffused light from illumined walls and ceiling so that all parts of the room can be clearly seen. To this add directed light at the points of interest—the work table, piano, or book page.

Good artificial lighting is not a question of the kind of illumination, but of how the illuminant is used. Oil, gas, and electricity produce satisfactory lighting, but each can be abused. Neither ordinary daylight nor ordinary sources of artificial light contain radiations which are injurious to the healthy eye. Electric lighting is clean and does not vitiate the air, and is therefore hygienically superior to gas or oil.

Proper lighting has a direct bearing on the economic running of a factory,⁴ and also on the efficiency of the men and the safety of the workpeople. The light must be directed onto the work in such a way that the greatest intensity will be where it is wanted. The amount required is from one to twenty foot-candles.⁵ Thus, boiler rooms and power plants should have three foot-candles, but a minimum of one; classrooms should have ten foot-candles on the desk tops, with a minimum of five; drafting rooms should have fifteen foot-candles on the table tops, with a minimum of eight. Under no conditions must the source of the light fall directly in the eye nor should there be any surfaces which will reflect a strong light into the eyes of the workmen. Flickering lights should always be avoided. Good illumination can only be prescribed where the uses to which the light is to be put are known (see also pages 460, 1268 and 1288).

Marked advances have been made in the art of artificial lighting. Illuminating engineers know how to give us better lighting at less cost, thus adding to efficiency, safety and comfort. See the splendid reports of the American Society of Illuminating Engineers.

Care of the Eyes.—Short periods of rest should interrupt all kinds of close work, during which the eyes should be directed on distant objects. This

⁴ *Code of Lighting for Factories, Mills and Other Work Places*, Welfare Work Series, No. 3, Advisory Committee, Commission on Labor, Council National Defense, Jan., 1919.

⁵ A foot-candle is the illumination afforded by a standard candle at a distance of one foot.

relaxes the muscles of accommodation and convergence. Reading in a recumbent posture should be avoided, and should not be persisted in when drowsy or physically tired. Very small type and poor paper, automobiling and moving pictures are fatiguing.

If the eyes feel hot and uncomfortable after exposure to the irritating effects of wind, dust, glare or undue strain, they may be washed with a saturated solution of boric acid. Bathing the closed lids with either very warm or cold water is refreshing and comforting.

The health of the eyes is dependent in large measure upon the health of the body. Reading and close work should be prohibited during fever, and the eyes should be spared during convalescence. After a child has recovered from measles or other acute illness and is able to walk about, the eyes are again ready for normal service. The eyes should be spared during protracted and debilitating disease.

Children should be taught as early as possible never to gaze long into the sun or upon any near object brightly illuminated. It is well to shade the baby's eyes when exposed to sunlight.

Children should be protected from all toys and articles with sharp edges or points which can injure the eyes. Workpeople also require protection against flying particles (page 459). When twilight supervenes, artificial lights should go on, unless the parent wisely utilizes this witching hour with amusement or instruction that rests the eyes.

For the purposes of ocular hygiene the direction, source, power and color of artificial illumination are all important. The amount of illumination should not be judged by the brightness of the lamps, but by the amount of light at the place it is needed. This varies; thus more light is needed for sewing on black cloth than on white cloth. The light should be steady. A flickering light tires the muscles that govern accommodation and leads to fatigue and pain. Reading in railway trains causes similar strain: the eye muscles tire of trying to follow the shaking page. It is contrary to the principles of ocular hygiene to face a glaring light, especially when reading, writing, or in any other work requiring close application. Even though the light may come from above, glaring reflections from polished metal or brass, from brightly varnished surfaces, or even from glossy white paper may be very trying, because a bright light from below falls on the part of the retina which commonly gets light only from grass or dark surfaces. For the same reason glare from snow and sand is not only disagreeable on account of its intensity, but because of the unusual direction from which it comes.

Vision should be tested by competent persons at regular intervals—frequently, during the period of growth and again at the presbyopic age, forty to sixty years.

From the standpoint of the conservation of vision, the important problem in ocular hygiene is the early recognition and proper treatment of glaucoma, phlyctenular keratitis, ophthalmia neonatorum, squint, progressive myopia,

industrial and other accidents, trachoma, and other preventable injuries and infections which impair vision.

The eyes may be a more frequent portal of entry to infections than we have suspected. The conjunctivæ communicate through the lacrimal ducts with the nose, and there is a constant flow of tear secretion in this direction. Microorganisms introduced into the conjunctival sac may be isolated from the nose in five minutes, from the throat in fifteen minutes, and from the stool in twenty-four hours.⁶ Thus, diphtheria, common colds, influenza, pneumonia, tuberculosis, and other respiratory infections and even intestinal diseases may be contracted. Face masks that do not protect the eyes are therefore inadequate.⁷

Normal eyes stand much abuse without causing organic injury. While, however, a disregard of sensible precautions with reference to ocular hygiene may not lead to serious damage, it is often the cause of discomfort and annoyance, and through fatigue may lead to painful local congestion, or even disabling reflex manifestations.

The conservation of vision and the principles of ocular hygiene in its various applications are further discussed in the sections on Light (page 833); School Hygiene (page 1288); Sunlight (pages 836 and 1336).

⁶ *J. Am. M. Ass.*, 1919, 72: 636; A. C. Posey, *Hygiene of the Eye*, J. P. Lippincott Co., 1918.

⁷ Conservation of Vision Pamphlets, I to XX, published by the American Medical Association. A series of popular articles on the care and preservation of good eyesight, prepared by the Committee on Conservation of Vision and issued by the Council on Health and Public Instruction.

SECTION IV

PUBLIC HEALTH MEASURES AND METHODS

SOME GENERAL CONSIDERATIONS

Sources of Infection.—There are two great sources of the communicable diseases of man, viz.: (1) man himself, and (2) the lower animals. Most of the communicable diseases of man, especially those which occur in epidemic form, are peculiar to man. This is the case with typhoid fever, cholera, leprosy, malaria, yellow fever, syphilis, mumps, measles, scarlet fever, typhus fever, infantile paralysis, smallpox, chickenpox, relapsing fever, dengue, and even tuberculosis in large part. It is quite true that some of these infections may be communicated to the lower animals under experimental conditions, but they do not, as a rule, occur in them under natural conditions. In other words, most of the communicable diseases from which man suffers are specific for man; the degree of specificity varying slightly with the different infections.

It is, therefore, plain *that man is the great source and reservoir of most human infections*. Man is man's greatest foe in this regard. The fact that most of the communicable diseases must be fought in the light of an infection spread from man to man is one of the most important advances in preventive medicine. This new thought has crystallized out of a mass of work in the sanitary sciences during the past decade, especially from researches upon tuberculosis, typhoid fever, cerebrospinal meningitis, and other communicable diseases. Formerly, sanitarians regarded the environment as the main source of infection. We now know that water, soil, air, and food may be the vehicles by which the viruses of the communicable diseases are sometimes transferred—that is, they are media of conveyance rather than sources of infection. Most of the microorganisms causing the communicable diseases of man are frail and soon die in our environment, as in the air, soil, or water. Most of them are obligate pathogens and cannot, or do not, grow and multiply under the adverse conditions of our environment.

From the lower animals, particularly the domesticated animals, man contracts a number of infections. Thus we contract rabies and echinococcus cysts from the dog; plague from the rat; glanders from the horse; trichinosis from hogs; anthrax from cattle; Malta fever from goats; foot-and-mouth disease from cattle; tuberculosis, in part, from cattle; flukes through fresh water snails, crabs, and fish; tapeworms and other animal parasites from the meat of fish, fowl, and some mammals. Various skin parasites are also contracted from the lower animals, as ringworm from cats, fleas from dogs, etc. The

number of these diseases¹ and the extent of their ravages are notably less than those contracted from man himself.

The association between man and the domestic animals is intimate, and the contact with rats, mice, and vermin is much closer and more frequent than we suspect. While man contracts several infections from such relations, animals on the other hand contract a few diseases from man, such as trichinosis, *Tænia solium*, *Tænia saginata*, and cowpox.

The fact that most infections are spread rather directly from man to man brings in the forces of sociology to aid those of preventive medicine. The task of preventive medicine is rendered much more difficult from the fact that most infections depend upon the control of man himself. We ruthlessly wage war against insects or against infected food or water. In other words, we can arbitrarily control our environment to a very great extent, but the control of man himself requires the consent of the governed. Thus, it is easier to stamp out yellow fever than to control typhoid fever. It is easier to suppress malaria than syphilis, rabies than influenza, trichinosis than measles. Cattle appear to be mutely thankful when protected by inoculation against blackleg or anthrax, but man rebels against one of the best of all specifics—vaccination against smallpox. The fact that man is the chief source and reservoir of most of his own infections adds greatly to the scope and difficulties of public health work and often makes the prevention of disease dependent upon education and coöperation; and even upon social and economic changes. In this sense preventive medicine is a very important factor in sociology.

Modes of Transference.—The viruses of the communicable diseases may take various routes of transference from man to man or from animal to man. These routes are spoken of as the modes of infection, the modes of transference, or sometimes as the vehicles of infection. Formerly they were spoken of as the "channels of infection," but now we restrict that term to the special channels by which the infection enters the body. Thus, the channel of infection in tuberculosis may be the respiratory tract, the digestive system, or the skin; whereas the mode of transference is through tuberculous sputum, either by direct contact or through the air, as in droplet infection, or through milk or some other vehicle.

The modes of transference may be grouped, for convenience, under three general heads: (1) direct, (2) indirect, or (3) through an intermediate host. In the great majority of cases the virus is transferred more or less directly by what is now known as contact infection. In many instances the virus is transferred indirectly through water, food, soil, air, etc. In a large group of diseases the transfer is through an intermediate host which furnishes the growing list of insect-borne diseases.

The transfer is usually quite direct from one person to the next. The agents of infection, as a rule, do not travel far. The danger diminishes in-

¹ A list of the diseases of animals associated with human diseases is given by Davis in *Science*, 1916, n. s., 44: 339.

versely as "the cube of the distance." However, viruses may be spread broadcast in water and milk; they are also transported great distances in the host along the routes of trade and travel, by cases and carriers.

Contact Infection.—"Contact infection" is a convenient term intended to include a group of circumstances in which infection is spread more or less directly from person to person. Contact infection assumes a quick transfer of fresh infective material. Actual contact between the two individuals is not necessary, but the conveyance is, nevertheless, pretty close in time and space. The contact may be with cases or with carriers. Contact infection alone may be responsible for epidemic outbreaks, even in the case of such diseases as typhoid fever and cholera, but especially in the respiratory infections.

The diseases in which contact infection plays a dominant rôle are those in which the virus leaves the body in the discharges from the mouth and nose, as tuberculosis, diphtheria, scarlet fever, measles, influenza, common colds, cerebrospinal meningitis, whooping-cough, mumps, etc. Contact infection also plays a large rôle in diseases in which the virus leaves the body in the fecal and urinary discharges, as in typhoid, cholera, dysentery, and other intestinal infections.

Contact plays a dominant part in such diseases as syphilis, gonorrhea, skin and other infections having open sores on the surface of the body.

In contact infection the virus may be transferred from man to man directly by actual contact, as in kissing, or more indirectly upon soiled hands, contaminated towels, or infected cups, spoons, toys, remnants of food, and other objects which have recently been mouthed or handled by the infected person. Droplet infection is also included under the convenient term "contact." As a matter of fact, the ways by which the infection may be transferred, and still be considered contact infection, are numerous and varied. In every instance, however, the transfer is brought about by pretty close association with the infected person.

Indirect Infection.—A large group of diseases are conveyed indirectly from person to person through water, food, soil, and fomites. Diseases may be conveyed great distances by means of food or water; they are never conveyed long distances through the air. In the large majority of the diseases contracted by indirect infection the virus is taken into the system through the mouth and discharged from the body in the feces. The best examples of this class are typhoid fever, cholera and dysentery. The relation of soil, food, water, air, and our environment to disease is discussed separately.

The insect-borne diseases form a large and important group, which are fully discussed in Section I, Chapter IV, page 260.

Carriers.—Upon recovery from an infectious process the body usually rids itself completely of the infecting agent. In other words, the immunity which follows an attack of an infectious disease is usually associated with a power the body has of disinfecting itself. In most cases the patient is convalescent or completely restored to health before the cause of the disease has

disappeared from the tissues. This bespeaks a vigorous protecting mechanism, but when this resistance is lowered for any reason a relapse may ensue.

In many instances recovery takes place, but the living virulent microorganisms continue to live in the body. This constitutes "immunity without sterilization," a term introduced by Ehrlich, though a more precise expression would be "immunity without disinfection." Such persons are now known as "carriers." The immunity protects the carrier but endangers his fellow-men.

By the term "carrier" we understand a person who is harboring a pathogenic parasite, but who, nevertheless, shows no signs or symptoms of the disease. Thus a person may have diphtheria bacilli in the nose and throat, but, nevertheless, be in good health. The same is true with the pneumococci, the meningococci, streptococci, and many other microorganisms. Persons may have typhoid bacilli, cholera vibrios, or hookworms in their intestinal tract without showing manifestations of these parasites. Furthermore, persons may have plasmodia in their blood or spleen without having clinical malaria, and so on through a long list of infections.

Persons who harbor pathogenic bacilli without showing symptoms are known as "bacillus carriers," those who harbor protozoa are known as "protozoön carriers," etc. Carriers may be acute or temporary, chronic or permanent, convalescent, passive or active, virulent or avirulent, intermittent, intestinal, oral, urinary, etc.

Convalescent carriers are those that continue to harbor the microorganisms during recovery; this is usually a matter of eight to ten weeks or less.

Passive carriers are those who harbor the microorganisms without having had the disease.

Active carriers are those who continue to harbor the microorganisms after an attack of the disease.

Acute, transitory or temporary carriers harbor the microorganisms for brief periods of time; *chronic or permanent carriers* for months and years. Both passive and active carriers may be either temporary or permanent.

Intermittent carriers shed the microorganisms from time to time.

Carriers further divide themselves into three groups: (1) *intestinal and urinary carriers*, as typhoid, cholera, dysentery, etc.; (2) *oral carriers*, as diphtheria, pneumonia, etc.; (3) *blood and tissue carriers*, as malaria.

The demonstration that many persons are carriers has thrown a new light upon the control of the communicable diseases. With the new facts has come a realization of added difficulties. Carriers can only be detected by painstaking laboratory examinations. When discovered their control is as difficult as it is important. We cannot lightly imprison persons in good health, especially in the case of breadwinners, even though they be a menace to others. In some infections there are so many carriers that it would require military rule to carry out such a plan. Fortunately in most cases absolute quarantine is not necessary. Sanitary isolation is sufficient. Thus the danger from a typhoid carrier may be neutralized if the person exercises scrupulous and

intelligent cleanliness, and is not allowed to handle food intended for others. Such a person might well engage as carpenter, banker, seamstress, etc., without endangering his fellowmen.

Bacillus carriers play an important rôle in spreading infections. They explain many mysterious facts in the epidemiology of diphtheria, typhoid fever, cholera, cerebrospinal fever, malaria, etc. The bacillus carrier is sometimes a danger to himself. This is seen in pneumonia, influenza, streptococcal and other infections.

While it is undoubtedly true that bacillus carriers play a very important rôle in spreading infection from man to man, the relative importance compared with other modes of transmission cannot be stated in percentage. The subject is still too young for definite quantitative figures. There is no doubt that bacillus carriers are more important in some diseases than others and play a variable rôle under different circumstances in the same disease.

Great sanitary reforms, such as the change from polluted to pure water, cause both a decline in the amount of the fever and a decrease in the number of carriers. It now seems evident that polluted water and infected milk will not always cause the disease directly in the persons drinking these fluids, but may produce carriers who either contract the disease themselves subsequently or give it to others by passing the virus on in a more concentrated and virulent form, or to more susceptible individuals.

It is evident from the nature of the case that the cure and control of bacillus carriers is one of the vital problems in preventive medicine. It is not only largely through them that infection is spread, but the infections themselves are kept alive in these carriers, who bridge over the interval between outbreaks. It is quite conceivable that with our modern methods of isolation and disinfection certain diseases could readily be rendered extinct were it not for carriers.

Immunity is, therefore, a double-edged sword, in that it protects the carrier but endangers his neighbor.

The fact that carriers exist in a large number of diseases makes their suppression one of great practical difficulty. The cure of carriers is one of the pressing problems in preventive medicine. One hopeful feature of the carrier situation is that their number may be diminished by isolating and diminishing the cases of the corresponding disease. Thus, the number of typhoid carriers falls off sharply as a result of any successful measure directed only against the clinical cases. In fact, there is a general rule that the number of carriers is directly proportional to the prevalence of the disease. The facts concerning carriers have been discussed separately under each disease in which they occur.

Missed Cases.—By missed cases we understand mild and atypical instances of disease which are not recognized clinically. Almost all diseases vary greatly in severity. Thus we have walking typhoid and ambulant plague. Whooping-cough, scarlet fever, yellow fever, influenza, and most other infections may be so mild that they escape notice. Even the patient himself may not know he is sick. These mild cases go to school, ride in street cars, attend

theaters, continue at their usual work in crowded factories and other places, handle our food, eat at restaurants, and thus spread infection. It is now well known that missed cases are a prolific source of spreading the infection of many of the communicable diseases; they form an important factor in preventive medicine. Many diseases are spread broadcast during the early and even prodromal stages. This is typically the case with measles and smallpox that are especially contagious during the preëruptive period. Whooping-cough is spread before the whoop; typhoid before the patients take to bed; and so on through a long list.

Channels of Infection.—There are numerous channels by which infection may enter the body. These are usually grouped under three headings: (1) the respiratory tract, (2) the digestive tract, and (3) through the skin. Perhaps 90 per cent of all infections are taken into the body through the mouth. They reach the mouth in water, food, fingers, dust, and upon the innumerable objects that are sometimes placed in the mouth. The fact that the great majority of infections enter by way of the mouth gives scientific direction to personal hygiene. Sanitary habits demand that the hands should be washed after defecation and always before eating or handling food; that fingers should be kept away from the mouth and nose, and that objects should not be mouthed unnecessarily. Food and drink should be clean and thoroughly cooked. These simple precautions alone would prevent many a case of infection.

Viruses taken in by the mouth and nose do not necessarily cause respiratory infections. Thus, the viruses of cerebrospinal fever and infantile paralysis enter the system by the mouth; so also with typhoid fever, dysentery, cholera and other intestinal infections.

“Contagious” and “Infectious.”—These are popular terms which lack scientific precision. The words have been used in very diverse senses.

A *contagious* (*contingere*, to touch) disease is one that is readily communicable—in common parlance, “catching.” A contagious disease implies direct or personal contact. If contagious diseases are limited to those contracted by direct contact or touch, as the etymology of the word signifies, only syphilis and diseases similarly contracted would be contagious. As a matter of fact, smallpox, measles, and influenza are types of contagious diseases, as the term is now usually understood.

An *infectious* (*inficere*, to put in, dip in, or mix in) disease is usually considered as one not conveyed directly and obviously, as in the case of contagion, but indirectly through some hidden influence or medium. In the days when specific febrile diseases were regarded as caused by miasmata and noxious effluvia, the terms “infectious” and “miasmatic” diseases were more or less synonymous. Typhoid fever was often taken as a type of an infectious disease. Malaria was the type of a miasmatic disease.

These distinctions are entirely artificial, and serve no useful purpose. Most of the communicable diseases may be transmitted from the sick to the sound in several ways. Infectious diseases may be contagious, and contagious

diseases are infectious. Dividing diseases into those which are contagious and those which are infectious entirely leaves out of consideration the important class of insect-borne diseases. The terms contagious and infectious have always lacked scientific precision and have been the source of some confusion. The word "communicable" is a much better term and should be given preference.

A *communicable* disease is one caused by a specific virus transferred in a great variety of ways. The term "communicable" ignores the mode of transference. There is a great difference in the degree of communicability; some diseases are readily communicable, others are transmitted with difficulty. The evidences of communicability are not so obvious in chronic infections, such as tuberculosis, or in diseases with a long period of incubation, such as typhoid fever. The relationship between one case and the next is often far removed in time and space. If tuberculosis were an acute infection with a short period of incubation like diphtheria it would be popularly regarded as being just as contagious as that disease.

Epidemic, Endemic, Pandemic, and Prosodemic.—A disease is said to be *epidemic* (*epi*, on, upon, and *demos*, people) when it is common to or affecting a large number of persons in a community in a short time. A disease which spreads rapidly and attacks many people at the same time is usually said to be epidemic. An epidemic involves the conception of time, place and numbers.

A disease is said to be *endemic*, (*en*, in, *demos*, people) when it is peculiar to a district or particular locality, or limited to a class of persons. An endemic disease is one which is constantly present to a greater or less degree in **any** place. An endemic disease smolders, whereas an epidemic bursts into flames. A *sporadic* (occurring singly) disease is one in which a few scattering cases occur now and then.

Endemic diseases are apt to flare up and become epidemic. Insect-borne diseases are the best examples of endemicity, as their prevalence is strictly limited by the geographic distribution of the intermediate host. Yellow fever has long been endemic in Havana, cholera in India, typhoid fever in Washington, and plague in Tibet. A great principle of prevention is to attack disease in its endemic home, if such nests exist. This has been eminently successful in the case of typhoid fever which has been controlled by cleaning up its endemic foci.

These terms not only lack precision, but are variously conceived and differently defined. Thus typhoid fever was said to be endemic in Boston, but a similar number of cases in Berlin would have been regarded as an epidemic. For the purposes of maritime quarantine a disease is considered epidemic if there is more than one focus of infection; that is, if several cases occur which have no apparent connection with each other. Strictly, therefore, according to this definition, two cases may constitute an official epidemic and the port would, therefore, be regarded as infected. Every case of communicable disease is a possible source of an epidemic.

It is not feasible to state just how many cases of a disease constitute an epidemic. Ordinarily a few cases of a communicable disease in a village or small town is not regarded as an epidemic; however, five cases of typhoid fever in Podunk (population 1,000) is the equivalent of 5,000 cases in a city of 1,000,000. By the same token, two cases in a small village would proportionately constitute an epidemic of unknown magnitude in a metropolis.

"Pandemic" (*pan*, all, *demos*, people) is a term used to describe a disease which is more or less epidemic everywhere. Pandemics affect a large number of people in a large number of countries at the same time. Thus there have been four great pandemics of plague, when it spread to the four quarters of the globe. In 1889-90 and again in 1918-19 influenza was pandemic. It is not usual, although quite proper, to regard tuberculosis, syphilis and typhoid fever as pandemic.

Sedgwick proposed the term *"prosodemic"* (*proso*, through, *demos*, people) to take the place of the word "endemic." Prosodemic suggests the creeping or smoldering of a disease which is being communicated from person to person through the community by various means, but especially by contact.

Fomites (from *fomes*, touch-wood or tinder) is defined as any substance capable of absorbing, retaining, or transporting infectious germs. Fomites usually refers to inanimate things, such as bedding, clothing, etc. The term was especially used in connection with yellow fever, in which the greatest variety of objects, such as a lock of hair, the false bottom of a trunk, coffee sacks, a mattress, and letters were said to be the fomites which touched off an epidemic. Woolen clothing or the doctor's beard are popular and supposedly dangerous examples of fomites.

The importance of inanimate objects as vectors of pathogenic micro-organisms is assuming a minor rôle in the minds of most sanitarians. Thus we no longer think of such objects as books, umbrellas, floors, walls, curtains, and furniture as likely to transmit the virus of disease. We know that most of the pathogenic bacteria soon die when exposed to dryness and other adverse conditions of environment. We now concentrate our efforts more upon handkerchiefs, towels, bed and body linen, drinking cups, remnants of food, toys, pencils, tableware, and other objects that have recently been mouthed by the infected individual. Such fomites may readily transfer fresh, live and virulent virus from one person to the next.

INFECTION AND IMMUNITY

Immunity.—Immunity or resistance to disease is the very foundation of preventive medicine. It is the overshadowing factor in hygiene. In this sense we use the term "hygiene" to include the care of the person, in contradistinction to "sanitation," which deals with the environment. There is no sharp line of demarcation—we speak of hygiene of the teeth, of sleep, of bathing, of exercise, of food and drink, and of those conditions which are more or less intimately associated with the body. We speak of the sanitation

of the home, of schools, of cities, of farms. Sanitary science considers the air, soil, climate, and our surroundings as they affect health. Sanitation, then, is largely impersonal; hygiene is personal, and, as far as the prevention of disease is concerned, one of the most important factors in hygiene is immunity.

The word "immunity" is a very old term—we still speak of immunity to crime,² but it is only of late years that we are beginning to understand the mechanism by which the body protects itself against infection. The advances have been so rapid that these studies are now dealt with in a separate science known as Immunology.

Immunity is a *function of all living beings* (animals and plants), and in its widest form is one of the fundamental properties of life. Thus, as long as we are alive the colon bacillus in our intestinal tract and the spores of the hay bacillus on our skin do us no harm, but the moment we die, and oftentimes shortly before death,³ these and other bacteria invade our tissues and disintegrate them.

Acquired immunity is a double-edged sword. We pay a high price for protection. This is well illustrated in tuberculosis, resistance to which develops with maturity, due to frequent association with the tubercle bacillus. Those who survive enjoy some degree of protection, but this immunity of the survivors is gained at the expense of those who fell by the wayside. In very much the same way our resistance to other diseases is raised by frequent contact with the causal agent. Communities who live in sanitary isolation are particularly susceptible to new and strange infections which have reached an equilibrium with us. This is the case with tuberculosis among natives from Africa, with measles in the Faroe Islands and the Fiji Islands, with syphilis in a virgin population, with malaria among newcomers, etc.

Definition of Terms.—*Immunity* may be defined as the power which certain living organisms possess of resisting infections. *Susceptibility* is the contrary condition to immunity. *Hypersusceptibility* is a special state of an exaggerated power of reaction and is synonymous with *anaphylaxis*. *Allergie* is an altered process of reaction, often used as a synonym for anaphylaxis. The word *resistance* has practically the same signification as immunity. The term *tolerance* is commonly used to describe a limited form of immunity usually acquired by the repeated use of alkaloids, alcohol, and other poisons of comparatively simple chemical structure. While a high degree of tolerance may be acquired to such substances, a true immunity in the sense in which the term is now used is never produced. In the case of tolerance, antibodies are not found in the blood. For the most part true immunity is obtained against colloidal substances, while tolerance is largely limited to the crystalloids; this distinction, however, is not absolute.

There are all gradations and various kinds of immunity. It varies in

² We may speak of immunity "from" a disease, "to" a disease, and "against" a disease.

³ Terminal infections.

degree from the weakest appreciable resistance to an absolute protection. It also varies greatly in *duration*—from the briefest period to a life span. Immunity, therefore, is a relative term. It may be *natural* or *acquired*, *active* or *passive*, *local* or *general*, *pure* or *mixed*, *specific* or *non-specific*, *family* or *racial*, *brief* or *lasting*, *strong* or *weak*, etc.

Antigens are all substances causing changes in the blood, which thereby acquires specific affinity for the antigen. All known proteins act as antigens when introduced into the body parenterally. Antigens, then, are substances capable of inducing the production of antibodies.

Antibodies are specific properties of the blood and other body fluids induced by antigens. They are not necessarily “bodies,” but may be colloidal or physical states of the blood or other body fluids. Antibodies come down with the globulin fraction and seem to have definite chemical nature. Examples of antibodies are antitoxin, agglutinin, precipitin, opsonin, lysin, etc. Antibodies are also called *immune bodies*.

A *parasite* is a plant or animal living in or with some other living organism (called its *host*), at whose expense it obtains food, shelter, or some other advantage. Parasitism is probably a form of specific adaptation. *Ectoparasites* or *external parasites* live upon the host, as fleas and lice. *Endoparasites* or *internal parasites* live in the body of the host, as intestinal worms, anthrax bacilli, malarial plasmodia, etc.

A *saprophyte* is a microscopic being that lives upon dead organic matter. Saprophytes and parasites belong to either the animal or plant kingdom; thus, the malarial plasmodium is an animal parasite, the meningococcus a plant parasite.

Obligate parasites are those that live only in the living host, refusing to grow and multiply otherwise except on artificial culture media.

Obligate saprophytes are those which under no circumstances can be made to develop within the living tissues. Diphtheria and tetanus bacilli come close to being strict or obligate saprophytes, for they usually develop and produce toxins on a localized area of dead tissue. Many parasites are *facultative*, in that while they thrive best in the living host, they may also be made to grow and develop upon dead organic matter.

Symbiosis is a form of parasitism in which two dissimilar organisms live together in more or less intimate association, to the advantage of one or both, and not harmful to either; while *antibiosis* is harmful to host or parasite, or both.

Animal parasites have feeble antigenic properties; that is, they have feeble or no power of stimulating the production of immune bodies. Animal parasites therefore lack the power of producing lasting or high grade immunity. For the most part, they produce a resistance that is effective only while the living parasites are in the host. One attack does not confer protection against subsequent attacks, as in syphilis, malaria, leishmaniasis, and most other infections due to animal parasites. On the other hand, many parasites belonging to the plant kingdom, such as certain bacteria, are particularly

active in stimulating antibody production, and thereby produce a high-grade and lasting immunity. There are exceptions to this general rule.

The diseases in which one attack usually conveys a lasting immunity are plague, typhoid fever, cholera, smallpox, chickenpox, scarlet fever, measles, yellow fever, typhus fever and mumps.

The infections which do not confer lasting immunity are pyogenic cocci, gonorrhea, pneumonia, influenza, glanders, dengue fever, diphtheria, relapsing fever, erysipelas, malaria, syphilis, tuberculosis, and sometimes diphtheria and scarlet fever.

An *infectious disease* is a reaction in the host caused by a parasite. It is the result of a struggle between two variable factors—the pathogenic powers of the parasite on the one hand, and the resistance of the host on the other, each of these again modified by variations in the conditions under which the struggle takes place. An infectious disease, then, is a reaction, not an entity.

Septicemia, or *bacteremia*, is a condition in which microorganisms become generalized and circulate in the blood. They may grow in the blood, but more often their presence in the circulating blood represents an overflow from a local focus or organ where they are growing. *Toxemia* refers to the general symptoms produced by the absorption of bacterial toxin. *Sapremia* is a febrile condition resulting from the absorption of putrefactive products (not microorganisms) caused by saprophytic bacteria.

A *pathogenic* microorganism is one that harms the host. There are all degrees of pathogenicity. A *non-pathogenic* parasite does not harm the host.

Penetrability is an expression of the power of the microorganism to penetrate skin, mucous membranes and other structures in order to gain entrance. After penetration, the power to do harm depends upon invasion and virulence.

The *invasive power* is often spoken of as *virulence*, in contradistinction to *toxicity*. Toxicity implies merely the ability to produce poisons, and is not necessarily associated with the power to invade. Some organisms, like tubercle bacilli and the spirochetes of syphilis, have a very slow, gradual, but progressive power of invasion owing to the lack of acute physiological reaction on the part of the host, resulting from the presence of the microorganisms. Microorganisms that possess active invasive powers produce general infections, which are often malignant, but not necessarily so.

Communicability means the facility with which parasites pass from one host to infect another. The degree of communicability varies enormously in different instances, and depends upon many variable factors, especially the mode of exit, the manner of transfer, the susceptibility, the channel of entrance, the dose necessary to cause infection, etc. There is no relation between communicability and virulence. Thus, smallpox, chickenpox, measles, epidemic influenza and common colds have a high degree of communicability; diphtheria and scarlet fever are much less active in this regard, while leprosy is spread with difficulty. We are ignorant of some of the factors concerned in the transmission of disease.

Inheritance plays an important rôle in immunity. Infection often takes

a mild course among those races in which it has long been endemic, whereas the same disease suddenly introduced among a new people is relatively more severe and spreads more rapidly. Resistance as well as susceptibility may be transmitted from parent to offspring. In the case of natural immunity, the transmission is probably an instance of true inheritance, but in acquired immunity it is probably congenital. Compare heredity and hereditary transmission of disease, page 580.

Koch's laws or Koch's postulates are now stated as follows: (1) A specific organism must always be associated with a disease, (2) when isolated in pure culture and (3) inoculated into a healthy susceptible animal it must always produce the disease and (4) should be obtained again in pure culture.

In Koch's first paper ⁴ on infectious bacteria, presented in 1878, he states that "the repeated findings of microorganisms in traumatic infections and experimental results connected with these findings would indicate that these diseases are of parasitic nature. This can only be proved, however, when it will be possible:

- "1. To find the parasitic organisms in every case of the disease.
- "2. To find them in such numbers and so distributed that all the symptoms can be ascribed to the parasite.
- "3. To identify morphologically a well-characterized organism with each traumatic infection."

In 1885, Hueppe, a pupil of Koch, wrote a textbook of bacteriology ⁵ at Koch's suggestion. This book was written in the midst of the "spontaneous generation" controversy which circumstance influenced the form in which Koch's postulates are given:

1. It is to be determined whether, in decomposition or disease, bacteria are present or not.
2. If bacteria are present it is to be determined what forms they possess.
3. Each form found to be present is to be cultivated by itself, free from all chemical and morphological mixtures—"pure cultures."
4. By transfers of really pure cultures to decomposable materials or susceptible animals, it is to be determined whether the bacteria found are the cause of the decomposition or disease.

It was not until 1890, however, that Koch gave a clear statement of his fundamental concept of the relation of bacteria to disease. In discussing the view held by some at that time that bacteria only became pathogenic under the influence of the disease process he argued that:

⁴ R. Koch, "Untersuchungen über die Aetiologie der Wundinfectionskrankheiten," *Centralbl. f. d. med. Wissensch.*, 1878.

⁵ F. Hueppe, *The Methods of Bacteriological Investigation* (1885), p. 11. Trans. by H. M. Biggs, 1886, D. Appleton & Co.

1. If the parasite is found in every case of a disease and under conditions which conform with the pathological changes and clinical picture;

2. If it is not found in any other disease as an accidental and non-pathogenic parasite;

3. If after being completely isolated from the body and repeatedly transplanted in pure culture it can reproduce the disease on inoculation; then it cannot have an accidental relation to the disease, but the parasite must be the cause of the disease.

PUBLIC HEALTH ADMINISTRATION

Public health administration is the science and the art of organizing and operating governmental agencies whose purpose is to improve the physical well-being of the general population. It aims at the promotion of health as well as the prevention of disease. It deals more particularly with mass phenomena of the community than with problems of the individual, despite the fact that public health is the sum total of individual health. All public health work is based upon the four fundamental needs of human welfare, namely: food, shelter, defense, and propagation.

Public health administration is much better under professional than under political control: (1) It is part of the political machinery of government and in that sense cannot be divorced from politics. (2) It should cooperate with volunteer health organizations, but there is a place for official and non-official agencies which should not be overstepped. (3) It should establish relations with medical, nursing, dental, engineering and other professions. A good health program cannot succeed in face of opposition of the medical profession. The proper relationship is a serious and difficult problem.

There are three ways of obtaining results: (1) through military or arbitrary ways; (2) through legislative or punitive ways; and (3) through the establishment through education of standards through group consciousness.

Public health administration must be kept elastic to meet the changing conditions and growing knowledge. Any rigid system will soon become antiquated in this period of transition. Continuity of service and effort are essential to success.

Public health administration necessarily varies with time and place. Sanitation comes first. After a community has a good water supply and an efficient sewerage system, it can then give attention to hygiene. Communities afflicted with threatening conditions as plague, malaria, typhus fever, hook-worm disease, must needs concentrate upon these problems. The threat of an epidemic or the fear of pestilence is a strong lever in public opinion to obtain legislation, appropriations, and support for public health activities.

A good public health administrator has an enviable opportunity to study disease and advance knowledge. Good administration includes investigation.

The project or concentration method has advantages. It consists in

focusing all energy in one direction such as a drive for diphtheria, tuberculosis, etc., instead of scattering and diffusing effort by doing a little of everything.

Official activity is limited by police power, hence the importance of education and coördination. The sympathetic understanding and coöperation of the individual citizen is essential for success.

Fortunately, in public health work, perfection is not necessary for the control of infection. Yellow fever or malaria will decline and even vanish long before the mosquitoes are exterminated in any region. Furthermore, there are multiplicity of causes which in conjunction, conspire to promote epidemics so that partial control of one of these causes may break the link in the chain. The prevention of disease is not the sole object of public health administration. It aims at improving health, increasing efficiency and promoting happiness, and affords better opportunity for service. Public health administration under rural conditions is discussed separately under rural sanitation, on page 499.

RELATIVE VALUES OF PUBLIC HEALTH WORK

It is evidently impossible to express with mathematical precision the relative importance of different public health procedures. Nevertheless, efficient administration requires a sense of proportion. The relative values of health activities vary with location, character of population, stage of civilization, etc. In many communities malaria, dysentery, hookworm disease, plague or yellow fever will need chief consideration. A well-sewered city can appropriate money for lines of work which in less fortunate places must be devoted to the never-ending task of privy sanitation.

Standards aid to balance health work and serve both as a guide and protection. Standards of public health practice should be based on service. In other words, work done is the criterion of excellence. Any rating based upon money expended, personnel employed, or mortality rates found may be quite illusory. Standards cannot be fixed and should not be rigid but should be revised from year to year. In determining the relative value of health work, it is necessary to take into account (1) the number of deaths and cases, (2) the economic factor, and (3) the feasibility of prevention.

Sanitation or municipal housecleaning logically comes first; this accomplished, hygiene must occupy most of the attention of the public health administrator. Disease prevention is important, but health conservation is fundamental; education is essential, but scientific research is basic. The ideal of preventive medicine is to build towards departments dealing with health rather than departments dealing with disease.

In what way should the appropriations for the health department be expended so as to save the most lives and prevent the most sickness? Chapin⁶ answers this question in the following table:

⁶*J. Am. M. Ass.*, 1917, 69: 90.

RELATIVE VALUES OF HEALTH WORK

Communicable diseases . . .	{	Medical inspection	100	
		Hospitalization	50	
		Immunization	50	
		Venereal diseases	20	
		Tuberculosis	{ Nurses 60 Dispensaries 40 Hospitalization 40	
Child hygiene . . .	{	School inspection	80	
		Infant mortality	Nurses	80
			Supervision of midwives	10
			Boarding houses	5
			Milk stations	5
			Consultations	20
			Prenatal clinics	10
			Privy sanitation	110
			Housing	20
			Plumbing	10
Nuisances	10			
Food	{	Fly and mosquito control	10	
		Adulteration	0	
Milk	{	Sanitation	10	
		Adulteration	3	
	{	Sanitation	17	
		Care of sick poor	50	
Laboratory			50	
Education			80	
Vital Statistics			60	
<hr/>				
1,000				

The figures given in the schedule are intended to indicate the real health conserving value of certain common functions of municipal health departments. For many activities we have no measurement, but only guesses.

A Score for Health Activities.—The New York State Department of Health has prepared an activities score for cities with a population of from 25,000 to 175,000 inhabitants. Of a possible thousand points for perfect, adequate public health nursing service counts seventy-five; other follow-up social service ten; adequate dispensary or clinic service seventy; hospital facilities for the communicable diseases forty-five; a day nursery ten; Little Mothers' League ten; good newspaper publicity regarding health matters fifty; and a physician in charge of the infant welfare station fifteen. This gives a total of 285 points for activities in which the nurse is directly concerned. In general the score provides the following distribution of credit:

Communicable disease control	
Tuberculosis, perfect score.....	60
Venereal diseases, perfect score.....	70
Other communicable diseases, perfect score.....	80
Adequate laboratory facilities and use of same.....	100
Infant and maternal welfare.....	90
Milk and food inspection.....	100
Water supply.....	100
Sewage, garbage and manure disposal.....	40
Record keeping.....	85
Public health education.....	120
An appropriation of at least 50 cents per capita for health protection....	100
Effective enforcement of regulations governing barber shops, common towels, drinking and eating utensils.....	20
Unusually meritorious public health work along either new or old lines..	35
 TOTAL.....	 1,000

In any system of scoring, the weight of a disease depends upon (1) the number of cases and deaths, (2) the economic importance of the disease, and (3) the feasibility of prevention. Rankin is an enthusiastic advocate of standardization of public health work. He appraises health work in accordance with a rating schedule for weighing health work in terms of group judgment. See Appraisal Form for City Health Work, by the Committee on Administrative Practice of the American Public Health Association, 1926.

A Public Health Program.—The principal elements in a comprehensive public health program, in ordered sequence, may be named as follows:

1. Eugenics, the principles of sound breeding and heredity. Immunity.
2. Maternity and the care, protection and encouragement of the function of motherhood. Includes prenatal work.
3. Infant welfare and the reduction of infant mortality.
4. The health and development (physical, mental and moral) of the preschool child, the school child and adolescent.
5. Food and nutrition, the relation of diet to growth and health.
6. Personal hygiene. Mental hygiene.
7. Industrial hygiene, the health of the worker.
8. The prevention of the communicable diseases. Epidemiology.
9. The prevention of the non-communicable diseases.
10. Sanitation, or biologic cleanliness, including improved environment. Sanitary engineering. Water and wastes.
11. Vital statistics, the bookkeeping of humanity.
12. Education, the diffusion of knowledge among the people in all these matters.
13. Research to extend the boundaries of knowledge.

Successful public health administration should be organized so as to include constructive work under each of these headings. In a broad sense, it should also include improvements in education for medical, dental, veterinary, nursing and public health practice.

Organization of Health Departments.—The administration of a health department should include as much as possible of the above public health program. Ordinarily, health work falls under the following headings:

1. Executive and administrative
2. Vital statistics
3. Communicable diseases—epidemiology
4. Infant welfare and child hygiene
5. Food and drugs
6. Sanitation
7. Industrial hygiene
8. Laboratories
9. Education

A health department should have a "commissioner of health" or "health officer" as executive head of all activities. The chief executive may have a "public health council" or "advisory board" to approve policies and regulations, but such boards should have no executive duties. In metropolitan cities and states, the health departments should have skilled and trained heads for each of the above-mentioned departments. It is sometimes advisable to have special departments for some of the major public health problems, such as tuberculosis or venereal diseases; or malaria, hookworm, plague, etc., where such diseases prevail.

The smallest health department must contain all the elements essential to public health work, and should consist of at least one health officer, one public health nurse and one clerk, all full-time employees. The health officer for small communities must be epidemiologist, educator, dispenser and school physician, bacteriologist, inspector, executive, etc.—a veritable Jack-of-all-trades. He should be free to call upon expert advice when confronted with an unusual situation. Such specialists are provided by the United States Public Health Service, by state departments of health, by universities and other organizations.

It is an advantage for a number of small communities or counties to pool their health interests and resources. This has been tried at Wellesley, Massachusetts, where five of the neighboring towns have entered into a coöperative health organization.

Each county may have its own health organization, and all progressive states are divided into sanitary districts presided over by a district health officer.

Cost.—The appropriation necessary to maintain a minimum health organization for cities varies from 50 cents to \$1.00 per capita per annum. For

a city of 10,000 population, 75 cents per capita would be sufficient to maintain a minimum organization as follows:⁷

One health officer.....	\$3,000
One public health nurse.....	1,200
One clerk	1,000
<hr/>	
Salaries	\$5,200
Maintenance	2,300
<hr/>	
Total (<i>all full-time</i>).....	\$7,500

In a city of 25,000 population, 75 cents would hardly cover the expense, as a city that size should have a small isolation hospital. The organization for a city of 25,000 follows:

One health officer.....	\$3,500
One epidemiologist.....	2,500
Three public health nurses—\$1,200.....	3,600
One milk inspector.....	1,500
One sanitary inspector.....	1,200
One statistical clerk.....	1,200
One stenographer.....	1,000
<hr/>	
Salaries	\$14,500
Maintenance	4,250
<hr/>	
Total (<i>all full-time</i>).....	\$18,750

In this organization, the epidemiologist performs the duties of bacteriologist, and he divides the duties of school inspector and dispensary physician with the health officer.

No provision is made for the collection of garbage and other city wastes, street cleaning, etc. These activities quite properly belong to a separate department of the city government.

In some cities and states, the management of public hospitals, tuberculosis sanatoria and insane asylums are placed under the control of the health office. The organization of health departments varies in different parts of the country to meet special conditions and local traditions. (For counties, see page 501.)

THE MEDIAN ENDEMIC INDEX

In epidemiological work, it is essential that the health officer have some system by which he may compare the incidence of disease in the past with that

⁷ C. Fox, "Minimum Standards of Organization for Municipal Health Departments," *J. Am. M. Ass.*, 1920, 75: 790.

of the present. For this purpose, W. H. Brown, then Epidemiologist of the Massachusetts Department of Public Health, devised an endemic index card. The endemic index was determined by averaging the number of reported cases for five years, exclusive of epidemics, for the various municipalities of the state. This average was the endemic index. The personal factor, however, entered into this determination as it was necessary for the epidemiologist to decide what constituted an epidemic.

To obviate this difficulty, Hitchcock and Carey of the Massachusetts Department of Public Health devised a median endemic index. This median endemic index is a record of actual occurrence and takes into consideration all cases reported. The data is arranged in arithmetical sequence, and the median selected as the index. For example, the number of cases reported in Boston for the month of January for nine years was as follows:

Year	Diphtheria	Scarlet Fever
1910	912	943
1911	992	631
1912	519	583
1913	555	833
1914	846	1,584
1915	921	953
1916	840	948
1917	869	673
1918	950	609

By arranging these numbers in arithmetical sequence and picking the median for the index, the median indices or "expectancy" for January are found to be:

Diphtheria—869

Scarlet fever—833

The monthly endemic index based upon deaths is shown in the following table (based on figures for 1910 to 1920):

Month	Tuberculosis, Pulmonary	Scarlet Fever	Typhoid	Diphtheria	Measles
January	403	17	12	63	24
February	391	24	13	63	26
March	450	23	12	56	29
April	428	18	9	50	31
May	394	20	14	40	36
June	356	13	13	35	30
July	349	8	18	34	24
August	322	5	23	27	10
September	318	4	30	37	6
October	317	8	35	55	7
November	321	9	24	66	10
December	350	16	22	71	18

The monthly endemic indices for the various diseases are plotted on a graph, and beside it is placed a dial marked off in suitable spacing to show

the daily increase. This is recorded by two clock hands, the hour hand pointing to the endemic index for the month and the minute hand progressing as cases accumulate.

An endemic index card is kept for each city or town in the state. This card is ruled off in six large spaces on each side for the months of the year. Each of these large spaces is in turn ruled off into smaller spaces for the days of the months and the disease reportable. The number of cases reported the previous year and the endemic indices for the various diseases are also recorded. As daily reports are received, they are tabulated in their proper places.

The median index chart used in conjunction with a spot map and daily index cards is a simple and effective device whereby the daily accumulation of reported disease may be watched and which will show at a glance any sudden or unusual increase.

HEALTH CENTERS

*Health centers*⁸ are local agencies where the health activities of a community are administered. This concentration increases efficiency and promotes economies. Health centers are needed in metropolitan cities as well as in country districts; their organization, however, will vary somewhat in large cities, in medium-sized towns and rural situations. Health centers are "clearing houses" for all hygienic, sanitary, medical, dental, nursing and other public health activities. They also serve as convenient places for advice and information; they are useful for promoting public health education and they may contain attractive social features.

One of the faults with public health administration is that it is too remote and removed from the people and their problems. Public health centers correct this fault in an admirable way. The health center is the means of reaching the individual and helping his problems of hygiene and difficulties of adjustment in the home, in school, and workshop. It provides a personal touch with individual and family units in their own environment. The Blossom Street Health Unit in Boston combines all of the health agencies, public and private, as well as the welfare agencies, coördinating their activities and conducting a battle against disease, with marked emphasis on prevention. It is proper for a health center to have facilities for prenatal work, maternity care, baby hygiene station, child welfare, tuberculosis clinic, venereal dispensary, dental service, nursing service, Red Cross activities, mental hygiene, distribution and administration of serums and vaccines, physical examinations, surgical emergencies, etc., etc. Health centers may also serve as the district office for federal, state and local health officials, as well as for private organizations. Charity and material relief should be kept separate from such centers. All these health and welfare agencies are grouped under one roof.

⁸ A symposium on the Health Center, *Am. J. Pub. Health*, 1921, 11: 212. Wilinsky, C. F.: "The Health Center," *Boston Med. and Surg. J.*, 1924, 190: 14; also *Monthly Bull.*, Health Dept., City of Boston, 1924, 13: 11.

The movement was started in 1912 by a resourceful leader in the tuberculosis campaign in Pittsburgh, W. C. White, who provided a common meeting place and office for the volunteer servants of the sick. A similar plan was adopted in Milwaukee where emphasis was laid on saving babies' and mothers' lives in a definite area by Wilbur C. Phillips. The next year (1913) New York established a health center in the Syrian district, for little children. It was not, however, until 1914 that a glimpse of this new method of health administration was established by S. S. Goldwater, Health Commissioner of New York, down in a tenement store on Division Street in New York's ghetto, in the shadow of a block where about one thousand persons crowd the acre. This was the first experimental station under municipal auspices, started as a health center, with a desk and chair, and manned by visiting nurse, a charity worker, the district's physicians and the sanitary inspector. In the same year Buffalo made use of district dispensaries; and in 1915 Cleveland put its city health stations on a district basis. It remained, however, for Boston to put the project on a definite basis and place it among the permanent features of civic government. Through the generosity of George Robert White and the wisdom of the trustees, a health unit was built and turned over to city, November 1, 1924, under the capable direction of Charles F. Wilinsky, Director of Health Units.

Health centers are the next step in health community service. Their possibilities are boundless, but their successful operation requires coöperation and leadership.

Hospitals.—*Medical social service* occupies a strategic position in the community for public health service. A hospital should be regarded as a community responsibility, with the chief function service to the patient. The hospital which confines its work within its four walls does not meet its obligations.

Only about 15 to 20 per cent of the sick in any community are cared for in hospital, which mostly serve the rich and poor. The great middle class (80 per cent) are largely omitted. The hospital has a legal responsibility for medical and surgical care, but preventive measures are sometimes sacrificed. There must be a connection between hospital and home, occupation, and habits. The curing stage in hospital is only an episode in a chain of events for the individual and this link must not be disjointed. Hospitals then must consider outpatient and dispensary service, diagnostic clinics, public health and visiting nursing and correlation with the activities of the various health agencies. These efforts should not be isolated and independent. The entire community should be mobilized for best results in the interest of every energy and of efficiency. The hospital is in a strategic position especially in rural communities for all public health activities. Medical, surgical, obstetrical, nursing, and dental and health services can be combined with advantage in small communities.⁹

⁹ "Hospital Administration and the Training of Hospital Executives," Reprint of Committee on the Training of Hospital Executives, April, 1922.

Good hospital administration, therefore, correlates its curative with preventive work. One of the functions of a useful hospital is to keep people out of hospitals. Hospitals do much effective preventive work as exemplified by their prenatal clinics, health examinations, mental clinics, baby and child welfare, and social service work, etc. Hospitals with their outpatient clinics can do much of the work of a health center.

The prevention of the non-communicable diseases has not been given the consideration it deserves. This list includes nervous and mental disorders of all sorts, nutritional derangements and the deficiency diseases, defects due to bad health habits, diabetes, cardiovascular troubles, and orthopedic abnormalities which combine prevention, care, restoration of function and re-education. The prevention of heart disease, which affects about 2 per cent of the population and is mostly contracted in early life, usually follows infections of the throat and teeth, or an attack of acute rheumatic arthritis, or scarlet fever, diphtheria, chorea, etc. This is fundamentally a problem of the prevention of such infections, although the cardiac complications are commonly considered under the non-communicable diseases. The prevention of cancer and other diseases is considered under such topic. One means of preventing this group of diseases is through a periodic health examination.

PERIODIC PHYSICAL MEDICAL EXAMINATION

Such examinations should begin in infancy and continue through childhood, puberty, adolescence, and adult life. Each period has its special problems. In general the periods may be on a birthday basis, although at certain ages, especially infancy, the examinations should be more frequent. Permanent and continuing records should be kept. The examinations serve best in the hands of the family physician who can use the conditions found in health with those that develop in disease. Special institutions, clinics, insurance companies, and other agencies are specializing in health examinations.

The work must be well done if it is to succeed. The technic of making the examination must be learned. The idea of a health examination is not new, but we have not been ready until recently to realize this ideal. It is a wise and useful innovation growing apace, and it is evident that it will in time become a part of community life. For centuries the medical profession placed its emphasis upon disease, and gave heed only when disorders were well developed. The beginnings of disease were largely neglected and the study of the normal not given the thought it deserved. We have worked backward and now due regard is paid to the borderline state between health and disease; still farther are our investigations being pushed for a better understanding of normal functions. In view of the fact that more is known about disease than about health, it is essential to proceed cautiously concerning abnormal deviations in those in apparently good health.

A complete physical examination should be done with deliberation and

care. It includes the family history; previous history; occupation; habits, as to food, sleep, exercise, alcohol, etc.; working conditions; present condition of the respiratory system, cardiovascular machine, nervous and mental states, and gastro-intestinal tract; a physical examination of nutrition, eyes, throat, teeth, genitalia, orthopedic defects, glandular disturbance, reflexes, and special tests such as blood-pressure, blood and urine examinations, Wassermann tests, x-rays, etc. The subject is well considered in *A Handbook for Health Examinations by Physicians* prepared by the Committee on Public Health of the Massachusetts Medical Society, *Boston Medical and Surgical Journal*, 1925, 193: 947.

No one would now wait for a toothache to drive him to a dentist. The same idea is sensible and will obtain with other structures and functions of the body. The day of practical prevention is at hand.

PERSONAL HYGIENE

The chief object of personal hygiene is to use and enjoy the blessings of health. Good health makes possible the highest enjoyment of life and the best service in the world. Health does not consist simply in keeping out of the hospital; it is positive, not negative. Good health consists in keeping the body and mind at the best level. It requires that each person study his powers, learn his needs, exercise his abilities, improve his resources, expand his possibilities, develop his mind and body, respect his limitations, and thus be prepared for the daily needs of life and be ready to meet emergencies as they come from time to time. There are times when it may be noble to neglect health; there are times when it may be justifiable deliberately to sacrifice health. Some things in the world can be done only by paying the price. This is the case with heroism, creative work and child-bearing. Sacrifice of health is sometimes made in the home and in the workshop as well as on the battlefield, but the sacrifice of health in the pursuit of personal or selfish gain cannot receive approval. Many of the greatest contributions to the welfare of mankind have been made by men in poor health; the list is legion.

We do not seek health for health's sake, but for the sake of usefulness in the world. Health is not the end, but a means to service and satisfaction. The strong and vigorous man, if selfish in these matters, is socially sick. A healthy man finds joy in play, satisfaction in work and inspiration in service. Health permits one to "live most and serve best."¹⁰

The student of preventive medicine is more interested in living well than in living long. Longevity is an index of good hygiene and sanitation, of peace and prosperity. Longevity is not an end in itself, except as it may serve us to live better, cleaner and more useful lives.

The physical and mental powers, if normal, should be exercised and strengthened; if handicapped, they should be corrected. If these deficiencies

¹⁰ This thought is developed in an inspiring way by Williams, in *Personal Hygiene Applied*, W. B. Saunders & Co., 1922, which the student is advised to read.

are not amenable to treatment, the individual must learn to adjust his activities within the power of his body. This is a problem of compensation. To drive at high speed a machine that is built to go thirty miles an hour invites disaster. This is a problem of personal hygiene—it is individual. It depends first upon a recognition of the trouble, its nature, its course and its effects; then an intelligent adjustment to make life satisfactory within one's limitations.

Those who are endowed with a robust frame, sturdy physique, sound functions, a good constitution and hereditary advantages must also learn the limits to which the human machine may be driven without permanent harm. One of the important lessons of personal hygiene is to develop good inhibitions, which are fundamental for a temperate, normal life. This is a problem of character building and the most significant aim of personal hygiene which each person must learn for himself.

To follow our instincts blindly would be to live on the plane of the lower animals. Persons who do this fall into grave error, especially in matters of hunger, exercise and sex. Instinct fails to protect us from disease germs; it fails to appreciate the needs and rights of others and thus falls far short of the mark. Our sensations are not always to be trusted: we may feel tired, yet need exercise; we may feel energetic and excited, yet need rest; we may not be thirsty, yet need water; we may feel hungry, yet not need food. Our sensations regarding sex may lead us astray and deplete our vitality. Instincts and sensations must be watched, studied, interpreted and often curbed, sometimes sublimated.

We are still ignorant of many of the fundamental factors which underlie the art of hygienic living. There are many unsolved problems concerning diet and nutrition, work and play, exercise and rest, bathing and cleanliness, air and ventilation; as well as water, clothing, etc. We are still densely ignorant concerning many facts in heredity, mental processes, and the sum total of effects which we call behavior. We know more about disease than we do about health. We are not sure enough of our ground with most of the common and daily things that influence health to justify dogmatic teaching. There is more loose talk concerning personal hygiene than any other chapter in preventive medicine. The field is still ruled largely by empiricism and somewhat by fads and fancies. There is some information, little knowledge and scant wisdom on these points. The safest plan is a common sense middle ground that will regulate our health habits to accord with clean, moderate and moral living.

The prevention of infection is only one phase of personal hygiene. The individual may protect himself against many diseases, but the problem of preventing the spread of many communicable infections is usually one of public hygiene and community sanitation. Personal prophylaxis is discussed in detail under each communicable disease in other parts of this book.

One way to maintain and promote health is through periodic medical examinations, which should start with infancy and continue throughout life

(see page 496). Mental health is discussed in the section on Mental Hygiene, page 433. There are hints concerning personal hygiene, as far as our knowledge goes, in every topic throughout the book.

UNITED STATES PUBLIC HEALTH SERVICE

The Public Health Service is a bureau in the Treasury Department presided over by a Surgeon General with a commissioned medical corps and other special experts. Its duties, broadly speaking, are: the protection of the United States from the introduction of disease from without, and the prevention of interstate spread; the suppression of epidemics; the investigation of diseases; and the dissemination of health information. It also supervises the manufacture of biologic products, and coöperates generally with state and local health departments in assisting and giving advice in health matters. The accomplishments of the Public Health Service are extensive and varied, ranging from the maintenance of foreign and domestic quarantine service, the control of plague and other communicable disease, to important discoveries in tulariaemia, Rocky Mountain spotted fever, typhus fever, pellagra, beriberi, trachoma, hookworm disease, and others.

The United States Public Health Service is an admirable example of governmental usefulness. Its many activities and achievements are recorded in the annual reports of the Surgeon General.

RURAL SANITATION

The country is the weakest link in the sanitary chain. This is largely due to economic reasons, with the added handicaps of inadequate organization, great distances, and imperfect education. Most rural health departments are undermanned, poorly supported, and render inadequate service.

The first full-time county health officer in the United States was established in 1911 in Yakima County, Washington, by L. L. Lumsden and E. R. Kelley. The movement is making slow but steady progress; in 1921 there were 161 counties having the whole-time services of a county or district health officer; in 1922, there were 202; in 1923, 230; in 1924, 250, and in 1925 the number increased to 280. There are 2,850 counties in the United States, wholly or in part rural. In 1925, the rural population of the United States was 51,406,017, of whom 7,138,838 or 13.88 per cent had local health service under the direction of whole-time health officers.

From the standpoint of many of the communicable diseases, our better cities are now much safer places to live in than country districts. Before the days of modern sanitation the cities had about double the death rates of the country. The improvement in the death rate which has been going on during the last two or three decades is more apparent in urban than in rural populations.

Infection flows from the country to the city in the water, milk and food

supplies. It is carried back and forth in the persons of cases and carriers. Opportunities for contact infection are much more frequent among the rural population than is ordinarily conceived; disease transmitted by contact infection often spreads through sparsely settled country districts like wildfire.

Typhoid fever, malaria, hookworm, and poliomyelitis are more prevalent in the country, while diarrhea, enteritis, diphtheria, measles, scarlet fever, tuberculosis, meningitis, smallpox and lobar pneumonia are more prevalent in cities. The contact diseases, especially those of the upper respiratory tract, which prevail in the wintertime, not only have a higher incidence in the city, but occur at an earlier age period.

Rural dwellings, schools and meeting houses are often unsanitary, crowded and dark. The water supply is often unsatisfactory. Sewage disposal is apt to be primitive and the control of insects, rats and vermin difficult and neglected. Heating, lighting and ventilation are often defective. There is plenty of sunshine in the fields, but often none in the houses. Further, it is rather difficult for each farmhouse to surround itself with the tidy environment equal to that of a city park. The best medical service has a tendency to desert country practice for city allurements. This is largely an economic problem, bound up with the gregarious instincts of man. The glorification of the country doctor is typified in the life and achievements of Jenner, and notably depicted in Ian Maclaren's *Doctor of the Old School*.

The tendency for the population to concentrate in cities is shown by the following figures of the United States Census giving the distribution of population in the United States, over 2,500 being considered urban.

Distribution	1920, per Cent	1910, per Cent	1900, per Cent	1890, per Cent
Urban	51.4	45.8	40	35.4
Rural	48.6	54.2	60	64.6

There is a different age and sex distribution, a larger number of middle-aged people living under urban conditions.

The farmer's work is fatiguing, intensive and seasonal. It often requires undue exposure to wind and weather both summer and winter. The hours are long and the conditions of work and life not always conducive to health, longevity and efficiency.

About 50 per cent of our population is rural, but this half receives scant attention. Nearly twenty million people in the United States, or about one-fifth of the population, live in villages, and thirty million farming folk use these villages for the purpose of business, education, religion, health and source of well-being. Fortunately the "remoteness" and "isolation" of the country is disappearing with better roads and transportation, telephone, radio, etc. Rural sanitation is expensive, but costs less than sickness, with its attendant inefficiency and block to progress caused by disease.

The line of demarcation between rural and urban is arbitrary. For

statistical purposes of mortality and morbidity the United States Census Reports up to 1909 regarded all communities over 8,000 as urban, but since 1910 the line of demarcation has been raised to 10,000. For the purpose of classifying the population irrespective of disease, the United States Census since 1900, however, draws the following distinction. All incorporated places (and all towns of Massachusetts, Rhode Island and New Hampshire) having 2,500 inhabitants or more are classified as urban, and the remainder of the country as rural.

Many factors have recently conspired to make conditions of life in the country more healthful. The situation is improving. The agencies that make for health and betterment of country conditions in chronological order are: rural free delivery, good roads, farm demonstrations of the Department of Agriculture, home demonstrations of dietetics and home economics, county health service, consolidated schools, and rural social service work. The health work is aided by many collateral factors in a general program to promote welfare and happiness in the country.

There should be a full-time health officer in charge of a county or a rural sanitary district, with a health center and staff to carry on the needed work. Hospital, transportation and nursing services should be organized and provided in each sanitary district on an economic basis.

The method of controlling disease, disposing of wastes, improving housing and ventilation and the art of sanitary living both in country and city are discussed in detail under specific chapters throughout this book.

Organization and Cost of County Health Service.—There is one tried and proved method through which public health work may be conducted satisfactorily: by local whole-time health service. State and federal health agencies must assist in initiating county service and share in the work. The International Health Board of the Rockefeller Foundation has shown commendable spirit in initiating, stimulating, and supporting better rural health service through full-time county organizations. The county itself should provide at least one-half of the budget at the outset, and a larger percentage in succeeding years. In 1925, in about twenty of the states such appropriations have been provided. The Public Health Service is giving assistance to about seventy-six counties in nineteen states and the International Health Board to about one hundred and five counties in about twenty-three states.

The cost of a county health department will vary with the area, population, and taxable resources of the county, and with the willingness of the people to provide themselves with health service. It is good economy to spend money generously for health administration.

For all except the most sparsely populated and poorest counties the minimum cost of a county health department should be at least \$10,000 a year. In the more populous counties a larger budget will be needed to secure adequate service. In general, it may be stated that an expenditure of 50 cents per capita per annum should furnish a county with reasonably adequate health service. (This does not include hospital expenses, bedside nursing, or pauper

relief.) A budget of 25 cents per capita should be the minimum in any except the poorest counties.

The minimum personnel should include a full-time medical health officer, one nurse or sanitary inspector, and an office clerk. A much more efficient organization will be secured if both a nurse and sanitary inspector are provided. Larger organizations include additional nurses and inspectors, and in some instances a dentist, sanitary engineer, nutrition worker, bacteriologist with laboratory, etc.

Typical budgets may be distributed as follows:

Item	County A	County B	County C
Salary, county health officer	\$ 4,800	\$ 4,200	\$ 3,600
Salary, public health nurse	2,000	1,800	1,800
Salary, sanitary inspector	1,800	1,500	1,500
Salaries, additional nurses or inspectors ..	3,000	1,500
Salary, office clerk	1,200	900	750
Travel expenses	2,400	1,800	1,800
Contingent expenses	800	800	550
TOTAL ANNUAL COST	\$16,000	\$12,500	\$10,000

In addition to the above amounts, the county should provide and equip suitable quarters for the health office, preferably in the court house or in some other central location.

Villages and sparsely settled districts either need subvention or should organize with their neighbors on a coöperative basis. In New England the town takes the place of the county organization.

Camp Sanitation.—The health hazards in tourist camps and in vacation resorts are often great because sanitation is primitive and neglected. Summer hotels, cottages, inns, and other vacation quarters come in the same category. Too frequently persons who seek health and recreation find discomfort and disease. The automobile and the airplane are new methods of dissemination of infection.

PUBLIC HEALTH EDUCATION

Public health education is fundamental. It must be considered under two aspects: (1) acquisition of knowledge and (2) diffusion of knowledge.

The first involves researches in laboratories, studies at the bedside and investigations in the field. The demonstrations of science underlie all sound health administration and are the only safe guide for health education. Special schools are needed to prepare for public health service; and better instruction in hygiene and sanitation is called for in medical schools.

In the diffusion of knowledge concerning health and the prevention of diseases, public health authorities should take a leading part. Many other agencies, however, assist in placing the facts plainly before the people. Health education should begin in the home where the developing child is taught the art of sanitary living. The schools should teach the structure and functions

of the body, hygiene of the person and sanitation of his surroundings. This is a most important part of the curriculum from kindergarten to university.

The popular methods in health education are through newspapers, periodicals, pictures, bulletins, exhibits, radio, lectures, movies and feature campaigns, such as "Health Week," "Baby Week," "Clean-Up Week," etc. There should be guides and counselors in health matters. Hospitals, dispensaries, and health centers, as well as nurses, social service workers and others, are effective and helpful.

Coöperation and assistance in health education may be obtained from the United States Public Health Service, from state health departments and local health officers; from the Federal Child Bureau, as well as the United States Bureaus of Education, Census and Mines; also from the United States Department of Agriculture, The American Society for the Study and Prevention of Tuberculosis, The Red Cross, The Russell Sage Foundation, The International Health Board, The Metropolitan Life Insurance Company, The American Society for Control of Cancer, The American Safety Council, The National Child Welfare Committee and other agencies.

It is very important that the facts stated in popular propaganda should tell the truth. It is more important to be correct than to be clever. Half-truths are often dangerous. Wrong teaching undermines confidence in health authorities. It is a question whether the subject is dignified by the use of circus methods or the antics of the clown.

HOUSING

Housing has an intimate relation to health. Housing influences morbidity and mortality, but in a rather indirect way. It is difficult to separate the factors of crowding, personal habits, poverty, food and other hygienic and sanitary influences from the actual housing conditions. A house after all is only an instrument that may be abused; thus a good house may be crowded and unsanitary, while a poor house may be clean and fairly adequate. Good housing conditions facilitate an adequate supply of fresh air and sunshine, promote cleanliness and dryness, and favor sanitary isolation; good housing also encourages higher standards of living, and thus favors better personal hygiene and improved sanitation of the environment.

Housing conditions may be taken as a good index of the general sanitary condition and hygienic habits of the occupants. Strict laws concerning new construction should be rigidly enforced, especially with respect to safety, air space, openings for air and sunshine, water supply and disposal of wastes, cellars, toilets, kitchens, etc. Regulations concerning the number of occupants and the use of the house should be made a matter of supervision through official inspection. A good zoning system has health advantages.

There is a difference of opinion as to the function of a health officer in relation to housing. In my judgment, it comes under the purview of good health administration, although it is largely an economic and social prob-

lem. Most of the laws in the United States concerned with housing deal more with structural safety than with sanitary requirements.

The modern tendency is to leave the country for the city. About one-half of our population is urban. Cities are congested and crowded, and many live in cells unfit for human habitation. The best results in health cannot be obtained and maintained when so large a proportion of our inhabitants live such a parasitic life. Housing is an index of these conditions and tendencies, which favor the spread of all communicable infections. Further, such artificial and unnatural conditions of life make it difficult to maintain tone, vitality and efficiency.

Housing affects health, morals and progress both directly and indirectly. Dark rooms favor the spread of tuberculosis. Ordinary window glass cuts out most of the ultraviolet rays that play a rôle in rickets. Dingy houses make cleanliness difficult and give comfort to vermin. Indirectly, bad housing conditions may undermine resistance to disease, especially through poor lighting, bad ventilation, and improper heating. A lack of housing facilities is a serious sanitary menace.

One of the most important factors in housing in relation to health is overcrowding. Strict regulations should be enforced by the health officer to prohibit the overuse of living and sleeping rooms. Careful sanitary supervision is necessary to insure an abundant supply of good water and an adequate system of disposal of wastes. The health officer should also take cognizance of nuisances often maintained both within and without houses. It is usually easier to prevent such nuisances than to abate them. The records of the health officer should show the cases of sickness occurring in each house, for much useful information is thus obtained.

The general types of dwelling concerned in urban housing problems are: (a) one-family dwellings, (b) two-family dwellings, and (c) tenements. The detached one-family dwelling with space all around it is the ideal.

Good housing includes a consideration of construction, site, soil, dryness and drainage, water and wastes, lighting, heating and ventilation, size, arrangement and use of rooms, facilities for cleanliness, and also the environment of the structure. Each one of these topics is discussed in detail in other chapters (see Index).

INFANT MORTALITY

"Infant mortality is the most sensitive index we possess of social welfare. If babies were well born and well cared for, their mortality would be negligible" (Newsholme).

Of every five babies born up to 1900, one died before it was able to walk and talk. In 1900, only one death occurred in ten; and in 1922 only one out of thirteen died before it was one year old. The awakening of the world to the consciousness of the immense and needless sacrifice of infant life is recent. Most of it has come since about 1870, as the result of statistical studies

showing how colossal has been this "slaughter of the innocents." The reasons for regarding infant mortality as a major public health problem are humanitarian, social and economic. Good results in lessening infant mortality depend entirely upon attacking the underlying causes with intelligence. This is one of the functions of every well-organized health department. Rewardful results are purchasable.

Queen Anne had eighteen or nineteen children, none of whom were living at her death. Most of them died in infancy, and only one—the Duke of Gloucester—reached the age of eleven years. In those days, they "did not expect to raise them all." While conditions have greatly improved, infant mortality almost everywhere is still excessive. The best rates are from New Zealand. Our own figures are meager and imperfect. Comparisons may be misleading unless we are assured of the accuracy of birth and death returns and other factors.

Definitions.—Infant mortality means deaths under one year of age. The infant mortality rate is the ratio of deaths under one year to births during the same period; the births being the easiest method of determining the number in this age group. There are a number of inaccuracies in infant mortality rates, which are mostly too high, because of incomplete birth registration, while that of deaths is usually fairly reliable (page 1170).

Stillbirths are not included in infant mortality. Registrars, however, require all premature and all stillbirths to be recorded by a birth and reported upon a death certificate. For the purposes of vital statistics, *abortion* includes fetuses up to four months of pregnancy; *miscarriage*, between four and six months; *premature births*, from six months (the age of viability), to fetal maturity. If the child breathes at all, it must not be recorded as a stillbirth.

Fundamental Factors.—The underlying factor of infant mortality is infancy itself—the period in which the flame flickers feeblest. Most infants die primarily from accidental and therefore preventable causes—the fundamental causes are the results of poverty, ignorance and neglect.

The chief specific causes that increase infant mortality are artificial feeding; hot weather; dirty, stale, and bacteria-laden milk; bad feeding; illegitimacy; lack of prenatal care; gainful occupations of mothers; midwifery; poor housing; lack of cleanliness; alcoholism, syphilis and other diseases; imperfect hygiene and sanitation. The causes, then, are multiple and exceedingly complex, and include social and economic factors.

Effect of City Life.—Infant mortality is greater in cities than in the country, not because of the better environment of country life and air, but because most of the important conditions which cause high infant mortality are concentrated in cities. Pearl¹¹ states that infant mortality rates exhibit a high degree of place variation in both rural and urban areas of the United States. Infant mortality rates are not determined by the size of the city or the density of population, but by the hygiene, sanitation and character of the

¹¹ *Am. J. Hyg.*, 1921, 1: 419.

population; thus, we have the following infantile death rates from the United States Census:

Area	1900	1915	1923
New York State	145	99	72
New York City	170	99	67
Nashua, New Hampshire	232	148	109
Lowell, Massachusetts	240	156	107
Fall River, Massachusetts	260	167	92
Charleston, South Carolina	323	182*	150
Winston-Salem, South Carolina	163†	140
Pittsburgh, Pennsylvania	162	110	98

* Data for 1919.

† Data for 1917.

Nashua, Lowell and Fall River are typical factory towns, but not large ones. A similar very high infantile death rate is seen in factory towns in England and elsewhere. This is due largely to the gainful employment of mothers in factories, and hence bottle-feeding and neglect of the babies. During the siege of Paris in 1871, while the general mortality doubled, the infant mortality fell 40 per cent; opportunities for outside work were shut off and women were compelled to stay home; so nursed their babies.¹²

Studies clearly indicate that high infant mortality coincides with low earnings, poor housing, the employment of the mother outside the home and large families.

Economic Causes.—There is a marked contrast between the death rate of children of the poor and those of the rich. Infant mortality is a class disease. In this case, money may purchase health and even life itself. Thus, Clay estimates that in England in the aristocratic families the mortality of the first year is 10 per cent; in the middle class, 21 per cent; in the laboring class, 32 per cent. Acute gastro-intestinal diseases are chiefly responsible for this. Halle states that of 170 infantile deaths from gastro-intestinal diseases investigated in Graz in 1903 and 1904, there were 161 among the poor, nine among the well-to-do, and none among the rich. Woodbury¹³ found a marked correlation between low earnings of father and high infant mortality, based on studies in Baltimore.

This relationship between earnings and mortality was found to be independent of type of feeding, of race and nationality, and of the factors involved in frequency of births. The influence of economic position is undoubtedly indirect. The study of the chain of causation connecting poverty with high infant mortality showed that the responsibility of low earnings extended over the excessive mortality associated with housing congestion and with the mother's employment both during pregnancy and during her child's first year of life. It showed also that a direct relation existed between lack of means and the kind of care available both for the mother during pregnancy and confinement and for the baby during his first year. The

¹² Brehmer, *Wehnschr. f. Säuglingsfürsorge*, 1907, 209.

¹³ *J. Am. Statistical Ass.*, 1924, 19: 137.

analysis suggests, therefore, that lack of care is another intermediate factor. Corresponding figures are obtained wherever this subject is studied.

A high infant mortality results in a sacrifice of the *unfortunate* as well as the *unfit*. Intelligence is a good preventive. Newton was a premature, posthumous baby, saved only by careful ministrations. Poverty and ignorance are, therefore, underlying factors of great importance in this problem. Infant mortality records are determined by the size of the family as well as the economic status.

Age Incidence.—The mortality is especially high during the first few weeks of life. Ten per cent of the deaths during the first year occur on the first day. Most of these (about 70 per cent) are due to prematurity and injury at birth. From 25 to 40 per cent of the total deaths during the first year occur during the first month of life. Some of these (about 10 per cent) are due to diarrheal diseases, but most of them are due to prematurity, congenital debility and malformation.

The highest infantile mortality is in the first days, weeks, and months of life. Although there has been a marked decline in total infant mortality, this improvement has almost wholly taken place between the ages of three and twelve months. The trend under three months has remained relatively stationery, hence, the rates for these periods show a proportionate increase. The causes of deaths during this early period is from the so-called congenital diseases. Therefore, further improvement in infant mortality depends largely upon prenatal and natal care. Perhaps two-thirds of this loss is preventable.

Medical Causes of Infant Mortality.—The death returns to the health officer usually cover only the last thing which happened to the child. The real causes which are back of this terminal disease do not appear on the records. Diarrhea, bronchitis and pneumonia are often end conditions which would not have occurred or would not have been fatal without some other condition, as malnutrition, neglect, ignorance, or marasmus, which existed weeks or months before the final few days of acute illness which closed the scene.

A study of 44,226 deaths under one year in the four largest American cities gives the following causes of infantile deaths:

<i>Causes of Death</i>	<i>Per Cent</i>
Acute gastro-intestinal disease.....	28.0]
Prematurity, congenital debility and marasmus.....	25.5 }
Acute respiratory diseases.....	18.5]
Acute infectious diseases.....	5.4
Tuberculosis (all forms).....	2.0
Syphilis	1.2
Malformations, injuries at birth and other conditions of the newborn	5.8
Convulsions	3.4
All others.....	10.2

From the above analysis, it is at once evident that 72 per cent of the total infant mortality is made up of the first three causes.

The relative importance of these causes varies with the age of the infant; thus, nearly 70 per cent of deaths occurring on the first day of life are due to group 1 of the following tabulation:

<i>Causes of Death</i>	<i>Per Cent</i>	
1. Prematurity, congenital defects, debility and accidents at birth.....	25	} 85
2. Diseases of nutrition.....	15	
3. Acute gastro-intestinal diseases.....	25	
4. Acute respiratory diseases.....	20	
5. Acute infectious diseases.....	3	
6. Tuberculosis	2	
7. Syphilis (direct effects).....	1	
8. Unclassified	9	
	<hr/> 100	

Sepsis in the newborn formerly carried off many babies. This has been largely reduced by aseptic methods in obstetrics. The toll is still large where these principles are not practiced. (See Tetanus Neonatorum, page 91.)

Vaccination has reduced deaths from smallpox, which used to be a children's disease. Improvement in the control of other communicable infections has also helped reduce infant mortality.

Gastro-intestinal Diseases.—Gastro-intestinal diseases comprise the largest single factor almost everywhere; in fact, the curve of diarrheal diseases largely controls the curve of infant mortality. These diseases embrace acute gastritis, gastro-enteritis, diarrheas, dysentery, infectious diarrheas, and cholera infantum—often termed summer complaints.

There are three underlying causes of gastro-intestinal diseases of the first year: (1) atmospheric heat; (2) methods of feeding; (3) infection; all of which are favored by city residence. Rickets and scurvy lead to gastro-intestinal disorders.

The withering effects of heat, especially when combined with humidity, are well known. Indoor air in crowded tenement districts may be hot and humid, while the temperature and humidity at the observation stations of the weather bureau indicate pleasant weather.

There is the closest possible connection between the frequency and fatality of diarrheal diseases and methods of feeding. The chances were about one to ten against the bottle-fed baby; now, about one to six. Eighty-five per cent of all infant deaths are bottle-fed babies; 90 per cent of infant deaths from diarrheal diseases are bottle fed. The figures are sufficiently impressive to emphasize the importance of breast feeding in prevention. This one measure alone would reduce infant mortality one-third.

Hope, of Liverpool, has shown that in 1,000 breast-fed infants under three months there were only twenty deaths from diarrheal diseases; while in 1,000 bottle-fed babies under three months there were three hundred deaths. Of 1,000 fatal cases of diarrheal diseases investigated by the New York Health Department in 1908, only ninety had previously been entirely breast-fed. Newsholme gives almost identical figures for England; namely, 10 per cent of deaths in breast-fed infants, and 90 per cent in bottle-fed infants.

It is not artificial feeding, *per se*, as is shown by the relatively fewer deaths in the bottle-fed babies among the well-to-do. Secondary factors are bad milk, unsuitable foods, improper methods of feeding, lack of maternal care, bad surroundings, especially heat, humidity, lack of cleanliness and overcrowding, which favor the spread of infection; in short, imperfect hygiene and sanitation.

Breast-feeding requires but little experience and may be very successfully done even by those with a very low grade of intelligence and among the poor; but artificial feeding is not successful, unless carried on with much intelligence and experience and at the same time with a certain amount of money to secure reliable materials, especially safe milk.

The method of feeding is especially important in prematurity, congenital debility, marasmus and inanition; also at the first indications of gastro-intestinal disturbance in a previously healthy child.

Avitaminosis is a factor. Scorbutic and rachitic conditions render children particularly susceptible to intestinal, respiratory and other infections.

Many of the diarrheal diseases of infants are true bacillary dysenteries, or infectious diarrheas of specific cause. They are transmitted in all the ways that typhoid fever and dysentery are transmitted. Sanitary isolation, the boiling of diapers and aseptic technic of food and clothing are essential to protect babies against these preventable infections.

Bronchitis and *pneumonia* are responsible for about 18 per cent of the total infant mortality. Overcrowding favors the spread of these infections, and common colds, influenza, streptococcal and pneumococcal infections are very apt to cause severe bronchitis or bronchopneumonia in infants. Scurvy and rickets predispose to bronchitis, pneumonia, and other infections. These conditions are often terminal and not at all the true cause of the baby's death.

All forms of *tuberculosis* constitute about 2 per cent of infantile deaths. This is doubtless an underestimate because of the difficulty in recognizing this disease in infancy. The care of the baby is often left to the sick father or mother while the other members of the family are at their work. Tuberculosis does not usually manifest itself early, but the infection contracted in infancy usually runs an acute fatal course. Childhood infection may remain latent and later in life break out into the clinical disease (see page 169).

Syphilis accounts for about 1.2 per cent of infantile deaths. These figures include only the direct effects of the spirochete. The prevention consists in prompt treatment of parents, etc. (see page 75).

Acute communicable diseases account for only 5.4 per cent of infantile deaths. Whooping-cough makes up about half, and measles and erysipelas most of the remainder, for the other diseases are relatively infrequent in the first year. There is an immunity during the first months of life to diphtheria, scarlet fever and measles.

Premature birth or feebleness at birth too great to support an independent existence are responsible for many early deaths. Alcohol and syphilis are prime factors favoring these conditions. This group includes congenital debility, marasmus and inanition. It is in this group that prenatal care and proper hospital provision for premature infants are especially helpful.

Prenatal care and especially a period of rest and good food before confinement will increase the weight, vigor and maturity of the baby.

Prevention.—The prevention of infant mortality consists in attacking the problem at its root, in concentrating upon the preventable causes, and focusing attention upon the mother. Attacking the problem at these points will give rewardful results. The preventable causes of infant mortality¹⁴ may be grouped as follows:

1. Those but little influenced by treatment:
 - Malformations
 - Extreme feebleness or prematurity (before the seventh month)
 - Certain accidents during birth
2. Those capable of considerable reduction, chiefly through proper hygiene, sanitary isolation and medical treatment:
 - Tuberculosis, syphilis
 - Acute respiratory diseases
 - Acute contagious diseases—whooping-cough, measles, and diphtheria
3. Those capable of a very great reduction through proper feeding and care:
 - Acute gastro-intestinal diseases
 - Marasmus and inanition
 - Prematurity, after seventh month

Poverty and ignorance require social justice and education. Other means of prevention are: prenatal care, hospitals for premature infants, milk depots, educational clinics, public health nursing, encouragement of breast feeding, competent and compulsory medical supervision of infancy, clean milk, pasteurization, escape from city heat, better housing, scattering of crowded tene-

¹⁴ Much of this material is taken from the excellent articles by Dr. L. Emmett Holt on "Infant Mortality and Its Reduction, Especially in New York City," *Journal of the American Medical Association*, February 26, 1910; and "Infant Mortality, Ancient and Modern," *Archives of Pediatrics*, 30:12, December, 1913. Both these publications contain selective bibliographies. Consult also the publications of the *American Association for the Study and Prevention of Infant Mortality*, the *Archives of Pediatrics*, etc.

ments, cleanliness, better artificial feeding when necessary, sanitary isolation from dysentery and other intestinal diseases, also from influenza, common colds, pneumonia and other respiratory infections. Milk depots, little mothers' leagues, social service workers, public health nurses, and improvement in medical and obstetrical practice are part of the program. Sunshine, fresh air and other hygienic measures should be stressed. Foundlings and orphans should be raised in homes rather than in institutions.

Considerations of the causes of infant mortality inevitably lead to the question of the care mothers are receiving during and after childbirth. In a few places the possibility of reducing this death rate about one-half through prenatal supervision in connection with prenatal clinics or maternity centers has been demonstrated.

The Sheppard-Towner Act.—The Sheppard-Towner Act became a law November 23, 1921, and provided subventions to the states for the promotion of the welfare and hygiene of maternity and infancy. It authorized an appropriation of \$1,240,000 for a five-year period to be expended by the Children's Bureau of the Department of Labor. The local administration in the states is usually in the child hygiene or child welfare division of the state agency of health. An extension for a further two-year period is pending in Congress.

A few of the states, Maine, Massachusetts, Connecticut, Illinois and Kansas, have not accepted the subsidy.

PUBLIC HEALTH NURSING

Public health nursing is one of the most important links in the chain of efficient public health administration. The duties of the public health nurse are vague and varied. They include instruction and social work as well as nursing; in fact, nursing is only part of the duties of a public health nurse. The office of the public health nurse is part of an organized community effort to prevent sickness, maintain efficiency, prolong life, relieve suffering and promote individual and public health. The nursing itself must be preventive as well as curative or palliative.

The public health nursing movement is recent but has grown steadily. The first district nursing association was established in Liverpool in 1859; the first in the United States was the Instructive District Nursing Association in Boston in 1886. The movement has now received recognition, is an established and important part of public health work, and is making rapid progress.

Specialists are required for certain problems, such as the venereal diseases, tuberculosis, infant and child welfare, school nursing; also mental hygiene, industrial nursing, medical social service, and dental hygiene. Each of these special fields requires special qualifications which can be had only through training and experience. In addition, communities require public health nurses prepared for general service. A model plan of organization includes both the general nurse and specialists.

The nursing service necessary for a city of 100,000 inhabitants:

For communicable disease control.....	1
For tuberculosis control.....	4
For venereal disease control.....	2
For infant welfare work.....	15
For school health work.....	8
	—
TOTAL	30

The need for public health nurses is a large outstanding problem. We should have approximately 50,000 public health nurses to serve the population of the United States, instead of the 11,000 now in the field. Special schools are required.

About one public health nurse is needed for each 3,000 population; in addition one tuberculosis nurse for each 20,000, and one school nurse for each 1,500 children. The public health nurse should be in close touch with the board of health and many other public and private agencies in the community. She should not be required to furnish material relief.

The history of nursing includes many noble figures to contrast with Sairey Gamp. The names of Clara Barton, Jane Delano, Frederica Fleidner, Linda Rickards, Florence Nightingale and Edith Cavell are enshrined.

REFERENCES

- BRAINARD, ANNIE M. *Organization of Public Health Nursing*, Macmillan Co., 1919.
 GARDNER, MARY S. *Public Health Nursing*, Macmillan Co., 1924.
 LAMOTTE, ELLEN. *Tuberculosis Nursing*, Macmillan Co., 1915.
 NUTTING, M., and DOCK, L. L. *History of Nursing*, Macmillan Co., 1912.
 STRUTHERS, LENA. *Handbook on School Nursing*, Macmillan Co., 1917.
 WRIGHT, FLORENCE S. *School Nursing*, Macmillan Co., 1919.

NUISANCES

A nuisance may be defined as "the use of one's property in such a way as to injure the rights of others, and to inflict damages." Popularly a nuisance is an annoyance. Statutory definitions are usually more explicit and include nuisances not directly related to public health. A comprehensive statutory definition is: "Whatever is dangerous to human life, and whatever renders soil, air, water, or food impure or unwholesome, are declared to be nuisances, and every person, either owner, agent, or occupant, having aided in creating or contributing to the same, or who may suffer to continue or retain any of them shall be deemed guilty of a misdemeanor."¹⁵

¹⁵ Utah, Chapter 45 of 1889. Sec. 1.

The following are considered nuisances in different states and cities: Filth, such as garbage, ashes, and slops, either on private property, or on public highways; cesspools, privy vaults, sink drains, dumps, and dirty yards; low, wet, and soggy lands; defective plumbing and draining; faulty cellars; overcrowding of tenements and lodging houses, or dwellings unfit for habitation; excavations; weeds; flowers with offensive odors; foul closets on railroad coaches; dirty street cars; use of salt on streets in snowy weather; disturbing noises; spitting in public places; keeping of horses and cattle in city limits; manure; hogs; hog-pens, stables and barns; fowls; dead animals; filthy shores; stagnant water and marshes; offensive businesses and trades; places where liquor is sold illegally; and offenses against decency. While some of these conditions may favor the spread of the communicable diseases, yet most nuisances are not serious health problems.

The phrase "source of filth or cause of sickness" used in the statutes of no less than fourteen states is copied verbatim from a law enacted in Massachusetts in 1797. At that time miasmatic vapors and the inhalations from decomposing organic matter were believed to be the principal causes of the contagious diseases. The Supreme Court of Massachusetts, however, has ruled that "in order to amount to a nuisance it is not necessary that the corruption of the atmosphere should be such as to be dangerous to health; it is sufficient that the effluvia are offensive to the senses and render habitations uncomfortable."

By far the greatest number of all the complaints reaching the health authorities deal with real or supposed nuisances. There seems to be a widespread belief that the chief function of the health officer is the abatement of nuisances. Formerly the health officer was a general scavenger and his main duties consisted in looking after nuisances. Nuisances often clog the health office and crowd out more important sanitary and hygienic matters. In most complaints the question at issue is whether the nuisance exists or not—a question of fact which could be decided just as well by the police courts as by a board of health or health officer. However, the abatement of nuisances is usually assigned to the health authorities by statutory enactment.

In a few cities, especially those with liberal charters, the ordinances covering nuisances are so definite and explicit that an inspector may determine a nuisance and issue an order for its abatement. In cases where condemnation of property of considerable value is involved, such, for example, as when an offensive trade is alleged to be a nuisance, it is usually necessary to prove the case in court before the nuisance can be abated. In court, substantial injury must be shown and the health officer should be sure he has the facts as to the nuisance before he appears in court. He will be required by the court to establish the source, frequency, and nature of the odors, or whatever is alleged to be the nuisance. It is exceedingly difficult to establish the fact that many nuisances are dangerous to the health of the community and the cause of sickness.

In general two methods are followed for the control of nuisances: (1) pre-

vention, (2) abatement. The first is the wisest and aims to regulate and control the different conditions likely to cause a nuisance or even to prohibit them. The second merely provides legal steps for their abatement. There are at least four ways in law of dealing with nuisances: (1) by criminal action; (2) by injunction; (3) by damages (private suit); (4) by abatement under statutory powers.

DRUG ADDICTION

The chief habit-forming drugs are alcohol; opium and its alkaloids, especially morphin, codein, and heroin; and cocain. It has only recently been realized that drug addiction has public health aspects in addition to its sociologic problem.

It is obviously impossible to give the exact count of persons addicted to narcotics. Kolb and Du Mez¹⁶ adduce evidence which seems to show that in the United States there is a maximum (in 1924) of 150,000 addicts taking daily 6 grains or more of morphin sulphate or cocain hydrochlorid. This is very different from the estimate of the Special Committee¹⁷ who studied the question and who gave it as their opinion that "the total number of addicts in this country probably exceeds one million at the present time" (1919). There is no way of determining the number of *users* of habit-forming drugs, as contrasted with *addicts* who take 6 grains or more a day. The Special Committee considered as addicts all habitual users of habit-forming drugs without medical advice, and irrespective of amount. While the numbers are probably large, the situation seems to be improving. Thus, Kolb and Du Mez place the number in 1900 at 264,000 and state that the number of addicts has steadily decreased since then. The greater number of addicts in prison at present as compared with former years is due to the rigid enforcement of recently enacted laws and not to an increased prevalence of addiction. While physicians have been credited with being responsible for the creation of many addicts in the past, but few cases of recent addiction can be so attributed. The proportion of the delinquent type of addict is gradually increasing. This apparently is not due to an increase in the number of this type, but to a gradual elimination of the normal types.

Commissioner Brown of Tennessee fixed 5,000 as the probable number in that state in 1915, and estimated 269,000 as the possible number of addicts in the United States—0.27 per cent of the population. These figures are based on registration by state law.

The Treasury Department survey in June, 1919, estimated 237,655 addicts under treatment for the entire country. They had evidence which suggested that the total number probably exceeded one million. The Pennsylvania survey found that in 1922 there were not more than 20,000 habitual drug

¹⁶ *U. S. Pub. Health Rep.*, Reprint No. 924, May 25, 1924.

¹⁷ *Traffic in Narcotic Drugs*, Washington, 1919. Report of Special Committee of Investigation appointed March 25, 1918, by the Secretary of the Treasury, June, 1919.

users in the state; on this basis there would be approximately 242,000 in the United States.

There were 3,284 rejections for this cause out of about 3,500,000 men which constituted the mobilization of man power following our entrance into the World War. The army rate cannot be applied to the entire country, for it represents only males of a specific age period.

The Bureau of Internal Revenue made a study of the records of thirty-three clinics in various parts of the country. The table shows that there were 4,123 addicts in thirty-four cities, having a total population of 4,182,952, or 0.98 per 1,000. At this rate, there would have been 104,300 in the United States in 1919. The New York City clinic, not in the foregoing during the period April 10, 1919, to January 16, 1920, registered 7,464 addicts. This would make about 140,600 for the entire country.

All the surveys have necessarily been fragmentary and incomplete. We do not know the number of persons addicted to habit-forming drugs in the United States. We know enough, however, to realize that we face a serious problem.

Anyone repeatedly taking a narcotic drug for thirty days is in grave danger of becoming a slave to the drug. The reasons for beginning the vicious practice are innumerable. In most cases, however, once learned, there is an underlying defect or weakness, either acquired or inherited, that chains the victim to the vice. The Harrison Law meets the situation only in part.

The question is being studied by Charles E. Terry and Mildred Pellens for the Committee on Drug Addictions, 370 Seventh Avenue, New York City.

ALCOHOL

Ethyl alcohol (C_2H_5OH) has been used since the dawn of history. Until recently, we had little precise knowledge concerning the effects of alcohol. No one doubts that alcohol is harmful when used in amounts sufficient to produce its full physiological effects. In sufficient concentration, it is a poison to all living matter, both animal and vegetable. The question in dispute is upon the effects of "small" amounts. Alcohol is a solvent for many substances, is locally irritating, is quite volatile, and has other well-defined physical and chemical properties, but perhaps the most striking characteristic is that "it is equally inflammable whether one touches a match to it or writes about it."

Local Irritating Action.—Applied to the skin in sufficient concentration (60 to 90 per cent), alcohol produces redness, itching, and a feeling of heat like other volatile and irritating substances. Upon wounds a concentrated solution causes a precipitation of the proteins and acts first as an astringent and then as a caustic. The effects of alcohol on mucous membranes are similar to those on wounds.

Alcohol as a Food.—Whether alcohol can be regarded as a food or not, depends upon the definition of a food. Alcohol is not a tissue builder, but

within limits it is burnt in the body and thus furnishes heat and energy. In this sense, then, alcohol is a food. Thus, one gram of alcohol furnishes seven calories of heat when burned. The human body is not able to burn more than about two ounces of alcohol a day. If more than this amount is taken, it promotes the storage of fat. The excess alcohol is slowly eliminated unchanged in the urine and by the skin and lungs.

Alcohol may be compared with sugar and starches as a food—both are oxidized in the body and furnish heat and energy, but there is this difference: sugar and starch help to repair waste, which alcohol cannot do.

Effect upon Digestion.—Alcohol in the mouth causes a very appreciable secretion of saliva, presumably by reflex action. It is not unlikely that the taste has some influence on this result; in those who enjoy the taste of alcohol, it induces a more rapid secretion and an immediate digestion; while in those to whom it is disagreeable, the effect is less marked.

Small amounts of wines and liquors used as a condiment to flavor sauces and desserts are pleasing to the taste of many persons, and thus used probably stimulate the appetite, and through the psychic effect of savory morsels promote the secretion of digestion juices. The augmentation of the activity of the digestive ferments caused by alcohol is so slight in any case that it does not seem likely that it plays any important rôle in digestion.

All are agreed as to the deleterious action of any but moderate doses of alcohol on digestion. Large quantities irritate the stomach and may lead to profuse secretion of mucus, nausea, and vomiting—irritative gastritis.

Action on the Nervous System.—The action of alcohol on the nerve centers seems to differ in individuals, but this is solely a difference of manifestation, for the physiological action is essentially the same in all persons. In small quantities, it produces a feeling of well-being and goodfellowship, along with increased confidence in physical power and mental ability. This confidence, however, is due to removal of repression and inhibition. Larger quantities are followed by laughter, loquacity, gesticulation, and other indications of animal excitement. The face becomes flushed and heated, the eyes brighter and livelier, and the pulse quickens. Even at this stage, self-control is partially lost, and the will power is weakened. The speech may be brilliant, but often betrays the speaker. The movements are more lively, but they are often undignified. The loss of self-control is often indicated further by furious outbursts of anger or unreasonableness, or by the indulgence in maudlin sentimentality and sensual fancies. The sense of responsibility and the power of discrimination between the trivial and the important are lost, and the individual has no regard for the feelings of others or the ordinary conventions of life. If more alcohol is imbibed, the movements become uncertain, the speech becomes difficult and stammering, the walk becomes a stagger, and a torpid slumber follows. Often nausea and vomiting set in, doubtless due to the direct irritating action on the gastric mucosa. On recovery from slumber, a very great depression is generally suffered, together with nausea and vomiting and want of appetite, which may last several days,

and is associated with all the symptoms of acute gastric catarrh. Very large quantities of alcohol produce total unconsciousness, resembling chloroform anesthesia. If unconsciousness lasts longer than ten to twelve hours, recovery seldom takes place.

On the lower part of the central nervous system, alcohol acts as a distinct depressant, for the coördination of movements suffers at an early stage. In the spinal cord, there is a depression of the reflex, which passes into complete paralysis. The medulla oblongata is the last part of the central nervous system to be acted on by alcohol. Dodge and Benedict find that alcohol depresses the lower centers most and the highest centers least. It retards the reaction time. In one sense, alcoholic depression may be regarded as a conservation process.

Alcohol is a nerve poison; hence, alcoholic neuritis is well known to the clinician. The repeated use of excessive amounts of alcohol causes brain injury, as Korsakoff's disease, alcoholic hallucinosis, delirium tremens, and other forms of alcoholic insanity.

Alcohol psychoses accounted for about 4.6 per cent of all first admissions to hospitals for the insane in the United States for the years 1917 to 1922. They occur about three times as frequently in men as in women, and in general the subjects come more frequently from the cities than from rural districts.

Alcohol as a Stimulant.—Alcohol acts like ether and chloroform and other narcotic drugs. There is a preliminary stage of excitement, mental confusion, and often excessive and incoördinate activity.

Binz and his pupils claim that alcohol first stimulates and then depresses the nervous cells, but the preponderance of evidence is clearly in favor of the views of Schmiedeberg, Bunge and their followers, that it depresses the central nervous system from the beginning. The apparent excitement is not due to true stimulation of the motor areas, but is the result of these areas being freed from control by the weakening of the highest powers of the brain—the will and self-restraint. Even the smallest quantities of alcohol tend to lessen important activities of the brain, for the drug acts first upon those qualities which have been built up through education and experience.

One of the most deceptive of experiences is the false sense of stimulation which alcohol gives. By depressing the higher cerebral centers, it releases the lower mental processes. Hence, its first effect is to cloud the judgment and dull the finer feelings. It takes off the brake of restraint, loosens the tongue, and sets free the lower animal passions. It is this blunting of the higher and finer mental powers which causes people to say things and do deeds under the influence of alcohol that would shock their sensibilities otherwise.

It is difficult to measure the harm done by this effect of inhibiting the nobler mental functions, but it must be great. Alcohol does not relieve fatigue, but makes one unconscious of fatigue.

Effect on Efficiency.—A large number of tests have demonstrated that the apparent quickening of the mind and body under alcohol are false sen-

sations. The performance of difficult operations involving muscular co-ordination and judgment are slower and less accurate under alcohol than normally. It is difficult, on account of the feeling of confidence that comes from alcohol, to convince a typist, a sharpshooter, or typesetter of this fact until shown the records. Even amounts of alcohol so small that the subject and observer are not aware of any abnormality of appearance or behavior interfere with efficiency.

It has been found that regiments not supplied with alcohol march farther and are in better condition at the end of the day than others to which it has been given. The experiments of Durig lead to the same results, the total amount of work being smaller under alcohol, and the expenditure of energy greater. Forms of work requiring intelligence are performed less correctly with alcohol than without it; thus, typesetters can do more work and make fewer mistakes when they abstain from its use. Even Binz, who claims that alcohol is a primary stimulant, admits that this action is transient, and is followed by depression, so that the total amount of work may thus be reduced. Kraepelin found in a series of careful measurements of the simpler processes that the responsive powers were weakened by very small quantities of alcohol, while the motor functions seemed to be facilitated by small, and retarded by large quantities. For example, a person under even a small dose of alcohol makes more errors than usual in adding a row of figures, or in reading a series of unconnected syllables. Of special importance is the fact that the subject of the experiment is quite unaware of the inferiority of his work, and believes it to be unusually good. Kraepelin has shown that even about a pint of beer will lower intellectual power, impair memory, and retard the simpler mental processes. Kraepelin's latest investigations tend to show that this effect of alcohol lasts much longer than is generally recognized, the mental equilibrium being reinstated only after twelve to twenty-four hours after even moderate indulgence. In fact, alcohol may be found in the blood twenty-four hours after its ingestion.

We have the authority of Connie Mack that alcohol spoils a good baseball player. He said in 1910, a year that the Philadelphia Athletics won the world's championship, that fifteen of his twenty-five players did not even know the taste of alcohol.

Pulse, Reflex, and Temperature.—The *pulse* is accelerated during the stage of excitement, but this is due to increased muscular effort, and not to any direct stimulating action on the heart. There is at first a slight rise in blood-pressure in some cases, but large quantities affect the heart in the same way as ether and chloroform, weakening the auricular and ventricular beat, and inducing dilatation and slowing of both chambers.

The flushed, perspiring face of the alcoholic is familiar. This indicates dilatation of the skin vessels, which is sometimes accompanied by a slight contraction of the internal organs. This effect of alcohol causes a sense of warmth and comfort, which underlies the popular belief that it is helpful to take alcohol when braving exposure to cold and damp, during a chill, etc.

Here again our sensations are misleading guides, for the temperature of the body may fall one degree centigrade, while the skin is flushed with a sense of warmth. This is due to excessive heat loss through conduction and radiation, and also to the fact that the heat regulating mechanism is rendered less sensitive by alcohol. Hence, alcoholics (when inactive) may freeze to death when the temperature is scarcely at the freezing point.

The changes in the respiration induced by alcohol are too small and too inconsistent to be of any special importance.

Alcohol and Venereal Disease.—Alcohol is the bedfellow of syphilis and gonorrhea. It is intricately interwoven into the warp and woof of sex hygiene. The story of many cases of sexual immorality begins with the influence of drink. Alcohol is generally accredited with increasing sexual desire, although we have the authority of Shakespeare that it interferes with the consummation of sexual intercourse. The unquestioned sexual excitement is not due to stimulation of the generative organs, but to the loss of self-control and the anesthetic action of alcohol upon the higher centers of the brain.

Resistance.—It has long been known clinically that persons addicted to the use of alcohol show less resistance to certain diseases and to operations accompanied by shock than more temperate individuals. In very intemperate persons the prognosis must be guarded in an attack which would ordinarily be accompanied with little danger. "Drunkards have a very slim chance of recovery when attacked by pneumonia" (Osler).

When animals are subjected to treatment with alcohol, and then inoculated with pathogenic microorganisms or their toxins, the results invariably show a greater susceptibility to infection and a greater mortality than in control animals. The reason for this reduced resistance is not clear. It may result from inactivity of the leukocytes (Rubin), or a reduction in the hemolytic complement (Abbott and Bergey).

The tolerance to alcohol is not so great as that acquired for morphin and nicotin. Antibodies do not develop in the blood.

Life insurance figures plainly show that even the moderate use of alcohol tends to shorten life.

Accidents.—Accidents often happen under the influence of alcohol. Many automobile wrecks and railroad collisions can be traced to the recklessness

<i>Accidents</i>	<i>Per Cent</i>
Railroad accidents.....	7
Street cars.....	8
Automobiles	10
Vehicles and horses.....	8
Heat and sunstroke.....	43
Machinery	7
Mines and quarries.....	8
Drowning	13
Gunshot	10

of the drinker. Figures show that industrial accidents are more frequent in those who drink than in abstainers. It is regarded as significant that three such accidents occur on Monday to two on other days. According to three insurance companies, the above percentages of accidents are attributed directly or indirectly to alcohol.

Poverty.—There would be much less poverty, crime, misery, and distress in the world without alcohol. It is not the sole cause of these ills, but a potent factor. The social and economic aspects of the alcohol question are plain to every social service worker. Of 352 able-bodied men who failed to support their families in Boston, 65 per cent (243) were drunkards.¹⁸ About 25 per cent of cases that come to charitable organizations, and about 37 per cent of poverty found in almshouses give an alcoholic history.

Three-fourths of the cases of children cared for by the Chicago Juvenile Protection Association in 1911 grew out of alcoholism in the parent or guardian.

Crime.—Crime is often committed while under the influence of alcohol. A large percentage of criminals have an alcoholic history. The relation between alcohol and crime is not disputed, but it is difficult to express its influence in precise percentages. Drink was said to be the sole cause of crime in 16.8 per cent of 13,402 convicts in twelve states of the United States studied by the Committee of Fifty. Further, drink contributed to 49 per cent of the crimes against property; 51 per cent of the crimes against person; and 47 per cent of all other crimes.

The first action of the police in a local uprising is to close the bars, and one of the first measures taken in the stress of the World War was to check the use of alcohol. Alcohol is a handicap for a nation in peace or war; it is also a handicap for an individual in the struggle for existence.

Heredity.—The relation of alcohol to heredity is difficult to appraise. Experiments upon lower animals give various results, but indicate lowered vitality in the offspring. The craving for drink is not transmitted, but mental deficiency with weakened will power that may express itself in intemperance is inherited. Many alcoholics are also defectives and transmit the mental defect. This subject is fully discussed on pages 552 to 566.

Alcohol in Medicine.—Occasionally alcohol is useful in the treatment of diabetes, where it may partly replace fats and carbohydrates when these are harmful. It is no longer considered good practice to use it as a stimulant in fevers, septic, and debilitated states. At the Massachusetts General Hospital, the amount of alcohol used for the patients fell 70 per cent from 1897 to 1906 and has declined still more since then.

It may be a serious mistake in first aid to give alcohol to persons who have fainted, or who have been injured, or who have lost consciousness.

Uses of Alcohol.—Because alcohol produces a fictitious sense of well-being, disguises fatigue, and smothers worry, it has been widely used by elderly

¹⁸ Report of the Boston Associated Charities, 1910.

persons and certain individuals, particularly convalescents and those upon whom life bears heavily. Alcohol is used in such cases for its physiological effect of partially benumbing the sensibilities and creating an artificial sense of well-being. In such situations, the use of alcohol often seems desirable. In some cases the depressing action of alcohol may be a conservative process, although the magnitude of the harm from its widespread abuse outweighs this theoretical advantage.

Summary.—The student of preventive medicine still regards the alcohol question as a public health problem. Alcohol is a habit-forming drug; it lowers resistance and shortens life, impairs efficiency, promotes poverty, increases crime, favors accidents, excites passion and diminishes self-control; it leads to immorality and tempts venereal infections. Alcohol increases economic waste and retards social progress. It is a narcotic rather than a stimulant. Its nutritional value is strictly limited. Its habitual use as an aid to work is physiologically unsound. Its local irritating action and its toxic effects upon nerve tissue account for a certain amount of harm; but the greatest harm perhaps results from the fact that alcohol, even in small amounts, clouds judgment, depresses will power, and takes the check off self-restraint. In short, it stupefies the highest and noblest functions of the mind.

REFERENCES

- An enormous number of titles is included in the available bibliographies, notably those of Abderhalden¹⁹ and Viazemsky.²⁰
- Psychological Effects of Alcohol.*—*An Experimental Investigation of the Effects of Moderate Doses of Ethyl Alcohol on a Related Group of Neuro-Muscular Processes in Man*, Raymond Dodge and F. G. Benedict. Pub. by the Carnegie Institution of Washington, 1915.
- Advisory Committee of the Central Control Board. *Alcohol: Its Action on the Human Organism*, Longmans, Green & Co., New York, 1918.

SANITARY SURVEYS

Sanitary surveys are designed to determine the general sanitary and hygienic conditions of a community. Surveys are often made to discover one or more special factors; thus we have tuberculosis surveys, school surveys, privy surveys, milk surveys, watershed surveys, housing surveys, etc. Survey means "to look," and the conclusions must be based on facts obtained by observation.

The following is an outline of the sanitary survey used by my students in the School of Public Health and also in the Harvard Medical School and intended as an educational discipline.²¹

¹⁹ Abderhalden, *Bibliographie der gesamten wissenschaftlichen Literatur über den Alkohol und den Alkoholismus*, Berlin and Vienna, 1904.

²⁰ Viazemsky, *A Bibliography on the Question of Alcoholism*, Moscow, 1909, Part I (Russian). The Russian original, together with an English translation made by H. A. Norman and H. B. Dine, are both on file at the Nutrition Laboratory.

²¹ See also *The Sanitary Survey*, Methods and Problems of Medical Education, Second Series, Rockefeller Foundation, 1924, and *Appraisal Form*, Committee on Administrative Practice, American Public Health Association.

SANITARY SURVEY OF A CITY OR TOWN

Each student is required to make a sanitary survey of a city or town, based on the following outline, and submit a written report of the same. The report should consist of (a) *data*; (b) *interpretation* of the facts, and (c) *criticisms* and *recommendations*.

Introduction.—General description of the town including: (a) History. (b) Geographical position. (c) Topography. (d) Geology. (e) Climate. (f) Population (number and constitution). (g) Organization of the board of health, budget. (h) Other information of sanitary significance about the town.

Water.—(a) The watershed—sources of pollution, methods of collection, storage, purification. (b) An analysis of the water and its interpretation. (c) Public or private wells. (d) Examine a sample of the water in the laboratory.

Sewage.—(a) System of disposal, purification or treatment. (b) Efficiency. (c) Relation to health of this and other towns. (d) Criticism of system.

Garbage, Refuse, Ashes.—(a) Method of collection. (b) Disposal. (c) Relation to health. (d) Criticism of methods.

Vital Statistics.—(a) Death rate. (b) Infant mortality. (c) Specific rates for: (1) typhoid; (2) tuberculosis; (3) measles; (4) scarlet fever. (d) Submit samples of blanks used by the department of health, especially those for deaths, births, marriages, and notifiable diseases. Fill out a death certificate and a birth certificate. State opinion as to thoroughness of reporting morbidity, mortality, and other vital statistics.

Milk.—(a) Report on the sanitary conditions of one farm and one city dairy, using score cards. (b) Amount of milk "certified." If possible, visit and report on farm producing it. (c) Examine a sample of the milk in the laboratory. Interpret result.

Sanitary Nuisances.—(a) Sources of odors. (b) Dust—causes and method of prevention. (c) Rubbish and general cleanliness. Empty lots. Dumps. (d) Flies and mosquitoes. (e) Rats and vermin. (f) Stables and manure. (g) Breeding places of mosquitoes. (h) Smoke. (i) Unnecessary noises. (j) Piggeries, etc. (k) Legal definition of "nuisance" and method of abatement.

Industrial Hygiene.—Report upon one industry based upon a visit to a factory or workshop.

Housing.—(a) Sanitary condition of one tenement. (b) Ventilation of one large building.

Infectious Diseases.—(a) Give a list of the diseases notification of which is required by the board of health. (b) Quarantine regulations. (c) Methods of disinfection and fumigation. (d) What measures are taken to prevent the spread of tuberculosis? (e) Should some other disease be prevalent what

measures are taken to control it? (f) Venereal diseases—reporting and control.

Schools.—(a) Visit and report on one school—ventilation, lighting, temperature, playgrounds, etc. (b) Medical inspection of school children. Dental service. (c) Diseases for which children are excluded from school.

Miscellaneous.—(a) Markets. (b) Provision stores and soda fountains. (c) Slaughterhouses and meat inspection. (d) Cold storage plants. (e) Kitchens of hotels and restaurants. (f) Wharves. (g) Barber shops. (h) Distribution of educational and other pamphlets. (i) Other activities of the board of health, as maintenance of diagnostic laboratory, meat inspection, etc. (j) District nursing and social service. (k) Charitable institutions or organizations of importance to public health. (l) City planning. (m) Food and drug administration.

General Summary of (a) Conditions found. (b) Criticisms. (c) Recommendations.

MANAGEMENT OF AN EPIDEMIC CAMPAIGN

Good public health administration, whether in an epidemic emergency or during ordinary times, requires a knowledge of the place and its people; hence, the usefulness of the sanitary survey. It is fundamental to know the population and its constitution, its epidemiology and medical history, its water supply, waste disposal, health administration and other factors bearing directly and indirectly on the problem (see the Sanitary Survey).

Knowledge of the Disease.—The first essential for success in the suppression of an epidemic is a knowledge of the epidemiology of the disease. The most important single fact from a practical standpoint is a knowledge of the mode of transfer of the infection. Yellow fever campaigns were crowned with success without knowledge of the cause of the disease because we knew it was transmitted through the bite of a mosquito. We know the cause of cerebrospinal meningitis, but there are still uncertain factors concerning its mode of transmission, and, therefore, our efforts against this disease have been unavailing. The established fact that hookworm disease results from soil pollution and is transmitted by the larvæ through the skin is of vital importance in the control of this disease. Without this knowledge more than 99 per cent of our efforts to repress hookworm disease would be wasted. When typhoid fever was regarded as a water-borne infection only partial success was achieved because contacts, milk, flies, carriers, and other modes of transference of the typhoid bacillus were disregarded.

In case the disease has an intermediate host or the virus is transferred by an insect or other animal, a knowledge of the biology of the animal in question is of prime importance. For example, the habits and habitat of the yellow fever mosquito are quite different from those of the malarial mosquito. A campaign against the rat and flea without an acquaintance with their breeding and feeding places and the best means available to repress or suppress

such vermin would be unsuccessful. The same is true in our campaign against tuberculosis with reference to cattle and man; in rabies with reference to dogs and other mammals; in sleeping-sickness with reference to the tsetse fly; in Texas fever with reference to the tick; Malta fever with reference to the goat; relapsing fever to the bedbug, and typhus fever with reference to the louse, etc.

Authority.—Proper authority is necessary in order to enforce the necessary measures. This authority may come from the municipality, the state, or the federal government. In localized outbreaks, municipal authority is sometimes sufficient. More frequently the wider authority of the state is desirable. In our country it is a recognized principle of government that the enforcement of health laws and regulations belong to the *police powers* of the *individual states*. In the United States, police power originates in the state; it can, however, be delegated. Police power deals with health, safety, and morals.²² In most instances the interstate powers of the federal government are essential, especially as interstate problems are almost always involved in epidemic outbreaks. The federal authority is limited in health matters by the constitution. It therefore may not act within a state unless invited to do so by the duly constituted authorities of the state. To send government health officers into a state against the will of the state corresponds to the sending of the regular army into a state to enforce measures against the will of the governor of that state. Such extreme measures are, therefore, only taken in times of emergency. Occasionally a state, refusing to take necessary action and protect the other states, is quarantined. Thus, when California refused officially to recognize the existence of plague in 1899, the federal government quarantined the entire state. On account of our dual form of government it is important that the federal government, the state, and the local authorities coöperate in a friendly spirit. Epidemic diseases recognize no geographical boundary, and energetic and coöperative action is always called for to suppress an outbreak.

It is the common experience of those who have to deal with epidemics that there is usually insufficient authority in law to provide for an emergency. It is, therefore, often necessary to take the bit in the teeth and adopt arbitrary measures which usually have the support of the community. Advantages may be taken of an epidemic to obtain laws to improve the health organization or the powers of the health officer. In this way an epidemic serves a useful purpose in arousing action.

In the conduct of an epidemic it is very important that all the authority should center in one person. To conduct an epidemic with a board of health or a health committee or a commission of any kind invites failure. It would be just as foolish to have a board of generals to fight a battle. Those who have been through many epidemics realize that it is no figure of speech to compare an epidemic campaign to a battle. It is a fight carried on at high tension, and

²² H. B. Hemenway, *Legal Principles of Public Health Administration*, Flood, Chicago, 1914. J. A. Tobey, *Public Health Law*, Williams and Wilkins, Baltimore, 1926.

although the foe is invisible, it is war in every sense of the word. The casualties often outnumber the bloodiest conflicts.

Ways and Means.—It is impossible to carry on a successful campaign against an epidemic without material resources. An epidemic campaign is expensive and success depends upon generous support. In most of the campaigns against yellow fever, plague, and cholera that have been waged in this country the expense has been borne in part by the federal government, in part by the municipality or state, and in part by private subscriptions. The government has an epidemic fund appropriated by Congress and which is usually kept at about a million dollars. This fund is available for plague, yellow fever and cholera, or other diseases specifically stated in the appropriation bill.

Organization.—Headquarters should be organized in a convenient part of the city or the infected area, and such quarters should have all the modern office equipment and transportation facilities necessary for the quick dispatch of business. The city is then divided into sanitary districts. These may correspond to the political wards or the police districts and a subordinate is placed in charge of the work in each district. These districts are known as sanitary divisions, and the officer in charge of each division must establish headquarters for the work of that division. The actual work is done from division headquarters, under the direction of the district chief.

It is also necessary to establish a laboratory in case laboratory diagnosis is necessary for the recognition of cases or carriers, and emergency hospitals and detention barracks must be provided. Few cities have sufficient hospital facilities to meet a sudden emergency. Temporary arrangements must therefore be made. A modern school building, hotel or armory makes a very good hospital and may be equipped for the reception of patients at short notice. Camp hospitals in suitable climates may be used. Various squads must now be organized to carry on the particular work at hand. In the case of yellow fever these will be mosquito brigades; in the case of plague, rat brigades and disinfectors, and in the case of smallpox, vaccinators, etc.

It is frequently desirable, in fact often necessary, to make a house-to-house inspection throughout the infected district in order to collect certain data, to determine whether cases are being reported or hidden, and to carry out special measures. These house-to-house canvasses are under the immediate direction of the officer in charge of the sanitary district and should be repeated as often as the occasion may demand.

It is essential that all cases or suspected cases of the disease be promptly reported, for a case of communicable disease known is a case neutralized. It is the missed cases and the hidden cases that are particularly dangerous.

Education.—A campaign of education should be carried on at the same time that the disease is being attacked. The people are keenly alive and hungry for information. Well-worded articles in the newspapers, circulars, pamphlets, lectures, demonstrations, and the other usual methods are available. The education of the community is important in order to obtain co-

operation, for it is a handicap to fight an epidemic without the active support of the people. While the first duty of the officer in charge is to allay panic and calm the unreasonable fears of the stricken community, the opposite extreme must be avoided. A healthy fear of the disease is one of the best instruments in the armamentarium of the sanitarian. It is almost hopeless to make progress against disease where the people are indifferent and supinely accept the conditions.

QUARANTINE AND ISOLATION

Quarantine.—The word “quarantine” is derived from the Italian word “quaranta,” meaning forty. Its present-day meaning dates from the middle ages when Venice and other Hanseatic cities detained arriving ships with cases of pestilence aboard for a period of forty days. This was the first systematic application of maritime quarantine, although from the earliest times lepers were segregated as unclean. To-day we have many kinds of quarantine: maritime quarantine, interstate quarantine, house quarantine, cattle quarantine, yellow fever quarantine, “shotgun” quarantine, etc.

A technical distinction is now drawn between quarantine and isolation. *Quarantine* refers to the detention of well persons exposed to infection for the period of incubation of the disease. *Isolation* refers to the segregation of the sick and carriers. The terms are often used interchangeably. Precise knowledge has greatly lessened the rigors of quarantine, which aims only at sanitary isolation.

The dominating principle in modern quarantine is that it must be a sieve or filter and not a dam. All quarantines based upon the principle of the Chinese wall are doomed to fail. The object of quarantine is, then, to destroy, detain, or isolate infection with the least possible hindrance to trade and travel. The art consists in regulating the openings in the quarantine sieve so as to hold back certain infections, but permit all else to pass. Maritime quarantine may be regarded as a coast defense against exotic pestilence, a defense which guards against an invisible foe oftentimes more damaging than hostile armies and navies.

The cure for quarantine is sanitation. Thus, if all communities, especially seaports, were to place their cities in the best sanitary condition in accordance with the teachings of modern science, there would be little danger of disease spreading to epidemic proportions and very little need of maritime quarantine. If the ports in our southern littoral would free themselves of the *Stegomyia* mosquito they could laugh at yellow fever. A city containing few rats could not have an epidemic of plague. A port supplied with a pure, well-protected water supply need not fear a water-borne epidemic of cholera. A thoroughly vaccinated community runs no hazard from smallpox. Typhus fever could not spread in a community with cleanly personal habits, that is, one free from lice vermin.

Quarantine of Contacts.—It often becomes a difficult question to determine whether the well members of a household should also be quarantined—

especially whether the well children should be permitted to attend school. This perplexing question must be decided for each disease separately, and the decision in each disease is sometimes modified by attending factors. Usually the other children in the family in the case of scarlet fever are excluded from school for one week from the last exposure to the infection. In most cities the same rule holds for diphtheria, although here we are able to determine whether the children are bacillus carriers or not. At least two negative cultures from the nose and throat should be required before such children are allowed freely to mingle with other children. The principal factors which determine whether the well children in a family shall be permitted to attend school or not in any particular infection rest upon our knowledge as to whether the disease is conveyed by a third person and the frequency of bacillus carrying and of missed cases.

One of the practical objections to quarantine and one reason that it meets with so much opposition from the public is that the compensation of the wage earner ceases through no fault of his own. It is evidently unjust practically to imprison and punish a wage earner for the good of the community, because he or some member of his family has contracted an infection, perhaps through some fault of the community itself. It is, therefore, reasonable and just that wage earners at least should be compensated and their personal interests safeguarded during enforced isolation.

Isolation.—In theory isolation is the most perfect single method to check the spread of a communicable disease. The results in practice, however, have been disappointing on account of unusual difficulties. The statement has frequently been made, especially with reference to typhoid fever, that if all the cases could be isolated (which includes the disinfection of the discharges) we would soon see an end of the infection. We now know that this statement is not true, on account of the bacillus carriers and the mild and unrecognized or "missed" cases. Because the isolation of the reported cases represents only a portion of all the foci of infection and, therefore, at best could not in itself control an epidemic disease, discredit has been thrown upon this procedure, which is one of the essential features of all systems of prevention. As a matter of fact, it has been shown that in certain diseases, like measles, which is communicable for three days or more before the nature of the disease is recognized, isolation has practically no influence in diminishing the prevalence of this widespread infection. It is true ordinarily that a case of measles does most harm before it is isolated; nevertheless, this is no reason why it should be permitted to further endanger the community. The value of isolation is also diminished by the prevalence of carriers. Chapin (*J. Prev. Med.*, 1926, 1:1) believes isolation has been effective in controlling scarlet fever.

If each case isolated prevents on the average only one other fresh infection, there would still be justification sufficient to continue the practice. As a matter of fact, the practical value of isolation varies with each disease, depending upon the degree of its communicability, the time when it is communicable, the promptness by which it may be recognized, the modes by which it is trans-

ferred, the existence of latent infections, missed cases, carriers, and other factors which influence the spread of the infection.

Young²³ reports the results obtained from three degrees of isolation in the home:

A—Isolation with trained attendant.

B—Isolation without trained attendant.

C—Impossible to isolate for lack of room for exclusive use of the patient.

It was found in the case of scarlet fever 1.087 per cent of secondary cases occur in Class A, 5.22 per cent in Class B, and 6.9 per cent in Class C. Cases cared for in hospital showed 2.32 per cent of secondary cases. In the case of diphtheria there were no secondary cases in Class A; 1.18 per cent in Class B; 4.88 per cent in Class C; and 0.15 per cent in those treated in hospital. These figures clearly show that the value of isolation depends upon the intelligence and care with which it is carried out.

The degree of isolation varies markedly with the different infections. A case of yellow fever may be isolated under a mosquito screen, and a case of diphtheria or scarlet fever may be effectively isolated in a bed in a general ward, provided intelligent and painstaking care is exercised to destroy the infection as it leaves the body. Isolation of the more readily communicable diseases, as smallpox and measles, call for special measures. Typhoid bacillus carriers need not be imprisoned. It is sufficient to limit their activities, especially to prevent their occupation in kitchens, dairies, or about foodstuffs. There is no good reason to isolate a consumptive or leper without open lesions—that is, cases in which the bacilli are locked up in the tissues and not discharged into the environment. A careful consumptive or leper may be allowed a wide latitude. On the other hand, isolation in chronic infections, such as tuberculosis and leprosy, with open lesions, while helpful is at the same time a most difficult procedure. The careless, indigent, ignorant, or helpless consumptive is a public menace that needs energetic and sometimes arbitrary measures.

Isolation of the acute infections may most readily and effectively be carried out in hospitals or sanatoria. Proper isolation in the home requires a special room or rooms, intelligent nursing, appliances for disinfection, etc., a combination often difficult to arrange. House quarantine varies with the different diseases. To carry it out rigorously in all cases and under all conditions is folly. Different diseases need different procedures. Sometimes it is sufficient simply to placard the house as a warning. At other times it may be necessary to station sanitary guards about the premises to enforce the requirements. The imperfections of strict isolation by the “shutting in of houses” are graphically described in Defoe’s *Journal of the Plague Year*.

²³ *J. Am. M. Ass.*, 1915, 64: 488.

Isolation camps or temporary barracks in times of epidemics are effective measures in checking the spread of some infections. This method has proved effective in actual practice in the case of smallpox, typhus, yellow fever, plague, cholera, and other diseases.

Isolation becomes one of our most valuable public health measures when communicable diseases affect persons working about milk, meat, and other foods capable of conveying infection.

In practice, isolation only reduces to a moderate degree the prevalence of disease. The limitations of this valuable procedure are now well understood. With improved methods of diagnosis and increased knowledge of the methods of the spread of disease, isolation will be made increasingly effective. Every case isolated is a focus of infection neutralized. Although not as satisfactory in practice as it is in theory, isolation will ever remain one of the administrative procedures for the control of the communicable diseases. A liberal policy with regard to isolation, the restraint of contacts, and recommendation of private funerals would take away some of the objectionable features of public health administration and help the coöperation of the public.

In the past, geographic isolation was one of the safeguards of the people against disease, but in modern times, since all means of transportation have improved, and communication has become more extended and more rapid, the diffusion of infection is facilitated.

MARITIME QUARANTINE

Maritime quarantine in this country is enforced against seven diseases, viz., cholera, yellow fever, plague, typhus fever, smallpox, leprosy and anthrax. We do not quarantine against typhoid fever, tuberculosis, measles, and other infections which are not so greatly feared and which are constantly with us. Persons infected with diseases of this character arriving at a port are permitted to enter, but must then comply with the local laws and regulations.

The period of detention is based upon the usual period of incubation for each disease and is as follows:

<i>Disease</i>	<i>Period of Detention</i>
Cholera	5 days
Yellow fever	6 days
Pneumonic plague	7 days
Typhus fever	12 days
Smallpox	14 days
Leprosy	not admitted
Bubonic plague	none
Anthrax	none

The period of detention is counted from the last possible exposure to the infection. This is sometimes difficult for the quarantine officer to decide, and in case of doubt the public is given the benefit.

Quarantine restrictions are now much less severe than formerly. This is justified by our increase in knowledge. The changes have been chiefly administrative and consist in shortening detention and simplifying procedures. Trade and travel enjoy fewer restrictions, while preventive measures are more precise and effective than formerly.

At well-equipped stations, where laboratory facilities are available, it is not necessary to detain cholera contacts for the full period of incubation. Properly conducted stool examinations for vibrios will furnish reasonably dependable evidence upon which to hold or release those persons who have been exposed to infection. In the case of smallpox, those who have had the disease or have recently been successfully vaccinated need not be detained. Last year (1924) was the first time in seventy years that our Gulf ports had no quarantine against Central and South America on account of yellow fever. Good sanitary conditions decrease the need of quarantine restrictions.

No communication is permitted with a vessel in quarantine excepting under the supervision of the quarantine officer; that is, no one is allowed to board the vessel or to leave it, and nothing is allowed to be thrown overboard, taken ashore, or brought on board without the express permission of the quarantine officer. These restrictions apply alike to foods, supplies and merchandise of all kinds.

The vessel may be disinfected, furnished with a fresh crew and released from quarantine while the passengers and crew are detained in suitable barracks. When practicable, vessels trading with infected ports should carry immune crews; that is, persons who have either had the disease or have been rendered actively immune by means of vaccines or viruses.

When a quarantinable disease breaks out on board a vessel it is of practical importance for the quarantine officer to determine whether the infection was contracted on board the vessel or on land. In the first case the measures necessary for the purification of the vessel may be more exacting. Thus, if plague appears within a few days after a vessel leaves an infected port, and no other case occurs, it is probable that the patient contracted his disease ashore and was in the period of incubation when he came on board. If, however, plague breaks out after seven days, and especially if secondary cases occur, it is evident that the ship harbors the infection. The same reasoning applies to yellow fever and the other quarantinable diseases.

The measures taken at quarantine to keep out these diseases depend upon an accurate knowledge of their causes and modes of transmission. Briefly summarized, the measures applicable in each case are as follows:

Cholera.—The sick are removed from the vessel and isolated. Care is taken to disinfect and properly dispose of the patients' discharges. Eating utensils and all other articles used in the sickroom should be disinfected before removal, and the room should be well screened to exclude flies.

All suspected cases, especially those with gastro-intestinal symptoms, should be isolated and treated as positive cases until they shall have recovered or shall have been definitely diagnosed. All contacts are segregated in small

groups and stool examination instituted immediately for the purpose of detecting "carriers."

In the meantime steps have been taken to destroy all sources of infection on board the vessel. The probabilities of infection on board can be determined largely from the history of the outbreak. If the outbreak has been of an "explosive" type, an infection of the water supply is suggested. This can readily be determined by a bacteriological examination of the water. If there be any scarcity of fresh water, a ship's supply should not be condemned unexamined. If there be ample fresh water available, the ship's supply can be disinfected by the addition of bleaching powder or permanganate of potash. The tanks are then emptied and refilled with clean water. Or the water supply can be purified by the addition of hypochlorite of lime.

If the voyage has consumed several days and there are only a few cases, the source of infection will generally be traced to a carrier or carriers. The protected water supply and water carriage system of sewage disposal on modern vessels have materially diminished the potentialities of cholera infection on ships. Formerly, before these sanitary conveniences were provided, the appearance of a case of cholera on a vessel was often followed by contamination of the water supply with consequent infection of a considerable proportion of the passengers and crew if the voyage was of long duration.

As an added precaution fruits and vegetables that are generally eaten raw may be destroyed. The clothing and baggage of the sick and of the carriers should be disinfected, preferably by steam, but this procedure is not necessary with respect to the clothing and effects of the ship's personnel in general. The bichlorid baths formerly in vogue are probably a waste of time.

Treatment of the cargo is not required as the holds remain sealed throughout the voyage and the chance of contamination is nil. The danger of spreading cholera through fomites is slight.

Disinfection of the vessel is not necessary except in the case of compartments which have housed the sick or carriers, and thereby may have been contaminated. The floors and lower walls of such compartments should be washed down with a solution of carbolic acid, bichlorid of mercury, or other equally satisfactory disinfectant. This is one of the few instances in maritime quarantine procedure in which bactericidal measures are indicated. The bedclothes, mattresses and other fabrics from the compartment where the sick have been should be steamed or soaked in a germicidal solution.

The ship itself is seldom infected, but the danger resides in the personnel. Therefore, attention should be focused upon the detention and isolation of cases and carriers and the safe disposal of their dejecta, until it is no longer possible to detect cholera vibrios therein.

If the quarantine station be not provided with adequate laboratory facilities, contacts will have to be detained for the legal period of incubation, i.e., five days. The detention of contacts for five days after the exposure to infection was formerly considered to be sufficient and still serves the practical purpose of excluding active cases of the disease but not carriers. It is now

known that the important rôle played by carriers makes it essential that a bacteriological examination be made (see page 133).

Smallpox.—Ordinarily those persons who have had smallpox or who have had a recent successful vaccination are not detained. All others who have been exposed to infection must submit to vaccination or remain in quarantine for the full period of fourteen days. Contacts may be released as soon as they show an immediate reaction, which indicates immunity. As a rule, it is not necessary to detain cabin passengers because there is smallpox in the steerage, or to detain the firemen because there is smallpox among the stewards. Vessels arriving with smallpox on board and on which the cases have been properly isolated, the personnel vaccinated, and other precautions sufficient to prevent the spread of the disease have been taken, need not be detained further than to permit the removal of the sick, and the disinfection of compartments, baggage and objects that have been exposed to the liability of infection (see chapter on Smallpox and Vaccination).

Plague.—In the management of a plague-infected vessel, a sharp distinction should be made between bubonic and pneumonic plague. From a quarantine standpoint these are two separate diseases, one transmitted by fleas from rats, and the other by direct personal contact (see page 341).

Bubonic Plague.—The treatment of a vessel infected with bubonic plague should be directed towards the destruction of all rats and fleas. Rats found dead or killed by fumigation should be immersed in coal oil and autopsied if possible, and in all cases the carcasses should finally be burned.

It is not necessary to detain passengers and crew who are in good health any longer than is incidental to the disinfection of the vessel, for the bubonic form of the disease is not communicable directly from person to person.

Formerly, much stress was laid on the examination of passengers and crew, but there is no record of bubonic plague ever having been introduced into any port or place by infected man. It is only the infected rat or flea that carries the disease from one locality to another.

In handling an infected vessel it is first necessary to make sure that no rat escapes. Preferably, the ship remains at anchor in quarantine, or lies alongside the quarantine station wharf, with all mooring lines protected by metal discs three feet in diameter fixed at right angles to the hawser to which they are attached. The discs act as barriers to the passage of rats from ship to shore. After the passengers and crew have been removed to the quarantine station, the vessel must be simultaneously fumigated in all parts to destroy rats and fleas. Hydrocyanic acid gas is the most efficient fumigating agency, but it is very dangerous and should be used only by those especially trained. The cyanogen chlorid gas mixture combines the efficiency of hydrocyanic acid with the warning of the cyanogen chlorid.

Sulphur dioxid is not very effective if the vessel be cargo laden, and is injurious to certain kinds of cargo, such as tea, silk, coffee, tobacco, etc., and to ship furnishings, and will often discolor paint, particularly white lead pigment. It is safe, however, and fairly effective when the compartment is empty and

penetration and diffusion not so essential. Details of ship fumigation are described under separate heading on page 539.

Funnel gas (carbon monoxid) is not lethal to insect life, and, while satisfactory for ordinary rat distruction, it should not be employed for this purpose on plague-infected ships, as it will not kill fleas.

Rodent destruction on a cargo-laden vessel is best accomplished by repeated fumigations, the upper layers of cargo being discharged after each fumigation.

While it is ordinarily not necessary to disinfect the clothing and personal effects of passengers and crew, as fleas are seldom found on them, certain groups of persons require attention along this line. Lascars and other Asiatic crew men harbor fleas in the voluminous folds of linen or cotton (generally soiled) with which they swathe their bodies. The quarantine officer must necessarily exercise discretion as to the disinfection of the clothes and personal effects of passengers and crew.

Domestic animals, particularly cats and dogs, demand special attention on infected vessels. When there has been a great mortality among the rodents the dislodged fleas are prone to secure temporary quarters on cats and dogs and thus these pets, themselves immune to plague, may become a serious menace. The animals should be immersed in a coal oil emulsion so as to kill fleas and other vermin.

Pneumonic Plague.—In the treatment of a vessel infected with pneumonic plague, the sick should be carefully isolated and the passengers and crew segregated in small groups. Recovered cases should not be discharged until a bacteriological examination of the secretions of the nose and throat indicate the absence of *B. pestis*, as cases may become carriers, at least for a short period of time. Contacts should be detained seven days before release, the time counting from the last day of possible exposure. Special care should be exercised to detect new cases among the groups of contacts. The temperatures of all contacts should be taken twice daily, and any person developing the least rise of temperature should be immediately isolated and held under observation until the diagnosis is established or until recovery.

The kitchen and dining room utensils used by the sick or convalescent should be sterilized. All living quarters on the vessel should be disinfected and washed down with a solution of bichlorid of mercury or other germicide. Personal effects and bedclothes should be disinfected by steam.

Plague Precautions.—In order to exclude plague infection, it is necessary to institute preventive measures, not only against vessels known to be infected, but also against all vessels arriving from ports known to be infected, with rodent plague. Infection may have been introduced into the hold of a vessel and spread among the rats without any evidence of the disease being noted. It is improbable that human cases will occur unless there are infected rats on board. It is possible for rodent infection to remain quiescent in a vessel or for an undetected epizootic to prevail among the rats even though the voyage may have consumed several weeks. Infected rats have

been taken from vessels that had not entered an infected port for several weeks, although there were no human cases reported and no extensive epizootic among the rodents (see also page 328).

Yellow Fever.—As a precautionary measure during the yellow fever season, vessels arriving at an infectible port from an infected port are fumigated and detained six days, even though there is no evidence of sickness on board. Vessels which were fumigated at the port of departure under the supervision of a medical officer of the Public Health Service are not refumigated. The yellow fever season usually extends from April 1 until November 1. In the United States the infectible ports are those situated upon the Atlantic seacoast south of the southern boundary of Maryland and on the Gulf of Mexico. Yellow fever is a vanishing disease and the restrictions of maritime quarantine have been largely removed.

The sick are isolated by the use of mosquito screens. Patients with yellow fever should not be moved if this involves exertion or excitement, which may aggravate the disease.

The vessel is fumigated with an insecticidal substance, preferably SO_2 , or HCN, or the cyanogen chlorid gas mixture in order to kill *Stegomyia* (*Aedes aegypti*). A search is made for breeding places, such as water casks, fire buckets, and other collections of fresh water where the larvæ and pupæ might develop. The disinfection of baggage and fomites is no longer practiced in the case of yellow fever. Experience has shown that wooden vessels are more likely to convey yellow fever than iron vessels. This is because wooden vessels usually carry water casks, favorite breeding places for the yellow fever mosquitoes, while iron vessels store their drinking water in tight compartments deep in the hold, inaccessible to mosquitoes. If practicable, vessels plying between infected and infectible ports should be manned by crews immune to yellow fever (see page 292).

Typhus Fever.—If typhus fever appears on board a vessel there are likely to be several cases, especially if the infection develops among the crew or the steerage passengers. For this reason all possible contacts should be detained in quarantine for the full period of incubation, which has been fixed at twelve days.

If the disease has been confined to the steerage, there is no necessity for detaining cabin passengers, and vice versa. If the disease has appeared only in the crews' quarters, and these are well separated from the passengers' quarters, there is no occasion for the detention of the passengers.

The sick should be removed to the quarantine hospital and their clothing, personal effects and baggage thoroughly treated for the destruction of lice. The patient's body is treated with a delousing solution (mixture of kerosene and vinegar in equal parts, or kerosene and soft soap), and the application of some such solution is especially important for the destruction of head lice. The hair should be clipped not only on the head but also in the pubic and axillary regions, the clippings burned, and finally, the patient given a thorough bath.

Clothing is best disinfested by steam. Hydrocyanic acid is effective for the destruction of lice, but is not a germicide.

All contacts should be treated with a delousing solution and bathed under supervision. Their clothing and baggage should be disinfested.

Persons detained should be segregated in small groups and their temperatures taken twice daily, but if the delousing procedure has been efficiently performed, secondary cases are of no further sanitary concern. To be on the safe side, however, secondary cases should be isolated immediately upon their detection.

As to the treatment of the ship, no action is called for other than the assured destruction of lice in living quarters. The galleys, storerooms and holds need no treatment unless there be unusual circumstances that render them probably vermin-infested. Staterooms, crews' quarters and steerage compartments should be fumigated with sulphur dioxid, hydrocyanic acid or the cyanogen chlorid gas mixture (technic as described on page 541), with bed-clothes and furnishings remaining in place. After fumigation the vessel may be released with the personnel that have not been exposed or a new crew furnished, if necessary.

The important features in the quarantine of typhus are that all vermin should be destroyed, the cases detained, and the contacts isolated until the period of incubation shall have elapsed (see page 370).

Leprosy.—An alien leper is not allowed to land. The law requires the vessel on which he arrives to take him back again. It is unconstitutional to forbid the landing of an American leper, but as soon as he lands he comes under the laws of the city or state in which he finds himself. Alien lepers are detained at the quarantine station and placed aboard again when the vessel is outward bound. The national leprosium is at Carville, Louisiana (see page 413).

Quarantine Procedures.—All vessels arriving at any port in the United States from a foreign port are considered to be in quarantine until they are given *free pratique*. The *pratique* is a certificate, signed by the quarantine officer to the effect that the vessel has, in all respects, complied with the quarantine laws and regulations, and all on board are free from quarantinable disease, or the danger of conveying the same. The master of the vessel must present the *pratique* to the collector of the port in order that his vessel may be admitted to entry.

Vessels in quarantine are required to fly a yellow flag (letter "Q" of the International Code) from the foremast. The quarantine officer boards the vessel, and examines the bill of health, the ship itself, the passengers, the crew, the manifests of cargo, and sometimes the log, the food and water supplies, etc. Ordinarily vessels arriving after sundown must wait until sunrise for this inspection; the time and details vary and depend upon circumstances.

The detection of infection on board a vessel requires knowledge, experience, and sometimes a detective instinct on the part of the quarantine officer. If one of the quarantinable diseases is suspected the temperature of every person

on board should be taken. As a rule, all hands are mustered at a designated place on board the ship and then passed in review, one by one, before the examining physician; the number of persons is counted and the result compared with the ship's papers; each person is critically scrutinized for evidence of disease, and suspects are placed aside for more careful examination later. The clinical records of the ship's surgeon are inspected with special reference to the incidence of quarantinable disease during the voyage. The manifest of cargo may be examined for hides, skins, hair, shaving brushes, rags, or other objects which under certain conditions are prohibited or may require disinfection. Finally, the ship itself may be inspected, attention being given especially to the fore-castle, the steerage quarters, the galley, etc.

The Bill of Health.—The United States Bill of Health is issued by the American consular officer at the port of departure to the master of the vessel. This document contains a description of the vessel, the number of officers, of crew, and of passengers (first cabin, second cabin and steerage), the sanitary history of the vessel and a statement as to the sources and wholesomeness of the water and food supplies. It also contains a statement giving the number of cases and deaths from yellow fever, cholera, cholera nostras or cholerine, smallpox, typhus fever, plague, and leprosy at the port of departure during the two weeks preceding the sailing of the vessel.

The American Bill of Health, which is a formidable document, must be obtained by the master of the vessel in duplicate, and presented to the quarantine officer at the vessel's port of destination. After these documents have served their purpose in affording to the quarantine officer the necessary information concerning quarantine inspection of the vessel, they are returned to the master of the vessel and by him surrendered to the Collector of Customs.

The Bill of Health is a consular document (State Department) at the port of departure, but becomes a customs' paper (Treasury Department) at the port of entry. Vessels arriving at any port in the United States, its possessions or dependencies from a foreign port without this official Bill of Health are subject to a fine not to exceed \$5,000. Before the days of telegraphy the Bill of Health often gave the quarantine officer the first information of a pestilential disease abroad. The quarantine officer must now keep himself informed of the presence and prevalence of quarantinable diseases at the places from which the passengers and crew are recruited as well as at the port of departure.

There are many kinds of bills of health; each country has a form of its own. Formerly a bill of health was simply a statement that the port of departure was or was not free of pestilential disease; that is, the bill of health was either "clean" or "foul." The American Bill of Health gives much more valuable information in detail. The only bill of health that is of service to a vessel upon arrival at a port in the United States is the American Bill of Health, although several bills of health may have been secured at the port of departure. Thus, a British vessel leaving the port of Rio de Janeiro for the United States takes three bills of health, one from the British consul, required

by the British admiralty laws, another from the Brazilian authorities, which is a clearance paper, and the third from the American consul.

The Equipment of a Quarantine Station.—The equipment of a quarantine station consists of wharves, boathouses, and boarding vessels, such as tugs, launches, and rowboats; an inspection place where passengers and crews may be examined (the facilities on board the ship are usually inadequate for this purpose); disinfecting apparatus for the use of steam, sulphur dioxid, hydrocyanic acid, cyanogen chlorid, formaldehyd, and insecticides; shower baths; detention barracks for steerage, intermediate, and cabin passengers, as well as the crew of the vessel; isolation wards for cases of quarantinable disease, and wards where suspects or non-contagious cases may receive treatment. A well-equipped quarantine station needs dining rooms and kitchens for the various groups detained; quarters for the quarantine officers and personnel, and some provision for the recreation of those in quarantine. A crematory or cemetery, and facilities for the proper disposal of sewage and garbage are important.

A well-equipped laboratory is an essential part of a modern quarantine station. It is frequently necessary to confirm or reject diagnoses by bacteriological examination. It is also important that the rats recovered after the fumigation of vessels be examined for evidence of plague.

Qualifications of the Quarantine Officer.—A competent quarantine officer must be a good diagnostician. He should have an especial acquaintance with the diseases against which he stands monitor. The diagnosis of disease on an arriving vessel is doubly important, as it is in the nature of a medicolegal decision. Failure to recognize one of the quarantinable diseases jeopardizes the public health of the country. On the other hand, an error in diagnosis may result in a needless loss of time and money to the passengers and steamship companies. The quarantine officer should be an experienced bacteriologist or have such an expert as an assistant, since the accurate diagnosis of several of the quarantinable diseases is essentially a laboratory procedure, and even in those quarantinable diseases whose diagnosis rests on clinical evidence, laboratory procedure is important in differentiation. The quarantine officer must also be familiar with the modes of spread of the quarantinable diseases, and must know the value and limitations of the germicidal agents and insecticides he uses. He must be familiar with matters nautical and have an extensive knowledge of geography. It is his duty to keep informed as to the prevalence of the quarantinable diseases throughout the world, with special reference to the seaports in frequent communication with his own port.

Disinfection of Ships.—The principles involved in the disinfection of a vessel do not differ materially from those of house and room disinfection. A certain familiarity with the intricacies of marine architecture and matters nautical is essential, for many conditions are met on board ship that are very different from arrangements found on shore. While the theory of disinfecting a vessel presents nothing unusual, the application of the necessary procedure calls for much ingenuity and the keenest vigilance on the part of the disinfector.

It is important to convince those on board of the necessity for disinfection, since the successful purification of the vessel may be materially aided by the cheerful coöperation of the passengers and crew.

Formerly a distinction was made between the method of disinfecting a wooden vessel and an iron vessel. This arose from the fact that almost all wooden vessels are rotten and spongy in places, especially about the forefoot and bilge. There are also many more cracks and open joints affording lodgment for organic matter about a wooden ship than a metal vessel. Since almost all wooden vessels leak more or less, a wooden hull is likely to be damper than an iron hull. It was formerly believed that the microorganisms of disease were likely to become deeply lodged in the moist dirt and organic matter of these crevices. We now know that this is not an important factor.

A vessel is rarely so badly infected as to need disinfection throughout. Just what portion of the vessel and its contents requires treatment is often a perplexing problem. There is no more reason to disinfect the hold of a vessel because smallpox has appeared in the cabin or steerage than there would be to purify the basement and sub-basement of a tenement house because a case of smallpox appeared in one of its upper stories. Infection may be confined to one or two compartments of a vessel quite as successfully as in the case of buildings on shore. "In case of doubt, disinfect," is not a bad rule for the quarantine officer to follow in his practical dealings with ships. Although the measures taken may exceed the absolute requirements, discrimination is necessary, for the disinfection of ships for the quarantinable diseases has been greatly overdone. Vessels often need fumigation, but rarely disinfection.

No routine method of disinfection for all infected ships should be prescribed, but judgment must be exercised. Disinfectants or fumigants which will destroy the virus and also the vermin vectors should be selected.

It is, therefore, the duty of the quarantine officer to require a very thorough mechanical cleansing of all parts of the ship which, in his judgment, require it. This matter is dwelt upon because filth and vermin are too frequently encountered on the sea and are of great importance to communities and nations.

While the general method of treating vessels is the same for most of the bacterial infections, special methods are required for each disease. In the case of cholera, particular attention must be paid to the water and food supply; in plague, the destruction of rats and fleas is of prime importance; in yellow fever, attention must be directed against the mosquito; in smallpox, vaccination and the disinfection of the living apartments, clothing, bedding, and the like are required; while for typhus fever, the warfare must be waged against lice.

Before the disinfection of a vessel is commenced it should be brought alongside the pier or barge containing the necessary apparatus. All of the passengers and crew, excepting the few who must be left on board for the safety of the vessel and those who are to help in the purification, are then

disembarked. The quartermaster, the boatswain, and the carpenter are useful aids on account of their practical knowledge of the construction of the vessel and their faithfulness in carrying out directions with intelligence.

The disinfection of ships applies only to the quarters actually occupied by the sick in the case of vessels infected with typhus, pneumonic plague, smallpox, leprosy or cholera. In handling vessels infected with bubonic plague or yellow fever, steam sterilization of the ship's furnishings and of the clothing and personal effects of the personnel is not necessary.

When the personnel have left the vessel all their effects are removed and disinfected, if necessary, in accordance with the methods outlined for objects of that class. Disinfected baggage, bedding, and other objects should not be returned on board until the treatment of the vessel is completed. This injunction applies equally to persons. No one should be allowed on the vessel except those actually engaged in the work, who, as far as practicable, should be immune and should wear suitable garments. All the bedding, bedclothes, hangings, floor runners, and other fabrics that have been exposed to infection must now be removed to the steam chamber. Especial care must be taken to obtain all the used and soiled linen, which is usually kept in special compartments called the "dirty linen lockers," under the care of one of the stewards. For some reason there is a reluctance to disclose the presence of this soiled wash to the quarantine officer.

After all the objects requiring disinfection by a special process have been removed, attention is directed to the vessel itself. The various compartments may be disinfected by any one of the methods described under Room Disinfection, formaldehyd being the choice of the gases and bichlorid of mercury (1:1,000) being the most suitable solution for the treatment of walls, floors, and surfaces.

Before the means of transmission of plague, typhus and yellow fever were known, bichlorid disinfection was much employed at quarantine stations. The tank holding several thousand gallons of the mixture was one of the most important features of a quarantine station. This practice has now well-nigh been abandoned, but still has a limited usefulness in washing down the floors and walls of compartments that have been contaminated by discharges from a cholera patient.

Fumigation of Ships.—The object of fumigation is to destroy rats, mice, fleas, lice, mosquitoes and other vermin that act as vectors of disease. (For distinction between fumigation and disinfection see page 1314). In the purification of a ship, the only practical gases are sulphur, hydrocyanic acid or cyanogen chlorid gas. For many years sulphur dioxid was the agency chiefly relied on. Cyanid gas was regarded as too dangerous to human life and too expensive for routine use. During the last ten or twelve years, however, the procedure has been so developed and improved as to make it reasonably safe when supervised by trained fumigators.

Cyanid gas has many points of superiority over sulphur dioxid. Sulphur dioxid is not very diffusive, and when used on cargo-laden vessels has but

little penetrating power, so that the air pockets in articles of cargo or between packages of merchandise will often afford the rats a sufficient protection against the effects of the sulphur fumes. Sulphur dioxide is highly destructive to finer textiles, especially if damp; it tarnishes brass and gilt fixtures, damages delicately adjusted instruments, and causes discoloration of lead-painted surfaces. The generation of sulphur fumes, if by furnace, requires expensive apparatus and, if by the "pot and pan" method, much labor and cumbersome apparatus. Considerable time is consumed in placing and removing the pots and pans, and the exposure required is from six to twelve hours. Added to these defects, there is always the risk of fire from the burning sulphur. In contrasting sulphur fumigation and cyanid fumigation, the one feature in favor of sulphur is that the hazard to human life is slight.

On the other hand, cyanid gas is highly toxic to animal and insect life in the strength of five ounces of cyanid per thousand cubic feet of space, and this with rather short exposure. It is not injurious to the finest textiles nor to such articles as tea, coffee, tobacco, sugar, etc. It does not tarnish brass or gilt, nor discolor painted surfaces. It is easily and quickly generated and requires but simple equipment.

Cyanid Fumigation of Ships.—The paraphernalia required and method of preparation of the gas, adopted by the Public Health Service, are as follows: tight wooden barrels (the well-made oak barrel used for kerosene serves excellently) for use in holds; earthenware crocks or jars for smaller compartments, and earthenware jugs as acid containers. The mixture of sulphuric acid and water is first prepared in the proportion of two fluid ounces of water to one and one-half ounces of commercial (66B) sulphuric acid. Finally, when most of the hatch coverings have been placed in position, sodium cyanid, in the proportion of one ounce to each three and one-half ounces of the above mixture, is lowered in a cheesecloth sack, dropped into the solution, bag and all, and the remaining hatch covers adjusted and battened down by a heavy tarpaulin.

The strength of the gas and the duration of exposure vary with the objects sought. The Public Health Service standards in this respect are as follows:

For destruction of mosquitoes: one-half ounce of sodium cyanid per thousand cubic feet of space; exposure one-half hour.

For destruction of fleas: two and one-half ounces sodium cyanid per thousand cubic feet of space; exposure one-half hour. In practice, ships are not fumigated for flea destruction only, but always with the idea of rat destruction as well as flea destruction.

For destruction of rodents (rats and mice): five ounces of sodium cyanid per thousand cubic feet of space; exposure for two hours.

For destruction of lice: ten ounces of sodium cyanid per thousand cubic feet of space; exposure two hours.

For destruction of bedbugs: five ounces of sodium cyanid per thousand cubic feet of space; exposure for one hour.

The above standards apply to empty holds and superstructures, except storerooms that have a large quantity of stores. In cargo-laden holds or in well-packed storerooms, the length of exposure is doubled.

If the potassium salt be used, a greater quantity is required. Three and three-fourths ounces of sodium cyanid is the equivalent of five ounces of potassium cyanid, and the latter when used is in the proportion of one ounce of potassium cyanid to one fluid ounce of sulphuric acid and two and one-half fluid ounces of water.

As a preliminary to cyanid fumigation the quarantine officer should make sure that no persons other than the fumigating force are on the vessel, and a certificate to this effect should be exacted of the master. This should further be confirmed by a search of the vessel by the quarantine officer himself or a trusted assistant. The compartments above the deck should have danger labels pasted on doorways after the fumigation has commenced.

The experience of the Public Health Service in fumigating several thousand vessels, as well as large storehouses, with cyanid makes it appear that there is no especial danger to the operator in the preparation of the gas.

The generation of gas is sufficiently delayed to afford ample time for the fumigators to leave a compartment after dropping the cyanid into the acid solution. The serious danger to human life arises incident to entering the compartment *after* the fumigation has been completed and the vessel opened up. To obviate this danger, powerful ventilating fans, driven by portable gasoline engines, are utilized at quarantine stations. These are lowered into the holds or placed within the doorways of above-deck compartments upon the completion of the fumigation.

The Public Health Service rules provide that no one may enter a compartment subsequent to cyanid fumigation until it has been pronounced safe by the quarantine officer. To determine this point, captive rats, cats, or guinea-pigs are used as indicators.

Cyanogen Chlorid Gas Mixture.—Cyanogen chlorid gas mixture is one of the most penetrating and toxic of all fumigants, though cyanogen chlorid gas in pure form is only approximately half as lethal as hydrocyanic acid gas. However, when generated by the procedure prescribed by the United States Quarantine regulations, an amount of hydrocyanic acid gas equal to approximately 30 per cent of the mixture is simultaneously generated, thus making the mixture more toxic than the pure cyanogen chlorid gas. Cyanogen chlorid is highly lacrimatory, even in non-lethal quantities. Due to the warning thus given, its advantages over hydrocyanic acid are obvious, and the hazard, as compared to hydrocyanic acid gas, is greatly reduced. The generation of the cyanogen chlorid gas mixture, when produced by the method described below, is somewhat slower than the generation of hydrocyanic acid gas, therefore more time is allowed the operator to reach safety.

When cyanogen chlorid mixture is used as recommended, and by careful and experienced operators, it is as safe as sulphur dioxide. Cyanogen chlorid for fumigation purposes is generated by a mixture of hydrochloric acid, water.

sodium cyanid, sodium chlorate, and talc. The sodium chlorate and talc may be mixed in the proper proportions and kept in bulk. The sodium cyanid should be kept in the original air-tight packages. When the cyanid is used in the form of cyanegg it should be crushed to pieces approximately one-half inch in diameter, but in no instance should it be powdered. The sodium cyanid should not be added to the sodium chlorate talc mixture until the moment of preparing the proper quantity for each fumigation. The dilution of the hydrochloric acid with equal parts of water may be done at any convenient time, as the generation of gas is not dependent upon the heat generated by mixture of the water and acid. The standard amount recommended below will give sufficient concentration in any given space to produce death in an average weight adult rat in not more than ten minutes. Such concentration requires approximately 0.941 ounce of cyanogen chlorid gas and 0.327 ounce of hydrocyanic acid gas per thousand (1,000) cubic feet of air space. The formulæ are given on page 544.

The length of exposure is dependent on the object of the fumigation with different periods for the destruction of mosquitoes, fleas, bedbugs, lice, roaches, and rodents. All parts of the vessel should be placed under fumigation simultaneously, but, on account of the slightly tarnishing effect on highly polished metals, the chart room and wireless room should be omitted.

Method of Use. Safety Precautions.—The paraphernalia required consists of tight wooden barrels, half barrels, or tubs, preferably of oak with wooden hoops, for use in holds. Earthenware crocks or wooden buckets, preferably of oak for smaller compartments; vessels for mixing acid and water; containers for mixing sodium cyanid, sodium chlorate, and talc; scales or scoops for weighing or measuring the same, and bags for holding the ingredients prior to placing them in the acid.

In the fumigation of superstructures, the acid and water mixture is poured into crocks and buckets, which are placed in position, with the necessary quantities of sodium cyanid, sodium chlorate, and talc mixture, in bags placed near the vessels containing the acid. All doors, portholes, etc., with the exception of those needed for the egress of the operator, are closed and sealed. After all ingredients have been put in place, but prior to the placing of the chemicals in the acid, the medical officer or fumigation inspector, accompanied by the master or his representative, personally inspects each compartment in order to establish the fact that no person other than those authorized by the medical officer or fumigator in charge remains on board. Prior to the beginning of this inspection, a guard is stationed at the gangway to prevent any unauthorized person returning to the vessel and, when practicable, guards are stationed at all unsealed openings. It is important that the personnel actually placing the sodium cyanid, sodium chlorate, and talc mixture in the acid mixture consist of experienced employees, and that the number thus engaged be reduced to an absolute minimum. This personnel is equipped with, and has ready for immediate use, gas masks of an approved design, complete with canister, properly charged for protection against cyano-

gen chlorid gas and hydrocyanic acid gas. The failure of the operator to be equipped with such a mask when engaged on such duty is deemed sufficient cause for disciplinary measures and immediate dismissal. When both the holds and superstructures are to be fumigated the hold is prepared for fumigation, the acid and water mixture mixed in containers, the sodium cyanid, sodium chlorate, and talc mixture placed in bags alongside of the container and attached by rope to the main deck. The hatch covers, with the exception of one section necessary for handling the rope, is in place and covered with tarpaulin. Staggering of hatch covers is strictly prohibited. Upon the completion of the operation of placing chemicals and the acid in the superstructures, the generation of the gas in the holds shall be begun by lifting the chemicals in the container and quickly replacing the remaining hatch coverings and tarpaulin.

The fumigation of crews' quarters, storerooms, and other compartments below the main deck is started, when possible, before the larger volume of gas required in the hold is generated. All persons engaged in opening up after fumigation and who are in any manner exposed to the gas shall under all circumstances wear gas masks during this procedure. On account of the danger to human life from the cyanogen chlorid gas mixture, specific arrangements should be made for debarkation or other safe disposition of the crew during the fumigation process, especially if two or more compartments of the vessel are to be fumigated. A written statement must be obtained from the master of the vessel or his representative that the vessel is ready for fumigation and that all of the crew, personnel, passengers, or others have been accounted for as not being in or on the vessel, except those specifically authorized by the medical officer or fumigator in charge to assist in the fumigation, or to give such care to the engine room or deck as may be necessary under insurance requirements. Compartments above the deck should have danger labels pasted over the doorways after fumigation has commenced.

At ports where physical or weather conditions render the removal of the personnel of a vessel impracticable, fumigation with cyanogen chlorid gas mixture may be performed with the personnel on board, but only after specific authority has been granted by the Public Health Service. When a vessel is under fumigation with cyanogen chlorid gas mixture, no one is permitted to enter the various compartments of the ship until the entry to such space shall have been declared safe by the medical officer in charge of fumigation. Before declaring a vessel safe for entry, the medical officer in charge of fumigation personally visits each compartment of the vessel; provided, in the discretion of the medical officer in charge of fumigation, the inspection of the holds may be made by the fumigator in charge, accompanied by a trained employee, each of whom shall personally enter the holds and report their freedom from dangerous quantities of the gas before release. Decision as to the safety of the vessel for entry is made only by the medical officer in charge of fumigation, who so certifies in writing over his signature.

The word "compartment" includes all superstructures, closets, baths, store-

rooms, holds, or other closed spaces, and such compartments are not declared safe until the absence of the lacrimatory effect of the gas has been positively determined.

Officers and other personnel, when engaged on inspection duty, are at all times equipped with gas masks ready for immediate use should such be necessary. All fumigating personnel engaged in the emptying of barrels, tubs, crocks, and other containers used for the generation of gas are required to use gas masks when removing and emptying the same.

All employees of the Public Health Service engaged in the fumigation of vessels with cyanogen chlorid gas are required to undergo a course of instruction as to the proper use and care of gas masks and to pass a satisfactory examination and to give satisfactory demonstrations of ability to use the mask properly before being permitted to engaged in the actual fumigation of vessels with cyanogen chlorid gas. Whenever practicable, natural ventilation is expedited by the use of such artificial means as windsails, ship ventilators, properly placed tarpaulins, fans, etc., and such mechanical devices as the aërothrust, blowers, and other available apparatus.

Standard Formulae.—The strength of the cyanogen chlorid gas mixture and the duration of exposure varies with the object sought. The Public Health Service standards in this respect are as follows:

A. For the destruction of mosquitoes:

Sodium cyanid	4 drams
Sodium chlorate	3 drams
Talc	2 drams
Hydrochloric acid	2 fluid ounces
Water	2 fluid ounces
Per thousand (1,000) cubic feet	
Exposure: $\frac{1}{2}$ hour	

B. For the destruction of rodents, rats, and mice:

Sodium cyanid	4 ounces
Sodium chlorate	3 ounces
Talc	2 ounces
Hydrochloric acid	17 fluid ounces
Water	17 fluid ounces
Per thousand (1,000) cubic feet	
Exposure: 2 hours	

C. For destruction of fleas, same exposure as for rodents.

D. For destruction of lice, bedbugs, and roaches:

Sodium cyanid	8 ounces
Sodium chlorate	6 ounces
Talc	4 ounces

Hydrochloric acid	34 ounces
Water	34 ounces
Per thousand (1,000) cubic feet	
Exposure: 2 hours	

The talc may be omitted and slight modifications of these formulæ are employed.

The above standards apply to empty holds and superstructures except storerooms that have large quantities of stores. In cargo holds or well-filled staterooms, the length of exposure shall be doubled.

Standard Unit for Fumigation Crews.—At all quarantine stations where the cyanogen chlorid gas mixture is used as the regular gas for fumigation purposes the minimum fumigation crew shall consist of eight persons, as follows:

- 1 chief fumigator
- 2 assistant chief fumigators
- 5 laborers (including 1 truck driver when necessary)

Sulphur and Cyanid Contrasted.—At one of the large quarantine stations on the Gulf, where some of the vessels were fumigated by sulphur and others by cyanid gas, a very thorough study was made as to the relative effectiveness of cyanid gas and sulphur dioxid when used on ships for rodent destruction. A very large force of trained trappers was available because of the anti-plague measures being carried out at the port, and, as the vessels generally stayed in port from one or two days to a week, ample opportunity was afforded for testing the effectiveness of the fumigation. Subsequent to fumigation, the vessels were flooded with traps. The observations extended over a period of one year and the results on several hundred ships were recorded. The number of traps placed varied from 20 to 140 per ship, according to the size of the vessel. After fumigation each ship was carefully searched for dead rats and the number and location of those found were recorded. Ships were trapped from one to ten days, depending on the length of their stay in port.

By contrasting the number of rats killed by fumigation with the number subsequently trapped, a fairly reliable estimate was afforded as to the effectiveness of the fumigation. The results indicated that cyanid fumigation destroyed 95 per cent of all the rats on the vessel whether the holds were empty or loaded, and without regard to the location of the rats. Under similar conditions sulphur fumigation killed only 77 per cent of the rodents on vessels. Studying the results according to the compartments fumigated, it was noted that in superstructures (including cabins, storerooms, poop deck and crew's quarters) cyanid fumes destroyed 94 per cent, whereas sulphur fumes, under similar conditions, destroyed only 55 per cent of the rodents. In cargo-laden holds, sulphur efficiency was 64 per cent in contrast to 80 per cent for cyanid. In empty holds, the results of the two practices were more

nearly equal; 99 per cent efficiency for cyanid, and 96 per cent efficiency for sulphur dioxid.

In this series of observations sulphur dioxid was used in the strength of three pounds of sulphur per 1,000 cubic feet of space, exposure for six hours, and the cyanid was used in the proportion of five ounces of cyanid per 1,000 cubic feet of space with exposure for $1\frac{1}{4}$ hours in holds, and $1\frac{1}{2}$ hours in superstructures. Had the duration of exposure been doubled, the effectiveness of both cyanid and sulphur would probably have been considerably increased.

While cyanid fumigation is undoubtedly the preferred procedure where trained operators are available and the conditions favorable, sulphur dioxid still has a wide field of usefulness, and on account of its safety in unskilled hands and the lack of operators experienced in cyanid fumigation, will doubtless continue to be commonly used for rodent and insect destruction on vessels.

Sulphur Fumigation of Ships.—The Public Health Service standard for sulphur dioxid as to strength and exposure is as follows:

For mosquito destruction: two pounds of sulphur per thousand cubic feet of space; exposure for one hour.

For destruction of lice: four pounds of sulphur per thousand cubic feet of space; exposure for six hours.

For destruction of rats and fleas: three pounds of sulphur per thousand cubic feet of space, exposure for six hours.

The above standard is for superstructures, partially filled storerooms, and empty holds. For cargo-laden holds and well-filled storerooms or in compartments that are packed with materials, the period of exposure should be doubled.

There are two methods in common use for generating sulphur dioxid for ship fumigation (see also page 1375). The method more generally utilized is that known as the "pot and pan" method, although formerly at a number of quarantine stations sulphur dioxid was generated by burning sulphur in a specially constructed furnace, the fumes being conducted into the holds through canvas funnels or hose. In the "pot and pan" method, ordinary Dutch ovens are used with a capacity of ten to twenty pounds of sulphur. These are placed in pans of water in elevated positions on the 'tween decks or piles of ballast and distributed about the ship. To aid combustion, approximately one-half gill of grain alcohol is added to the sulphur in each container. When all the pots are lighted, the hatches are battened down, the doors of the superstructures closed, and cracks closed by the use of paste paper. A pan of water is placed under each sulphur pot. This serves as a protection against fire and furnishes the moisture which is essential for the effectiveness of sulphur dioxid as a germicide.

For the purpose of computing the amount of sulphur or other gas to be used, a registered ton contains one hundred cubic feet. A vessel of 5,000 net tonnage would therefore contain 500,000 cubic feet of air space in the cargo holds alone. Gross tonnage of a vessel indicates the actual cubic capacity; net tonnage, the cargo-carrying capacity. The difference between the

net tonnage and the gross tonnage indicates the space taken up by the engines, fireroom and the structures above deck. In sailing vessels and freighters, therefore, there is not as much difference between the gross and the net tonnage as there is in large passenger vessels. In estimating freight-carrying capacity, forty cubic feet of merchandise is considered a ton, but this unit should not be confused with the registered tonnage, which is the basis for the measurement of the vessel.

Special Precautions in Fumigating Ships.—The various details in connection with the fumigation of vessels are quite as important as the nature of the fumigant used, and the observance of these details determines, to a considerable extent, the effectiveness of the fumigation. All possible care should be observed by the quarantine officer to see that dead space in the vessel is opened up and all practical measures should be taken to aid the diffusion of the fumigating gas; this is most essential when sulphur dioxid is used. All dunnage and loose material in the holds of a vessel that is not cargo-laden should be arranged in compact order and placed on elevated platforms to avoid rat harborage. If sulphur dioxid is generated in a furnace and led into the vessel it should be introduced at the lowest point and the hatches left open for a short period to permit the escape of air and hasten diffusion of the sulphur fumes. Pipe casings should be opened up and from one end of the vessel to the other there should be a certain number of limber boards removed so as to permit penetration of the gas into the bilges. Any planked-over space between the outer and the inner sheathing of a vessel should be freely opened. In fact, wherever there is dead space it should be opened up so that there will be free circulation of the gas. Careful attention should be given to the lifeboats, which are often infested with rats seeking water. Preferably, lifeboats should be cleaned out and flooded with water prior to fumigation. Very close attention should be given to the poop deck, which frequently contains a heterogeneous collection of litter and is generally badly rat-infested. As a rule, the engine room and fireroom do not harbor rats, but in the treatment of a plague-infected vessel they should be fumigated.

Cargo.—As a rule, the cargo of a vessel infected with pestilential disease needs no disinfection. Individual articles of the cargo, such as rags, household goods, second-hand articles, hides, wool, or food products, from infected localities may need treatment. New articles of merchandise or new manufactured goods seldom carry infection. Hides and hair need attention on account of anthrax.

Foreign Inspection Service.—To aid the quarantine officer every American consulate is required to report regularly certain facts concerning the presence and progress of epidemic diseases. Medical officers of the government are also stationed in various countries in order to supervise the sanitary condition of vessels, their cargoes and passengers leaving for the United States. This may be called preventive quarantine, for it is a distinct help in keeping out infection and facilitates trade and travel. Thus, in Italy, during a cholera epidemic, an officer of the Public Health Service stationed at Naples succeeded

in keeping that disease off vessels sailing from Naples to the United States, whereas, in several instances, vessels sailing from Naples to other ports and without sanitary supervision carried cholera.

The Pan-American Sanitary Code.—The most important advance in maritime quarantine in recent years was the adoption at Havana, Cuba, in the autumn of 1924, of the Pan-American Sanitary Code.²⁴ When this code is ratified and put in force by the signatory governments it will standardize and increase the efficiency of quarantine procedure on the Western Hemisphere.

The objects of the code are to prevent the international spread of communicable infections; to promote coöperative measures for the prevention of the introduction and spread of disease into and from the territories of the signatory governments; the standardization of the collection of morbidity and mortality statistics; the mutual interchange of information which may be of value in improving the public health and combating the disease of man; and the standardization, simplification and improvement of quarantine measures. The code must be ratified and put in force by the signatory governments. When this is done it will make a sanitary unit of the Western Hemisphere; it will be a model example of friendly coöperation between nations and a boon to preventive medicine and hygiene.

National versus State Quarantine.—All the maritime quarantine stations in this country are now controlled by the national government, and are administered by the United States Public Health Service, a bureau of the Treasury Department. The last local quarantine disappeared in 1921.

It is evident that maritime quarantine should be administered uniformly so as not to damage or favor the commerce of any port. One of the objections to local control of quarantine is that there is frequent rotation of quarantine officers due to local political changes. In addition to its other advantages, national control of quarantine insures the availability of a large corps of trained officers, whose experience in quarantinable diseases and in quarantine technic has been increased by duty in foreign countries and insular possessions where such diseases prevail. Federal control of quarantine permits greater coöperation with the other branches of the federal government such as the Immigration Service and the Customs Service. The federal government is better qualified to observe the obligations of international sanitary treaties, and to obtain reciprocal action on the part of the foreign signatories of such treaties. Finally, since the object of quarantine is the protection, not only of the local port, but of the entire country, and since the benefits derived therefrom are not merely local in effect, the expense for the maintenance of quarantine stations should be borne by the country as a whole.

INTERSTATE QUARANTINE

The regulations prepared under this act are more comprehensive with respect to quarantinable diseases than are the National Quarantine Regula-

²⁴ Copies of this Code may be had by addressing the Surgeon General, U. S. Public Health Service, Washington, D. C.

tions, since the former include not only the diseases enumerated in the maritime quarantine regulations, but also a number of communicable diseases, such as scarlet fever, typhoid, measles, whooping-cough, poliomyelitis, Rocky Mountain spotted fever, and epidemic cerebrospinal meningitis.²⁵ There is a distinct difference, however, between maritime quarantine and interstate quarantine. The provisions of the United States statutes with respect to the former give to the quarantine officer no latitude. Every ship entering from a foreign port must be inspected and the quarantine officer must certify that it is free from quarantinable disease, whereas, with respect to interstate carriers or travel, quarantine laws and regulations are enforceable only to the extent that the federal government may provide proper agencies for such enforcement. A vessel from a foreign port may not enter a port of the United States except it be provided with a certificate of discharge from quarantine (free pratique), but a common carrier is not barred from interstate travel because of the lack of any such certificates. The interstate quarantine regulations impose numerous obligations on common carriers, and potential restrictions on interstate travel, but they have not been actively enforced except under exceptional conditions. For obvious reasons, interstate travel is not subjected to the same rigid inspection as the personnel of vessels at seaboard quarantine stations.

It is evident, however, that interstate sanitary regulation is one of the important phases in which government activity can accomplish especial good, for while the government has limited authority within a state, it has practically unlimited authority so far as interstate relations are concerned. In the control of diseases by local authorities coöperation of the government is essential since it is evident that if one state should rid itself of typhoid fever, measles or tuberculosis, it would speedily become reinfected from the neighboring states unless such reintroduction were prevented through the application of interstate quarantine restrictions. Interstate quarantine regulations, however, should be enforced with the same appreciation of relative values as applies to maritime quarantine. It would be indefensible to impose restrictions on commerce and on travel for the purpose of excluding measles or typhoid from a community which itself was doing nothing to diminish the prevalence of such diseases. On the other hand, if sanitary control of local conditions by state, county or city officials ever progresses to the extent that a community has rid itself of a communicable disease, it would have the assistance of the federal government to remain clean.

By application of the provisions of interstate quarantine regulations, the national government has wide powers in influencing local sanitary reforms. While it cannot prevent the pollution of interstate streams, the Oil Pollution Act of 1924 authorizes the Secretary of War to prescribe regulations for the

²⁵ This list has been extended by the Regulation of July 30, 1918, to include: plague, cholera, typhoid fever, pulmonary tuberculosis, yellow fever, smallpox, leprosy, typhus fever, scarlet fever, diphtheria, measles, whooping-cough, poliomyelitis, Rocky Mountain spotted or tick fever, anthrax and epidemic cerebrospinal meningitis.

prevention of oil pollution ; also to investigate other forms of pollution and to report to Congress the results. By means of a proper standard for purity of water for interstate carriers, it can thus indirectly raise the standard of purity of local supplies. Furnished with adequate funds and facilities, federal health authorities can exert a tremendous influence, through the enforcement of interstate sanitary regulations toward assisting local and state authorities in achieving sanitary reforms.

SECTION V

HEREDITY

CHAPTER I

HEREDITY AND EUGENICS

Heredity may be defined as the genetic relation between successive generations. It is a condition of all organic evolution. Castle defines heredity as organic resemblance based on descent.

It is now perfectly evident that heredity is one of the fundamental factors in preventive medicine—and of first importance in sociology. It is well known to students of biology that education and environment have but a limited power to improve imperfect human protoplasm.

One of the best protections we have against diseases of body and mind is that which is inherited from our forebears. The whole problem of improving the human stock, not only from the medical view, but from the broader sociologic standpoint, is based upon the breeding of the fit and elimination of the unfit. The science of eugenics (normal genesis), therefore, assumes especial importance in preventive medicine. The physician, as well as the sanitarian, stands impotent before many deplorable conditions both in the individual and in society at large, which are inherited from our ancestors and are, therefore, incurable—but largely preventable. We are interested in educating the present generation to the facts of eugenics so that future generations may have that best of all birthrights—good human protoplasm.

The discoveries of Mendel have made it quite clear how certain characters are inherited, why certain characters skip a generation and reappear in the grandchildren, and why it is that certain defects are carried from generation to generation through many centuries.¹ The defects transmitted hereditarily are not all of equal practical importance. Thus, it makes comparatively little difference to the individual if he has a supernumerary spleen, an extra finger, or an unusual arrangement of the lobes of the liver. The defects which are of especial importance both to the individual and to succeeding generations are the defects of the nervous system. These comprise the class known as defectives. A slight defect in the structure of the brain which would be unnoticed in the lung, bone, or musculature may render the individual vicious

¹ Mendel's work has not only made it possible for us to predict with precision whether certain good or bad traits may or may not appear in the future offspring, but also to foretell with considerable precision in what proportion certain characters will appear and reappear.

instead of useful. The principal factors which are believed to start a line of defectives are injuries of various kinds, such as poisons and toxins, x-rays and accidents of all sorts. There is evidence that alcohol, lead and other poisons, syphilis and other diseases, x-rays and other agencies, antibodies and other qualities of the blood may injure the germ-plasm. The causes are discussed below.

MENTAL DEFECTIVES

Mental defectives include mainly the great class of feeble-minded, but also some delinquents, and incorrigibles. The condition results from a serious and permanent arrest of brain development, causing defective intelligence which dates from birth or early life. They rarely acquire sufficient judgment and competency to lead an independent existence in society or to successfully manage themselves in their own affairs, and therefore need guidance, supervision and even support. Defectives constitute about 1 per cent of the pupils enrolled in the elementary grades and are found in about 2 per cent of all families.

Mental defectives are the result of restricted mental development and must be regarded as children, and whatever their age must be treated as children. They are unmoral, not immoral. They need treatment, not punishment, for they are as innocent as children, not naturally bad, vicious or wicked. Parents should understand that such a child is an invalid just as if he were crippled as a result of any form of bodily disease.

The problem has magnitude and depth and far-reaching consequences, and therefore should be familiar to all students of preventive medicine and other fields of sociology. Much of it is preventable, some curable.

Causes.—*Feeble-mindedness* is due to a great variety of causes that classify themselves into (1) accidental, or (2) hereditary. Often the cause is not known.

1. *Accidental causes* include illness or weakness of the mother during pregnancy, or of the father at the time of conception; injury to the brain at birth or in early infancy. Mental defects sometimes follow an attack of measles, whooping-cough, pneumonia, meningitis and other diseases during the first six years of life. Often there is a history of normal, healthy infancy, then the occurrence of some severe illness, after which the child is less bright and is never right mentally afterwards. Syphilis of the parent is associated with about 6 per cent of the mental defects found in Massachusetts institutions for feeble-minded. Alcohol, lead and other poisons may injure the germ-plasm so as to produce defective offspring. Cretinism is caused by defective action of the thyroid gland, and mongolism is probably due to disordered endocrine action. Irradiation with Roentgen rays produces defects in experimental animals. The causes are varied and often unknown. *Accidental causes that injure the body cell are acquired and therefore not transmitted hereditarily*, but injury to the germ cell may be irreparable and transmissible.

2. *Hereditary Causes.*—The hereditary factor is evident, but has per-

haps been over-emphasized. There is a difference of opinion concerning its importance. This difference of opinion arises in part from the fact that feeble-mindedness includes a group of defects due to fundamentally different causes. It is plain that heredity is the chief cause and the distressing feature of a large group of mental defectives. Some students find that from 50 to 75 per cent and more of family trees studied show distinct evidence of the hereditary nature of the defect. The children of two feeble-minded parents are almost without exception defective. Defectives beget defectives. Whether or not the condition Mendelizes is questioned. According to Davenport, feeble-mindedness gives Mendelian expectancy; it acts as a recessive unit character. Goddard² studied 327 families in which feeble-mindedness entered. There were 144 matings of feeble-minded with feeble-minded, producing 482 children, of which all but six were feeble-minded. In these six the paternity was questioned.

Recognition.—The supposition that mentally defective persons may readily be recognized by their physical appearance, or by some outward expression, such as the movement of the eyes, is a mistake. It is also important not to confuse illiteracy with mental deficiency.

The defective individual is very easily recognized when the condition is well marked. The mental abnormality is usually accompanied by prominent physical defects known as the stigmata of degeneration (Lombroso and Weismann). The typical degenerate is of poor bodily development; the brain is smaller than normal, with convolutions less abundant, and less fully formed. He has a degraded physiognomy, lacks capacity for sustained attention or for prolonged thought, is cunning rather than intelligent, deficient in moral sense—in all points resembling the stigmata of the lower, less developed races of our species. The whole gives the impression of a reversion to a lower type. An unfortunate side to this problem is that degenerates and defectives generally are not only irresponsible morally, but are very prolific. They lack self-control and have abnormal sexual appetites. The defects are transmitted, and thus feeble-mindedness, social inadequacy and some psychoses are propagated.

Symptoms.—The symptoms of mental defect vary according to the degree of defect. In *extreme cases* evidence appears in early infancy. As the child grows older, his teeth may not appear at the usual age or he may learn to walk late and with an awkward, shambling gait, or he may be late in using his hands, or his untidy habits may persist for a long time. He is very apt not to talk until he is three or more years old. In general, he remains a baby for a long time. In *less severe cases* the defect is commonly not recognized until the child is found to be unable to learn in school at the usual school age and cannot be promoted from year to year like other children. He may limp through the first and second grades and be unable to go beyond the third, especially in arithmetic. He shows his defects in other ways. He may

² *Feeble-mindedness: Its Causes and Consequences*, New York, 1914.

not be able to get on with other children in games and sports. He is often teased and picked on by playmates of his own age who do not regard him as an equal, so he usually associates with children younger than himself. He is, as a rule, easily influenced. He shows poor judgment and reasoning power. In general he finds it difficult to adjust himself. He is not able to meet new situations. As he grows older, he is apt to be led into mischief as he finds it hard to resist temptation. Feeble-minded persons, especially the girls, are unusually susceptible to sex temptation at puberty or adolescence. If neglected or allowed to associate with evil companions, they are more likely than normal persons to acquire immoral or vicious habits and tendencies. Some defectives seem innately vicious and troublesome from early childhood, but the majority seem about as amenable to proper associations and proper bringing up as do normal children. Mental defectives are apt to be left-handed. In schools for mental defectives, 18.2 per cent are left-handed, against 7.3 per cent in ordinary schools.

Cretinism.—Cretinism is discussed under iodine deficiency, page 1042.

Mongolism.—The Mongolian³ type is described by Shuttleworth as follows: "The skull is a short oval, the transverse and longitudinal diameters approximating, while there is a tendency to parallelism of the frontal and occipital planes. Children of this type have a skin coarse in epidermis, if not furfuraceous; many have sore eyelids, some fissured lips; but one of the most striking peculiarities is the state of the tongue, which is transversely fissured, and has hypertrophied papillæ. Dr. John Thomson states that in the early weeks of life the tongue is normal: between the third and ninth months the papillæ get enlarged, while during the third and fourth years the transverse fissures appear. This latter peculiarity is possibly due to tongue sucking, which is so common in this type of defective, acting on an abnormally vulnerable mucous membrane. Many of them have almond-shaped eyes, obliquely set, and this feature, with the squat nose, epicanthic fold, and wiry hair, gives the Mongol aspect from which they derive their name. The hands are usually broad and the fingers short, and often the little finger is incurved. The feet also are characteristically clumsy, with a marked cleft between the big toe and the next one. Laxity of the joints is a marked feature. There is no reason to believe that they are essentially *unfinished* children, and that their peculiar appearance is really that of a phase of fetal life."

Mongolism is definite and easily recognized and should be diagnosed early, usually before the sixth or eighth month. This is important for the sake of the parents. Goddard states that a diagnosis of Mongolism settles the following points: (1) The condition is not hereditary. (2) It is more frequent in the better families. (3) The child will never develop beyond the seven-year mentality, and the great majority have a mentality of almost exactly four years. Consequently (4) they must always be cared for. Unpleasant as the

³ This type has no connection with the Mongolian race. It is so named on account of the resemblance to that type of countenance.

duty is, yet in the long run it is a kindness to the parents to inform them of such an outlook.

The Feeble-minded.—*Idiots, Imbeciles, Morons.*—Feeble-mindedness is characterized by a lack of normal development of the *intelligence*. Feeble-mindedness always appears in early childhood, while insanity occurs later in life in a person who has usually enjoyed good mental health hitherto. The feeble-minded are divided into three groups: (1) *idiots*, which comprise those whose mental age does not advance as far as normal children of two years; (2) *imbeciles*, whose minds remain somewhere between two and seven years; and (3) *morons*, or fools, whose intelligence does not get farther than the seventh to the twelfth year of normality. All shades of mental inferiority between the moron and the normal occur. In addition, there is a group above the moron whose conduct shows that they are not normal. These are sometimes called *dull normal*, or *backward*. Of these three groups the moron is perhaps the most important from every standpoint, for this group of defectives propagates itself, and the crop is large. The high-grade defective or moron is the most troublesome, partly because he is not easily recognized as defective, and partly because he has sufficient mentality to go about by himself and get into all kinds of mischief, either on his own account or led on by someone else. The male morons grow up into paupers, drinkers, hoboes and ne'er-do-wells and fill our hospitals and asylums. The female morons grow up into irresponsible women who replenish the ranks of the prostitutes and other defectives.

The Royal Commission of England reports that in that country the feeble-minded are increasing at twice the rate of the general population. Butler of Indiana states that feeble-mindedness produces more pauperism, degeneracy, and crime than any other source; that it touches every form of charitable activity; that it is felt in every part of the state, and affects, in some way, all the people, and that its cost is beyond comprehension. The Committee of Visitors of the State Charities of New York in 1914 reported that there were in that state 32,000 feeble-minded persons. Of these, 4,900 were provided for in institutions especially designed for their care, and 4,500 in other institutions, leaving at large 22,600. It has been estimated that of the 32,000 feeble-minded, 10,000 are girls and women of childbearing age, 1,750 of whom are cared for in institutions designed for the care of such persons, and 1,625 are confined in reformatories, prisons, and almshouses, leaving about 7,000 at large in the community. Goddard estimates that in the way of spreading disease, immorality, and increasing the stock of the feeble-minded, a girl or a woman of this class, of childbearing age, is three times as great a menace to the community as is a feeble-minded boy or man. It is estimated that the feeble-minded constitute a disproportionate percentage of the inmates of our prisons, 15 to 30 per cent of the almshouses, and a still larger percentage of prostitutes.

Upwards of 2 per cent of juveniles are so defective mentally as to be incapable of self-support and self-direction, and are largely irresponsible.

The Measurement of Intelligence—The Intelligence Quotient.—Intelligence can be accurately measured and a very close estimate made of mental development. The intelligence of a child at a given time is expressed in terms of mental age. The mental age of a feeble-minded child is always less than his real or chronological age. Thus, a defective child of twelve may have a mental age of only nine, six, three or even less.

The ratio between the mental age and the real age is called the *intelligence quotient*, or the I.Q., and it tells just how backward the child really is compared to a normal child. Thus a normal child of twelve years with a mental age of twelve has an I.Q. of 100. A defective child of twelve with a mental age of eight has an I.Q. of 66. A defective child of twelve with a mental age of six has an I.Q. of 50, and so forth.

Albert Binet, in 1908, first suggested that it was possible to take the mental measure of normal and defective mentality. He devised a measure scale for intelligence. He deduced from his experience the following formula: "Children who are under nine years of age and show a backwardness of more than two years, are probably mental defectives to the extent of being actually feeble-minded; those who are nine years or more and show a backwardness of more than three years are also feeble-minded." Goddard states that Binet's formula is well within the truth. The "Binet-Simon" test consisted of a series of fifty-four tests for children between the ages of three and twelve years, a certain number of questions being assigned to each age group. Both the method and the technic have been greatly modified and developed, from which the following has been determined:

An intelligence quotient of less than 70 generally means that the child is feeble-minded. Morons have an I.Q. between 50 and 70; imbeciles, between 25 and 49; and idiots less than 25. If the I.Q. is between 70 and 80 the case may be a doubtful one and is frequently called "borderline." About 88 per cent of children respond normally; some 1.5 per cent are super-bright with quotients over 100; about 9 per cent are subnormal or borderline; and about 1.5 per cent are hopelessly defective.

The school-grade work that a feeble-minded child will probably be able to do at the various mental-age levels is as follows:

I. Q.	Adult Mental Age	Final School Grade
30	5	kindergarten
40	6	first grade
45	7	second grade
50	8	second or third grade
60	9	third or fourth grade
65	10	fourth or fifth grade
70	10-11	fifth grade

Meaning and Limitations of the Intelligence Test.—The I.Q. is a measure of only one quality of the mind, namely intelligence. We have no precise definition of intelligence. Furthermore, a good mental test measures the keenness and brightness of one's mental development at the moment, but not

the force or skill or patience with which one is going to use it or the edge it may acquire. Above all, the I.Q. is not a measure of character, coöperation, personality or behavior. While the I.Q. is a good index of the measure of a child in its school work, it does not necessarily correlate with success in life. No intelligence test measures persistence, courage, cheerfulness, humility, patience, morality or interest, and these elements of character determine usefulness in the world's work. In other words, the super-bright may be vicious, and the dull may contribute to the creative progress of the race. Special tests have been devised to determine temperamental traits, such as temperament, aggressiveness and creative imagination.⁴ Trade tests include mental alertness, aptitude, social relations and interest.

The general mental characteristics that can be determined without special psychological tests are (1) lack of control emotions and impulses, (2) inability to adapt to new conditions, (3) inability to generalize from experiences or to deal with abstractions, (4) general lack of good judgment and good sense.

Finally, it must be remembered that mental tests have little significance for adults; in fact, satisfactory tests have not been devised for persons after the fourteenth year.

Training of the Feeble-minded Child.—The *mental age* tells how intelligent a defective child is at the time of examination. The *intelligence quotient* tells how bright he is compared to a normal child and how intelligent he will be when he reaches the age of fifteen or the adult level. The mother or the teacher must disregard the child's real age and base instruction and training on his mental age. A defective child with a mental age of less than five years can be well cared for and trained in a good home. If the child becomes an intolerable burden in the home from his noisy, destructive and untidy habits, he can be well cared for in the institutional schools which are now found in nearly every state.

School Work of the Feeble-minded Child.—It is no use for a defective child with a mental age of less than five years to go to school. For the defective child with a mental age of six or over, the public schools in most cities and large towns have special classes. These children may do well in regular school classes if they are understood by the teacher and work given suited to their mental age, and if the teacher is satisfied with the best they can do. The opportunity here is largely a matter of social training; that is, training in inhibition, in emotional control, in conforming to the amenities of life, and in learning to do as other children do. There is no special pedagogy for feeble-minded children. The principles are the same as for the normal child. But conditions are different, the progress is much slower, and to keep up the interest of the defective child we must use much graphic school material.

Social, moral and emotional discipline and example are more important for the defective child than strictly scholastic training. He will learn as much

⁴Helen H. Dolan, "Developments in the Field of Mental Testing," *U. S. Pub. Health Rep.*, 1924, 39: 2505.

from other children as he does from the teacher. He must be protected from bad knowledge and bad associates. This is especially necessary at puberty and adolescence. The girls in particular must be protected from sex temptation and experience at that time.

The "bringing up" of a defective shapes and modifies his traits of disposition, personality and character, and these are more important than his intelligence in determining his adult conduct and behavior. We cannot increase the defective child's intelligence, but we can do much to strengthen desirable traits and to repress undesirable ones. Thus we can make him more affectionate, more anxious to please, to do and say the right thing, more obedient, self-reliant, persistent, honest, truthful, and so forth. We can help him to control his emotions. His self-respect must be preserved by not asking him to do things that he is not able to do.

Industrial and manual training from an early age will do much to give him self-respect and furnish him with interests and resources as well as prepare him to become a useful and perhaps self-supporting man. Defective children who succeed in life do so because they become capable of doing worth while work with their hands. This is really the end and aim of all training with such children.⁵

Degenerate Families.—A careful study has been made of the records of several families in which the mating of unfit individuals has begotten a swarm of unfit descendants. Some of these broods have such an infamous record that they are known throughout the world. The published account of these tribes is a dreary history of degenerate mating with degenerate, generation after generation. Here we have inbreeding of the most intense kind, incest of the highest degree. These people have been given chances, have been removed from their environment, taken to reputable homes with good influences, educated according to their capacities and have remained degenerate. The explanation is clear; homozygous strains with a bad heredity.

Significance and Caution.—The significance of the histories of these consistently degenerate families has been questioned. Perhaps their importance has been overemphasized. It must be noted that the data is obtained long after most of them are dead and gone. The information often rests upon doubtful evidence of notoriously unreliable persons. The paternity is not always clear. Furthermore, the facts are sometimes collected by unskilled assistants and edited by credulous investigators. It is difficult enough to get genealogical records of the best families.

These histories are extreme, unusual and exceptional. They are examples of low cultural level and are not to be taken as representing a usual or common state of affairs. Nevertheless, despite errors, they give a fair picture of one extreme and illustrate the fact of the inheritance of certain types of mental defects. The following illustrations are given with this understanding concerning their significance and caution concerning their reliability.

⁵ W. E. Fernald, *Men. Hyg.*, 1924, 8: 964.

The Jukes Family.—One of the best known families of this type is the so-called Jukes family of New York State investigated by Dugdale.⁶ This is an inbred family whose record of pauperism, prostitution and crime has been traced for six generations. It started with the five daughters of a lazy and irresponsible fisherman born in 1720. In five generations the descendants of Jukes numbered about 1,200 persons, including nearly 200 who married into it. The histories of 540 of these are well-known, and about 500 more are partly known. Some 300 died in infancy. Of the remaining 900, 310 were professional paupers living in almshouses (a total of 2,300 years); 440 were physically wrecked by their own diseased wickedness; more than half of the women were prostitutes; 130 were convicted criminals; 60 were habitual thieves; 7 were murderers. Not one had even a common school education; only 20 learned a trade, and 10 of these learned it in state's prison. The descendants of Jukes in five generations have cost New York State over one million and a quarter dollars, and the cost is still going on.

The Family Zero.—Probably the most complete family history of this kind ever worked out is that of the "Familie Zero," a Swiss family whose pedigree has been studied by Jörger.⁷ In the seventeenth century this family divided into three lines. Two of these have ever since remained valued and highly respected families, while the third has descended to the depths. This third line was established by a man who was himself the result of two generations of intermarriage, the second tainted with insanity. He was of a roving disposition, and in the Valla Fontana found an Italian vagrant wife of vicious character. Their son inherited fully the parental traits and himself married a member of a German vagabond family—Marcus. This marriage sealed the fate of their hundreds of descendants. The pair had seven children, all characterized by vagabondage, thievery, drunkenness, mental and physical defects, and immorality. This is the Zero Clan, a name indicating their value to the world. How much of this is due to heredity and how much to environment will be discussed presently.

Other Degenerate Families.—There is the "Tribe of Ishmael," a race of indigent vagrants since 1790, consistent in their ways of life no matter what their surroundings.⁸

There is the Nam family, descendants of an Indian-white mixture, and characterized by vagabondage and stupidity.⁹

Another interesting example of the same type has been described by Poellmann—a distressing example of illegitimacy and pauperism. This family was established by two daughters of a woman drunkard who in five or six generations produced, all told, 834 descendants. The histories of 709 of these are known. Of the 709, 107 were of illegitimate birth, 64 were inmates of almshouses, 162 were professional beggars, 164 were prostitutes, and 17

⁶ *The Jukes*, 1877, 4th Edition, 1910.

⁷ *Arch. f. Rassen- u. Gesellsch.-Biol.*, 1905, 2: 494.

⁸ "The Tribe of Ishmael: A Study in Social Degeneration," *Proc. Fifteenth Nat. Conf. Char. & Cor.*, 1888.

⁹ "The Nam Family," *Mem. 2, Eugenics Record Office, Cold Spring Harbor*, 1912.

procurers, 76 had served sentences in prison, aggregating 116 years, 7 were condemned for murder.

The Kallikak Family.—Henry H. Goddard¹⁰ has investigated and compiled the results of his work on the heredity of a most remarkable family, the Kallikak family. During the Revolutionary days, the first Martin Kallikak (the name is fictitious), descended from a long line of good English ancestry, took advantage of a feeble-minded girl. The result of their indulgence was a feeble-minded son. This son married a normal woman. They in turn produced five feeble-minded and two normal children. Practically all of the descendants of these defectives have been traced, as well as those of the two normals. The tragic story follows:

From both normal and defective descendants of this union came a long line of defective stock. There were 480 in all. Of these 36 were illegitimate, 33 sexually immoral, 24 confirmed alcoholics, and 3 epileptics. Eighty-two died in infancy, 3 were criminal, 8 kept houses of ill fame, and 143 were distinctly feeble-minded. Only 46 were found who were apparently normal. The rest are unknown or doubtful. But the scion of the good family who started this long line of delinquent and defective progeny is also responsible for a strain of an entirely different character. After the Revolutionary War was over, he married a Quaker girl of good ancestry and settled down to live a respectable life after the traditions of his forefathers. From this legal union with a normal woman there have been 496 descendants. All of these except two have been of normal mentality. The exceptions were cases of insanity, presumably inherited through marriage with an outside strain in which there was a constitutional psychopathic tendency. In all the 496 there is not an instance of feeble-mindedness. The offspring descended from this side of the house have universally occupied positions in the upper walks of life. They have never been criminals or ne'er-do-wells. On the other hand, there has not been a single instance of exceptional ability among the descendants of the first Martin Kallikak and the feeble-minded girl. Most of these descendants have failed to rise above the dead level of mediocrity; indeed, most of them have fallen far below even this minimum standard.

The fact that the descendants of both the normal and the feeble-minded mother have been traced and studied in every conceivable environment, and that the respective strains have always been true to type, tends to confirm the belief that heredity has been the determining factor in the formation of their respective characters. In the cities the descendants of the legal marriage with the normal woman are physicians, lawyers, and prominent business men, while the descendants of the feeble-minded mother are almost invariably found in the slums. In the rural districts the descendants of the normal mother and her consort are wealthy and influential farmers, while the others never rise

¹⁰ *The Kallikak Family, a Study in the Heredity of Feeble-Mindedness*, Macmillan Company, 1912.

See also "The Story of the Dack Family: A Study in Eugenics," Finlayson, *Bull. No. 15*, Eugenics Record Office.

above the rank of farm laborers and shiftless men and women, who are unable to subsist without the aid of charity. Many representatives of the defective branch are inmates of almshouses, while there are no paupers at all among the normal descendants.

In many ways this study of Goddard's far outweighs in importance the famous comparison by Winship of the Jukes and Edwards families. In that case the simple fact was demonstrated that a good family like that of the illustrious Jonathan Edwards had given rise to innumerable examples of the highest intellectual and moral worth, whereas the criminal Jukes for seven

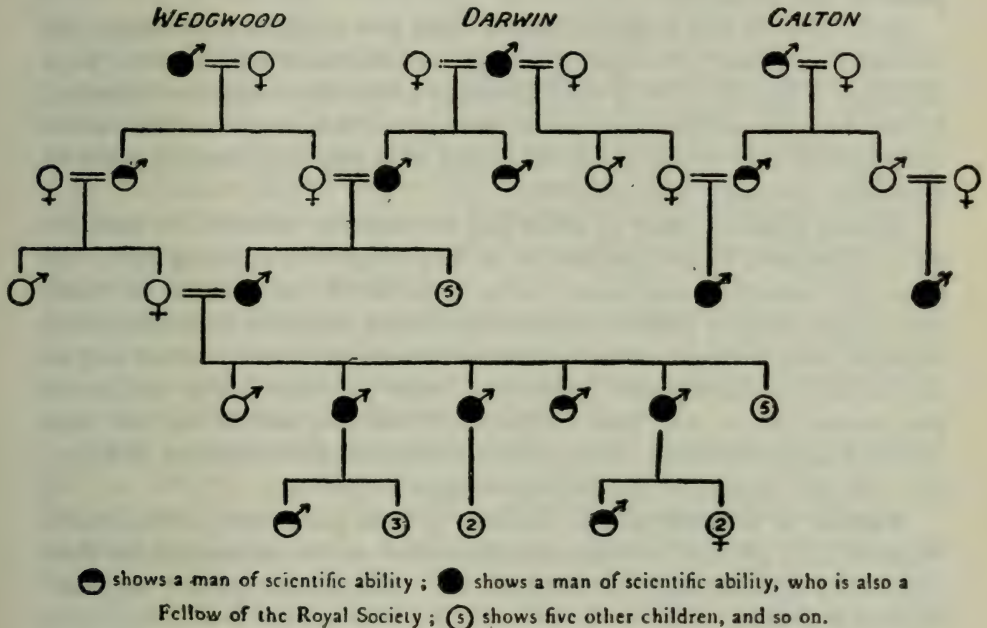


FIG. 47.—HISTORY (CONDENSED AND INCOMPLETE) OF THREE MARKEDLY ABLE FAMILIES (After Whentham) (Kellicott).

generations contributed nothing to the common good and cost the state of New York large sums of money. But the Jukes family and the Edwards family had no ancestor in common. Their environment was totally different and they lived in entirely separate communities. Although from sociologic and economic points of view the history of the Jukes family and its comparison with that of the family of Jonathan Edwards has great value, it is of but scant scientific importance as compared with that of the Kallikak family, for here a natural object-lesson in eugenics shows unmistakably the manner in which after-coming generations from a given mating receive the characteristics of the dominant strain, which in the elder (illegitimate) Kallikak line was the inferior strain, with only a debased and enfeebled heritage to hand on.¹¹

¹¹ *J. Am. M. Ass.*, 1912, 59: 1545.

In contrast to these we have the descendants of the families of Wedgwood, Darwin, and Galton, the Edwards family and the Ward family. These noted families contained a large number of statesmen, jurists, professors, physicians, officers in the army and navy, prominent authors and writers, and occasionally men and women of genius. They show a long line of usefulness in every department of social progress, and not one of them ever has been convicted of a crime.

The family of Bach is an illustration of the inheritance of musical talent. This family produced twenty eminent composers and twice as many who possessed marked ability.

How much of this is due to heredity and how much to environment are debatable questions. Students of biology are convinced that heredity plays the major rôle in the lives of the individuals in the above-mentioned families. In how far such extreme instances as those given above represent the rule or exceptions, it will require much additional data and long years of study to determine (see pages 438 and 573).

Bearing upon the effect of crime and the mentally defective, we have the study of Bernard Glueck, psychiatrist at Sing Sing Prison, during 1917. Of some 600 consecutive admissions during a period of nine months, he found that 59 per cent, in addition to showing various disorders of conduct, also exhibited some form of nervous or mental abnormality, which in one way or another had conditioned their behavior. Twelve per cent of these sick people were insane; 28 per cent were intellectually defective, and 19 per cent were classified as psychopathic. Among the defectives we find recidivism in 81 per cent, the rate among the psychopaths rising to 87 per cent.

Number of Defectives.—On January 1, 1923, there were in the United States 893,637 persons in institutions dependent on the community for their support, or approximately one in every 100. The official figures¹² are summarized as follows:

<i>Institutions</i>	<i>Number of Inmates</i>
Hospitals for mental diseases	290,457
Psychopathic wards of general hospitals	1,842
Institutions for feeble-minded	46,722
Institutions for epileptics	9,153
Total	348,174
Federal penitentiaries	4,670
State prisons, reformatories, etc.	77,340
County and city jails, workhouses, etc.	26,929
Total	108,939
Inmates of almshouses	78,090
Institutions for adults and children, as: Homes for adults and children, child-placing agencies, day nurseries, homes for wayward women and unmarried mothers, private homes for delinquent women and girls, etc.	329,091
Institutions for juvenile delinquents	29,343
Grand Total	893,637

¹² Collected by the Department of Commerce, *U. S. Pub. Health Rep.*, 1924, 39: 1804.

Salmon estimates that there are about four mental defectives per thousand of population. Among school children about 1 per cent are mentally deficient. The incidence among males is higher than among females, at least in institutions.

Goddard states that there are about 400,000 feeble-minded in the United States. Further, that two-thirds of them owe their condition to heredity, and that they are propagating their kind at a rate two to six times as fast as the good stock. However, they have a higher mortality, especially in infancy, which helps keep the balance. It is estimated that the feeble-minded constitute 25 to 40 per cent of the inmates of our prisons, 15 to 30 per cent of the inmates of almshouses, and a still larger percentage of prostitutes.

We have to segregate and support about 1 per cent of the population. Many of these are not only dependent but dangerous. A new plague of such magnitude, largely preventable and costing a vast treasure, would attract universal attention. One-sixth of the total appropriation of the State of Massachusetts is for the maintenance of insane and feeble-minded in institutions. We have become so used to crime, disease, and degeneracy that we take them as necessary evils. "That many of them were so in the world's ignorance is granted; that they must remain so is denied."

Statistical studies seem to indicate a rapid increase of the unfit, defective, insane, criminal; on the other hand, a slow increase, or even a decrease (?), of the fit, normal, or gifted stocks. It is plain to the student of eugenics how such conditions account for the rise and fall of nations.

There were 91,959 known insane in the United States in 1880, a rate of 183 per 100,000. In 1903 the figures rose to 180,000, a rate of 225 per 100,000. On January 1, 1920, there were 232,680 patients with mental disease in hospitals for the insane in this country; including those on parole the number would be over 250,000. The figures for 1923 are given in the table, page 562. These figures must not be taken as an index of the increase of insanity in the population at large—for institutional care has been growing much more popular during the past decade, especially since more humane methods have been adopted. Further, the classification of insanity now includes many cases that were formerly little noticed.¹³ This subject is fully discussed in Section II.

¹³ A special census of the insane confined in institutions was taken by the Bureau of the Census in 1910, and it was found that 187,454 patients were confined in hospitals for the insane in the continental United States.

While the population of the United States increased about 11 per cent in the interval between 1904 and 1910, the population in insane asylums increased about 25 per cent. The number of insane in asylums per 100,000 population increased from 186.2 in 1904 to 203.8 in 1910. The number of persons annually committed to hospitals for the insane per 100,000 population increased from 61.5 in 1904 to 65.9 in 1910. If these ratios are accepted as representing insanity rates, it would appear that the number of persons becoming insane, in a community comprising 100,000 persons, was greater by 4.4 in 1910 than it was in 1904. It must be remembered, however, that these figures include only the insane who are committed to hospitals. As to the number of cases of insanity not resulting in commitments to hospitals the census has no data. It is entirely possible that the increase in the number of commitments per 100,000 population is not due to any considerable degree to an increased prevalence of

PREVENTION

Prevention of Propagation of Defectives.—Four methods have been proposed to prevent the propagation of defectives: (1) education; (2) legislation; (3) segregation; (4) surgery.

Education.—Education directed toward the defective is a failure, for he is incapable of profiting by the lessons and lacks self-control. The education of the better class of the community is indirectly helpful in calling attention to the situation as being largely preventable, and to the necessity and means for controlling it.

Restrictive Legislation.—Restrictive legislation is a praiseworthy effort, but has signally failed as a preventive measure, for the evident reason that it only adds illegitimacy to degeneracy, and thus the children enter on life's battle doubly handicapped. Most of the states now have laws providing that within the bounds of the state no marriage shall be permitted, either party to which is epileptic, imbecile, feeble-minded, or afflicted with insanity, unless the woman be over forty-five. The so-called "Eugenic Marriage Law," requiring a marriage certificate attesting freedom from venereal disease, was first passed by Wisconsin in 1913 and subsequently by Oregon, North Dakota and Alabama in 1919, North Carolina and Wyoming in 1921, and Louisiana in 1924. Proposals of the same general character have been voted down in 37 state legislatures since 1901.¹⁴ In practical operation these laws provide little actual protection to wives. While such laws are not ideal, they serve the purpose of education and bring to everyone who contemplates marriage a realization of the importance and hazards of the problems involved.

Segregation.—Segregation would be an ideal and humane method of isolating those who are incapable of having normal offspring, but the isolation of all degenerates and defectives would be an enormous and impractical task. Further, the great difficulty is to detect the unfit individual who starts a strain of defectives and degenerates. It is evidently a hopeless task to know where to draw the line between the fit and the unfit, so that for the present we must be satisfied to enforce restrictive measures upon only those who are evident and well-marked examples. Insane asylums, homes for epileptics, reformatory schools, as well as special hospitals and institutions for advanced cases must not be regarded as preventive measures in the true sense, for such segregation provides care and comfort as a terminal measure; that is, it is usually a last resort. Frequently defectives propagate their kind before and sometimes after they are interned in such institutions. Preference, nevertheless, should be given to women of childbearing age in institutional care.

insanity, but simply to the extension of this method of caring for the insane. It is a change which might result from an increase in the number of institutions of this class and from the increasing disposition on the part of the public to resort to such institutions. In this connection it may be noted that the number of institutions for the insane reported by the census increased from 328 in 1904 to 372 in 1910, an increase of about 13 per cent. The average number of inmates per institution increased from 458 in 1904 to 504 in 1910.

¹⁴ Fred S. Hall, *Medical Certification for Marriage*, Russell Sage Foundation.

Although segregation of all persons with higher types of mental defect is never likely to be accomplished, there is hope of devising a plan of registration and guardianship in the community which will protect a great many and prevent their marriage.¹⁵ (See program below.)

Surgery.—Sterilization is an effective means of controlling the propagation of defectives. This is done either by severing the vas deferens in the male or the fallopian tube in the female. It may also be accomplished by exposure to Roentgen rays. The operation of vasectomy consists in ligating and excising a small part of the vas deferens. This distal end of the duct is tied, but the testicular end is left open in order to avoid cystic degeneration of the testicles. The operation is very simple and easy to perform; it takes about three minutes and does not need an anesthetic. There is no diminution of the sexual power or pleasure. The discharge at the orgasm is but slightly diminished. In the female, the severance of the fallopian tube (salpingectomy) is a more formidable operation. It is necessary to make a median incision exposing the peritoneal cavity. The tubes are ligated near the uterus and severed beyond the ligature.

Following the lead of Indiana, eleven states passed laws providing for the sterilization of defectives. The operation was performed at the Indiana Reformatory, but in 1921 the law was declared unconstitutional.

Opinions vary greatly concerning the proper use of sterilizing criminals, insane, degenerates, and defectives generally. There is no doubt concerning its effectiveness.

Sterilization is a measure which contains great potential possibilities for abuse and injustice. The difficulty consists in determining upon whom the operation shall be done and when. Even in perfectly clear cases, such as the insane, the epileptic, or the high-grade degenerate, the harm has often been done before the operation is decided upon.

The Salvage of the Backward Child.—*A Program.*—Feeble-mindedness is not an entity to be dealt with in a routine way, but is an infinitely complex problem. No two defective persons are exactly alike. The causes are multiple. What is good for one may be bad for another. No routine procedure will meet the needs of this highly differentiated group. Feeble-minded persons may be male or female; young or old, idiots, imbeciles or morons; well-behaved or vicious; industrious or idle; from good homes or from bad. They may live in the city or in the country, in a good neighborhood or a bad one, or they may have good, bad or indifferent heredity.

The vast majority are able to adjust themselves at home as they have always done in the past. They should be looked for and recognized in their school life. For this purpose school and traveling psychiatric clinics with better training in special classes should be provided. Much of the present

¹⁵ Fernald, "What is Practicable in the Way of Prevention of Mental Defect," a pamphlet distributed by the National Committee for Mental Hygiene, 1915. Also, Salmon, "Outlines of a State Policy for Dealing with Mental Deficiency," *Med. Rec.*, April 17, 1915.

scholastic discipline should be supplemented by industrial training. The schools must provide special social supervision. The bad defective individuals and the neglected ones should be sent to institutions before they come to grief. After the school age it is the duty of the state to provide extramural supervision for all neglected or badly behaved defectives. The parole system under guardianship is practical, but the price of liberty should be good behavior. The big brother movement has solved and saved many a case from "the gang."

In order to carry out this program the state should establish a department of mental hygiene, with psychiatrists, psychologists and social workers. The keynote of success is to recognize all defective children at an *early age*. They must then be registered, diagnosed, treated and watched throughout life. The clinics must reach the smallest and most remote towns. The traveling teacher visits and helps the teacher and parents. It must be emphasized that the problem is largely an individual one. Finally, stress should be laid upon the importance and necessity of scientific study of the causes of feeble-mindedness with the hope of furnishing knowledge which will prevent the occurrence of certain types. This subject is also discussed in the section on Mental Hygiene.

EUGENICS

Eugenics has been defined as "the science of being well born." According to Galton, "eugenics is the study of the agencies under social control that may improve or impair the racial qualities of future generations either physically or mentally."

The aim of eugenics is to increase the number of best specimens in each class; that done, leave them to work out their common civilization in their own way. It also aims to leave a good heritage to the next generation and to repress the propagation of the vicious and defective classes.

The success of eugenics depends almost entirely upon our knowledge of heredity and sociology. Therefore, the fundamental principles of heredity should be familiar to all students of preventive medicine.

The present movement started in 1865 when Francis Galton showed that mental qualities are inherited, just as are physical qualities, and pointed out that this opened a way to an improvement of the race in all respects. Galton's work on "Hereditary Genius," published in 1869, was the first real study of the inheritance of mental capacity and again emphasized the possibility and desirability of improving the natural qualities of the human race. The word "eugenics" was coined in 1883 in his "Inquiries Into the Human Faculty."¹⁶

Not only physical appearance, but mental and emotional traits and behavior are transmitted hereditarily. Musicians are born, not made; hence the importance in education of finding and developing natural aptitudes.

¹⁶ See also W. E. Castle, *Genetics and Eugenics*, Harvard University Press, Cambridge, 1924; *Eugenics, Genetics and the Family*, Vol. I; and *Eugenics in Race and State*, Vol. II, issued by the 2nd International Congress on Eugenics, Williams and Wilkins Co., Baltimore, 1922.

There is no doubt concerning the desirability of breeding better human stock, but how this may be accomplished practically is a difficult question. The program of the eugenist is perplexing and complicated. To follow the theoretical extremists would require a social revolution—a change from the present method of haphazard mating. The threshold of the subject has scarcely been passed, and we must bear in mind that some of the striking men of genius from whom the world has greatly profited have been individuals whom the student of genetics would regard as degenerates or defectives. The fact cannot be neglected that the brightest examples of inherent ability have come and will come from chance mating in the general population, the common people so-called, because of the variability there existent. The upper and gifted families tend to run out and are recruited from “below.” Eugenics does not mean free love, nor does the eugenist recommend Burbanking the human race to produce great physical strength, beauty, endurance, mental or moral power. Two points only in the program are perfectly clear; (1) a check upon the propagation of the crop of defectives, and (2) the encouragement of parenthood with good hereditary traits. We must take thought of the child of to-morrow as well as the man of to-day.

The known facts of heredity and the study of eugenics make us examine more critically some of the directions which preventive medicine, including philanthropy and social uplift, has taken. We must now ask ourselves the question whether it would not be better for the future generations if we helped the fit instead of concentrating all our attention and sympathies upon the weakling and the unfit. These are problems raised by Galton, who questions whether some of our philanthropic efforts are well balanced and well directed.

It is important to recognize that many diseases are due to defects of society. Sociology, therefore, must come to the aid of preventive medicine. Crime is often a defect that needs treatment rather than punishment. Poverty is one of the chief causes of diseases, and ignorance one of its first allies. Hence, constructive reforms must aim for social justice, education, and the teachings of eugenics.

According to the teachings of genetics, all men are not created free and equal; but bound by their protoplasmic make-up and unequal in their powers and responsibilities.

It is evidently now of great importance to collect a large number of pedigrees, in which the data shall be stated with scientific exactness and in minute detail. Such a mass of facts may then be studied in the light of science in order to determine in how far the laws of heredity apply to human characters. This is being done by the Eugenics Record Office at Cold Spring Harbor, New York, under the patronage of the Carnegie Institution.

In brief, then, the aim of eugenics is through heredity to give the individual the greatest of all birthrights, good human protoplasm—and to eliminate, as far as may be possible, bad human protoplasm.

Inbreeding may be hazardous for reasons that are well understood. The marriage of cousins will be evidently hazardous if the objectionable hereditary

characters are dominant, for in this case the danger is plain; if the characters are recessive the danger is specially tragic, because of unexpected outcroppings in the offspring. Inbreeding tends to secure homozygous combinations, and this brings to the surface latent or hidden recessive characters. Cross breeding brings together differentiated gametes which, reacting on each other, produce offspring of greater vigor. On the other hand, continued cross breeding only tends to hide inherent defects, not to exterminate them; inbreeding only tends to bring them to the surface, not to create them. It is not, therefore, correct to ascribe to inbreeding by intermarriage the creation of bad racial traits, but only their manifestation. Further, a racial stock which maintains a high standard of excellence under inbreeding is certainly one of great vigor and free from inherent defects (Castle).

On the other hand, contrary to the former belief, some students of eugenics think that inbreeding is not inimical to the welfare of the offspring. As early as 1887, Huth¹⁷ collected all the available data and came to this conclusion; Crawley¹⁸ likewise; while Hutchinson,¹⁹ also East and Jones²⁰ feel that the only injury due to inbreeding comes from the inheritance received and is not the consequence of consanguinity. The results depend upon the freedom of the stock from undesirable hereditary traits.

Owing to the existence of serious recessive traits there is objection to indiscriminate, irrational, intensive inbreeding in man; yet inbreeding of sound stock is the surest means of establishing families which as a whole are of high value to the community.

The influence of isolation and the results of *consanguineous marriages* are well brought out when we study certain localities. Thus, consanguinity on Martha's Vineyard results in 11 per cent deaf mutes and a number of hermaphrodites; in Point Judith, 13 per cent idiocy and 7 per cent insanity; in an island off the Maine coast the consequence is "intellectual dullness"; in Block Island, loss of fecundity; in some of the "Banks" off the coast of North Carolina suspiciousness and an inability to pass beyond the third or fourth grade of school; in a peninsula on the east coast of Chesapeake Bay the defect is dwarfness of stature; in George Island and Abaco (Bahama Islands) it is idiocy and blindness (G. A. Penrose, 1905). There is thus no one trait that results from the marriage of kin; the result is determined by the specific defect in the germ-plasm of the common ancestor. For examples of the dreadful effects of certain kinds of inbreeding of the most intense sorts, read the dreary stories of defective families, page 558.

PRINCIPLES OF HEREDITY

For a clearer understanding of the hereditary transmission of disease, malformations, and defects, it is necessary to have an understanding of the

¹⁷ *The Marriage of Near Kin*, New York, Longmans, Green & Co., 1887.

¹⁸ *Exogamy and the Mating of First Cousins*, etc., Oxford, Clarendon Press, 1907.

¹⁹ "Marriage of First Cousins," *Polyclin.*, 2, 1900.

²⁰ *Inbreeding and Outbreeding*, Philadelphia, J. B. Lippincott Co., 1919.

principal views upon organic evolution and the theories of heredity. The student of preventive medicine should especially have a clear comprehension of Mendel's work, which has thrown a flood of light upon the problems before us. Mendel has opened new vistas in biology, which have a practical bearing upon public health work. It is evidently impossible in a short space to do justice to such large subjects as evolution and heredity, and the student is, therefore, referred to the authorities given at the end of this chapter, who will repay careful study.

Variation.—It has been a matter of common observation that like *tends* to beget like rather than “like *begets* like,” for there is a tendency toward new departures.

Two distinct sorts of divergences may appear among the members of a single family. The first is known as variation; the second as mutation.

By variation we understand those slight differences which invariably distinguish all the members of every family. They consist of individual differences which affect every part and every character. Such differences are also known as fluctuating, normal, or continuous variations to distinguish them from abnormal, definite, or discontinuous variations, which are more properly termed mutations. As examples of variation in man we may cite the differences in size or stature, color of skin and eyes, curliness of hair, configuration of features, etc.

Darwin lays particular emphasis upon the importance of variation in his views of organic evolution.

Darwin's Theory, The Survival of the Fittest.—Darwin's views²¹ of heredity form the basis of his theory of organic evolution. Two separate factors are primarily concerned: (1) the fact of fluctuating variation, that is, that no two members of the same family ever resemble one another exactly; and (2) the occurrence of a struggle for existence between organisms, owing to the geometric rate of increase of living things. From these two facts it follows that, when a change of environment takes place, certain members of an existing species will be somewhat better adapted than others to withstand the new conditions, and the former will tend to survive to the exclusion of the latter. Darwin assumes that during long series of generations this process will result in a steady change in the character of the species in the direction of better adaptation to the new conditions. In other words, Darwin considers that an accumulation of a series of small changes due to the direct and indirect effect of the environment is subjected to natural selection and those best fitting are transmitted hereditarily.²²

The remarkable effects produced in the case of domestic animals and plants by the action of artificial selection greatly influenced Darwin's views upon the selective influences which exist in nature. Darwin believed in the hered-

²¹ *On the Origin of Species by Means of Natural Selection, or Preservation of Favored Races in the Struggle for Life*, Murray, London, 1859.

²² Lamarck (1809) was the first to insist upon the transmission of acquired characters, since so vigorously denied by students of heredity.

itary transmission of acquired characters and regarded organic evolution as proceeding by a slow, gradual, or continuous process. There can be no doubt that natural and sexual selection have a great influence, but whether sufficient to originate new species or even new specific characters is a question. Now that the transmission of acquired characters is denied by students of heredity, and the fact that DeVries believed he saw new species arise suddenly, this portion of Darwin's theory of organic evolution and the origin of species is receiving critical examination.

Darwin firmly believed that the characters of organisms can be modified by selection, and he made this the foundation stone of his theory of evolution. The brilliancy of the mutation theory of DeVries, coupled with his great service to biology in rediscovering the Mendelian laws, has somewhat dazzled our eyes. Castle believes, after years of continuous work in selection, that much may be accomplished by this means quite apart from the process of mutation, and considers that the work of DeVries himself argues strongly in favor of this idea, although his interpretation of it is adverse to selection. From the evidence at hand we must conclude that Darwin was right in assigning great importance to selection in evolution, that progress results not merely from sorting out particular combinations of large and striking unit characters, but also from the selection of slight differences in the constitution of gametes representing the same major unit character combinations.

Mutation.—Mutations comprise definite differences or modifications usually of considerable magnitude—differences that indicate specific characters or the beginning of new species. Such differences are also known as abnormal, definite, or discontinuous variations, but more properly they are termed mutations, sometimes "sports." Mutations may be either useful or harmful. They arise "spontaneously" and are transmitted in accordance with Mendel's law. As examples of mutations in man we may cite albinism, polydactylism, brachydactylism, etc.

DeVries, Bateson, and the "mutationists" are convinced that mutation is a much more important factor in the origin of species than variation, as understood by Darwin. In the light of Mendel's work mutations appear to be unit characters which represent recessive characters that have remained dormant for many generations.

DeVries; Discontinuous Evolution.—The observations of DeVries upon the evening primrose (*Oenothera lamarckiana*) convinced him that species may arise suddenly, that evolution is discontinuous and goes by leaps and bounds rather than by the slow or continuous process of organic evolution described by Darwin.

Mutation is the term applied by DeVries to express the process of origination of a new species or a new specific character, when this takes place by the discontinuous method at a single step. DeVries believes that this is the most important, if not the sole, method by which new species or specific characters arise. To those who are convinced that acquired characters are not inherited, the explanations of Lamarck and Darwin have always been incom-

plete. Darwin insisted that nature does not make jumps and that new species arise slowly through the action of natural selection on minute variations—a gradual or continuous evolution.²³ From his experiments DeVries concludes that when selection is really efficient the full possible effects of this process are exhausted in quite a small number of generations, and that then the only further effect of selection is to keep up the standard already arrived at. DeVries actually observed quite a number of new types of plants which arose suddenly and naturally. When they made their appearance the majority of the new types came true to seed. We now know, however, that the evening primrose of Lamarck is polymorphous; that is, a persistent hybrid. Therefore, many of DeVries' "new species" were the outcroppings of recessive characters. He saw novel forms but not new species. A few of DeVries' mutants, however, represent new characters and are new mutations. Conklin and others believe that genuine gene mutations which lead to the appearance of new characters are by no means uncommon.

Weismann's Views.—Weismann was one of the first, as he was the most important opponent of the belief in the inheritance of acquired characters. No one has yet produced indubitable proof of such inheritance. It must be very rare, if it occurs at all. Weismann's²⁴ views are based largely upon his assumption that the germ-plasm is distinct from the body and that acquired characters are not inherited. The parent is composed biologically of somatic, or body cells, which are mortal, and reproduction cells, or germ-plasm, which are distinct, continuous, immortal. The germ cells undergo the least modification from their original condition. Indeed, Weismann believes that there is no reason for supposing that they have undergone any modification at all. From this point of view we may consider the nature of a given series of animals as being determined only by the particular series of cells which constitute the direct ancestry of the germ cells in each individual. The cells which make up the bodily structure may be regarded as the result of so many offshoots which come to an end at the death of the organism and have no progeny of their own.

The minute study of the germ cells taken in connection with modern experimental work on the methods by which inheritance takes place shows a

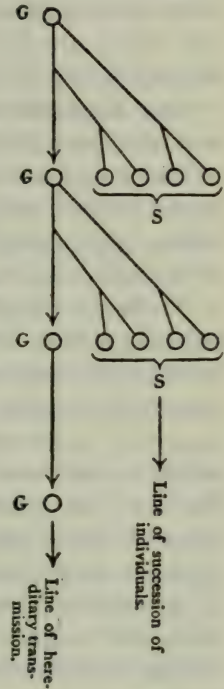


FIG. 48. — WILSON'S THEORY OF INHERITANCE MODIFIED BY LOCK. (G, germ cells; S, somatic cells.)

²³ Darwin, however, recognized the facts of mutations or "sports" as he called them and dwelt upon their importance.

²⁴ A. Weismann, *Essays upon Heredity*, 1889, and *The Evolution Theory*, 1906.

strong tendency to confirm Weismann's views, so far as the inheritance of distinct and definite characters is concerned.

Wilson²⁵ has expressed Weismann's theory as follows: It is a reversal of the true point of view to regard inheritance as taking place from the body of the parent to that of the child. The child inherits from the parent germ cell, not from the parent body, and the germ cell owes its characters not to the body which bears it, but to its descent from a preëxisting germ cell of the same kind. Thus, the body is, as it were, an offshoot from the germ cell. As far as inheritance is concerned, the body is merely the carrier of the germ cells which are held in trust for coming generations. Fig. 48 illustrates Wilson's theory of inheritance as modified by Lock.

Can Acquired Characters Be Transmitted?—Students of heredity almost unanimously deny the hereditary transmission of acquired characters, but recent experiments have shown that the germ-plasm can be injured so that altered progeny are produced, and these defects are transmitted through succeeding generations. The work of Guyer, Stockard, Pearl and MacDowell in this direction is significant.

Professor Guyer's remarkable results are important.²⁶ By repeatedly injecting a fowl with the substance of the lens of the eye of a rabbit he obtained anti-lens serum. On injecting this "sensitized" serum into a pregnant female rabbit it was found that, while the mother's eyes remained apparently unaffected, some of her offspring developed defective lenses. The defects varied from a slight abnormality to almost complete disappearance. The lens was sometimes opaque, sometimes liquid, or some other defect appeared in the eyes. No defects appeared in untreated controls, no defects appeared with non-sensitized sera. On breeding the defective offspring for six generations these defects were found to be inherited, even to tend to increase and to appear more often. When a defective rabbit is crossed with a normal one the defect seems to behave as a Mendelian recessive character, the first generation having normal eyes and the defect reappearing in the second. Further, Professor Guyer claims to have shown that the defect may be inherited through the male as well as the female parent and is not due to the direct transmission of anti-lens from mother to embryo in utero.

Stockard²⁷ found that the daily inhalation of alcohol fumes causes no apparent injury, in the doses used, upon the health of guinea-pigs. However, the alcohol affects the germ cells so that the offspring of the alcoholic parents have a high prenatal and postnatal mortality, and the number of defectives or sterile individuals is large. This occurred whether the intoxication was applied to the male or the female. Of course the defective offspring were unable to play a part in the maintenance of the race, and their gradual elimination left a superior stock.

²⁵ Wilson, *The Cell in Development and Inheritance*, p. 13.

²⁶ *Proc. Nat. Acad. Sc.*, 1920, 6: 134; *Tr. Internat. Ophth. Cong.*, 1922.

²⁷ A summary of Stockard's elaborate investigations at the Cornell University Medical College, New York, will be found in the *Proc. Am. Phil. Soc.*, 1923, 62: 311.

He concludes²⁸ from his experiments upon several generations of guinea-pigs that "alcohol injures and eliminates the weaker and less resistant germ cells of the stock and that it therefore acts to improve the quality of the race." He believes alcohol to be a decidedly selective agent and to injure only the weaker and less resistant germ cells. Stockard states that those nations of men that have used the strongest alcoholic beverages through many generations have now, from a standpoint of performance and modern accomplishments, outstripped the other nations with less alcoholism in their history. He believes that this may be due to some selective effects as those he finds in his guinea-pig experiments.

It is plain, then, that acquired defects due to injury of the germ-plasm can be produced and transmitted. It is denied, however, that these defects are specific characters. In other words, the injury to the germ-plasm is general and shows itself in the eye or delicate structures of the central nervous system, because these are particularly vulnerable. Furthermore, there is a wide difference between such *destructive* action and any *constructive* process. *Factors have been subtracted, but characters have not been added.* If a factor could be destroyed by a needle or picked out with a fine forceps, the effect of the operation would persist and be transmitted. Much of the confusion and interminable controversies about the inheritance of acquired characters is due to the neglect of the distinction between factors and characters: factors (genes) may be transmissible, characters as such never are.

It now seems quite evident that poisons, such as alcohol or lead, or toxins as those of syphilis, or antibodies or other injurious qualities of the blood, may start a line of defectives. In a practical world it is small comfort to say that these are general and not specific; congenital and not hereditary; destructive and not constructive. They may not be true instances of heredity as defined by students of genetics, but they are serious defects transmitted with Mendelian expectancy and constitute a large group of preventable tragedies.

Mutilations of the body, such as circumcision, docking of tails, or dehorning of cattle, are not transmitted, because such somatic injuries do not affect the germ-plasm. Man, however, differs from all other animals in that he transmits directly a social inheritance, which becomes a cumulative heritage, stored, sorted, tested and transmitted in the archives of human experience. It has been claimed that conditioned reflexes can be transmitted but this lacks confirmation.

Heredity versus Environment.—How much of our physical and mental make-up is due to heredity (nature) and how much to environment (nurture) is one of the much-discussed problems. It seems evident to students of biology that by far the overwhelming factor in our organization is set and definitely fixed at our birth. Heredity appears to be the overshadowing influence of first and prime importance. Herbert Spencer well said that "inherited constitution must ever be the chief factor in determining character."

²⁸ *Am. J. M. Sc.*, 1924, 167: 469.

Environment may influence the individual, but apparently has small and slow power of propagating itself for good; great and rapid power for evil.

Much has been learned from a study of twins. Human twins are of two types; (1) twins that differ from each other come from separate eggs, (2) identical twins come from one egg which divided into two embryos. These were studied by Francis Galton, for they throw light upon the relative importance of heredity and environment. It is indeed marvelous to see how such twins, even though living far apart and under different influences, remain the same inside and out. They have the same emotional disturbances and similar diseases, often at the same time. Their initial likeness continues as do their "association tests" and fingerprints. The overpowering influence of heredity is also illustrated in the differences among children in the same family who have the same environment.

We are nevertheless convinced that life is inexorably conditioned by its environment, despite the impelling force of heredity. Unicellular organisms, as bacteria from pure line strains, are susceptible to environmental influences and show great variability and plasticity. Environment also is able to influence markedly higher creatures. We do not inherit the characters themselves, but the power to produce certain characteristics under certain conditions. Many inherited characters will be realized or not depending upon whether the environment is properly constituted. Nature and nurture, heredity and environment, both play important rôles in the final molding of the individual's body and mind. The only sensible attitude is to be concerned with progeny rather than ancestry, and to continue the struggle for betterment.

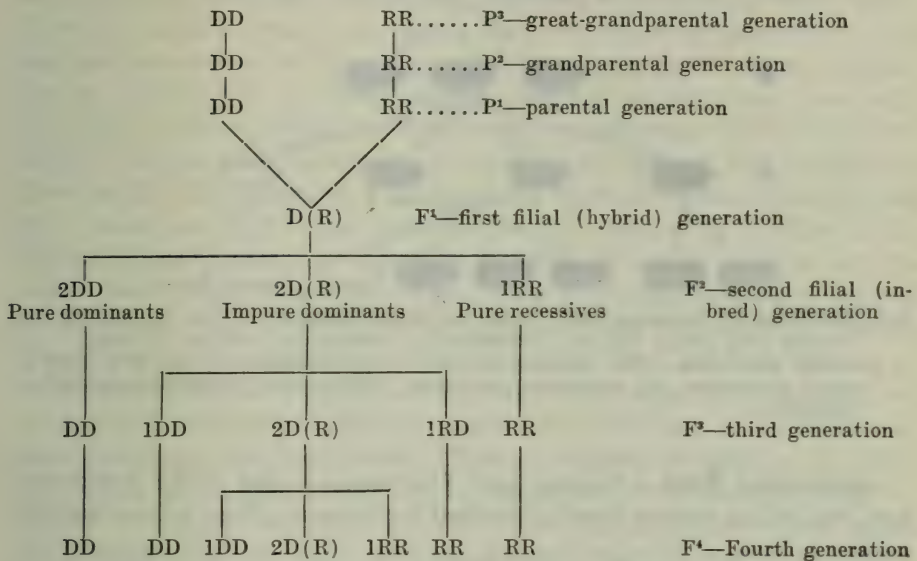
Mendel's Law.—We are indebted to Mendel²⁹ for one of the most important observations of biology—the most important, in fact, with reference to heredity. The essential factors of Mendel's discovery are: (1) unit characters, (2) dominance, (3) segregation. By a unit character is understood any characteristic of an individual that is transmitted from parent to offspring through successive generations and which conforms to the following: When parents with complementary unit characters unite, it is found that one character predominates over the other. This is known as *dominance*. It has further been found that the unit characters contributed by the respective parents do not, as a rule, blend, but remain separate or distinct. This is

²⁹Gregor Johann Mendel was born July 22, 1822, at Heizendorf in Austrian Silesia. In 1843 he entered the Augustine Convent at Althbrunn as a novice, and was ordained priest in 1847. Mendel was a teacher of natural science in the Brunn Realschule from 1853 to 1868, when he was appointed abbot of his monastery.

Mendel published only the results of his work upon hybridization with peas and a few of his experiments with *Hieracium*. The original paper on "Hybridization" was published in the *Verhandl. Naturf. Ver.* in Brunn, Abhandlungen IV, 1865, which appeared in 1866: the paper on "*Hieracium*" appeared in the same journal, VIII, 1869. The student is advised to read *Mendel's Principles of Heredity* by W. Bateson, 1909, in which he will find a translation of these two important papers. A clear exposition is also given by R. C. Punnett in his book entitled *Mendelism*, 1911.

known as *segregation*. The principles of segregation and dominance have been found to apply to the inheritance of many characters in animals and plants. It should be carefully borne in mind that the unit characters themselves are not transmitted as such in the germ cells. Just what is transmitted is not definitely known. It is quite sure that the only thing that is inherited in the germ cells is something which determines the development of the unit character. This something is called a *determiner* or gene.

A SCHEMATIC REPRESENTATION OF MENDEL'S LAW



D and R represent complementary unit characters, D the dominant character, and R the recessive character. D(R) represents a dominant with the recessive character unexpressed but potentially present. DD means pure dominants, and RR pure recessives.

The essence of this great discovery was published by Mendel in a short paper in 1866. By some extraordinary chance Mendel's observations were entirely lost sight of until the same facts were independently rediscovered in 1899 by DeVries, working in Holland, by Correns in Germany, and by Tschermak in Austria.

Mendel's law may best be understood from a concrete illustration. One of the simplest cases is that of the heredity of color in the guinea-pig.

Black and White Guinea-Pigs.—If black and white varieties of guinea-pigs are crossed, the offspring are all black, like one parent.

The black seems to cover up or wipe out the white. The black color is, therefore, said to be dominant and the white recessive. The white character, however, has not disappeared, for when the black offspring are crossed together the progeny falls into two groups: some black and some white. Three-

fourths of the progeny are black; that is, they resemble the hybrid form and at the same time one of the grandparents, while the remaining fourth resemble the other white grandparent. Some of these blacks will breed true and are, therefore, known as *homozygotes*. Some of the blacks contain a mixture of the black and white characters and are, therefore, known as *heterozygotes*. The hereditary transmission of the color character in these two illustrations through the germ cell is shown in the accompanying diagram.

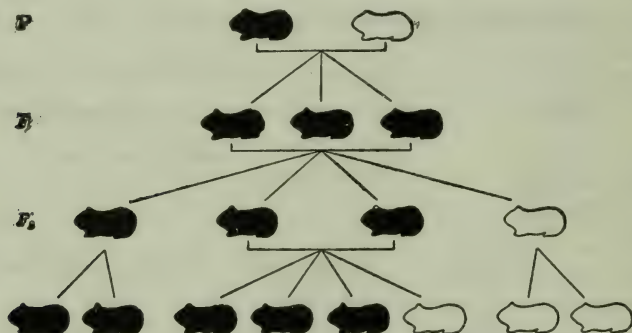


FIG. 49.—DIAGRAM SHOWING THE COURSE OF COLOR HEREDITY IN THE GUINEA-PIG, IN WHICH ONE COLOR (BLACK) COMPLETELY DOMINATES ANOTHER (WHITE). *P*, parental generation. The offspring of this cross constitute *F*₁, the first filial or hybrid generation. *F*₂, the second generation. Bottom row, third filial generation. (Kellicott).

Andalusian Fowl.—Another good illustration is that of the Andalusian fowl, which has been so clearly described by Bateson. Here we can actually see the hybrids.

There are two established color varieties of this fowl: one with a great

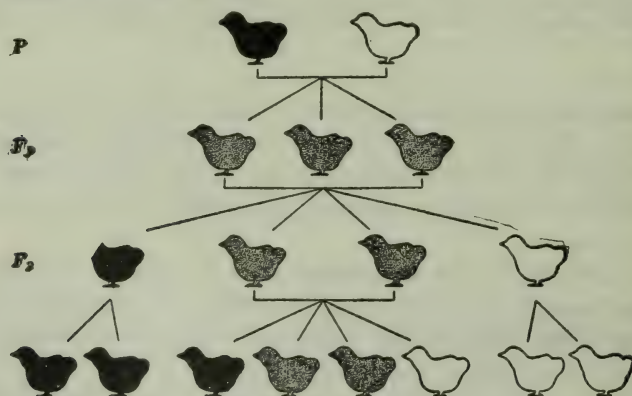


FIG. 50.—DIAGRAM SHOWING THE COURSE OF COLOR HEREDITY IN THE ANDALUSIAN FOWL, IN WHICH ONE COLOR DOES NOT COMPLETELY DOMINATE ANOTHER. *P*, parental generation. The offspring of this cross constitute *F*₁, the first filial or hybrid generation. *F*₂, the second filial generation. Bottom row, third filial generation. (Kellicott.)

deal of black and one that is white with some black markings or splashes. For convenience we may refer to these as the black and white varieties respectively. Each of these breeds true by itself. Black mated with black produce none but black offspring. White mated with white produce none but white offspring. Crossing black and white, however, results in the production of fowls with a sort of grayish color called "blue" by the fancier, though in reality it is a fine mixture of black and white. If we continue to breed succeeding generations from these blue hybrid fowls we get three different colored forms. Some will be blue, like the parents, some black, like one grandparent, some white, like the other grandparent. Further, these different colors appear in certain definite proportions among the three classes of descendants. Of the total number of the immediate offspring of the hybrid blues, approximately one-half will be blue, like the parents, approximately one-fourth black, and one-fourth white, like each of the grandparents. Thus, black bred together produce only blacks; the white similarly produce only whites; the blues, on the other hand, when bred together produce a progeny sorting into three classes, and in the same proportion as that produced by the blues of the original hybrid generation. The fact that the black grandchildren and the white grandchildren respectively breed true is a very important fact. In this illustration no race of the hybrid blue character can be established, for the blues always produce blacks and whites as well as blues (see Fig. 50).

Unit Characters.—Segregation and independent assortment were the two fundamental principles of heredity discovered by Mendel. Since 1900, four other principles have been added. These are known as linkage, the linear order of the genes, interference, and the limitation of the linkage groups.³⁰

One of the most important conclusions from Mendel's observations is that the different inherited traits act independently; that is, they do not blend. The principle of the separableness of inherited traits has been demonstrated in hundreds of plants and animals and is probably a universal law of heredity. In other words, the definitely hereditary characters act as independent units that are without any apparent relation to other peculiarities of the individual concern. Furthermore, these units do not interfere with each other. It follows that all the unit characters of an individual are to be regarded as mutually independent assemblages, each of which is derived from one or the other of the two parents but not from both. This is the doctrine of unit characters. According to this doctrine, each individual is of dual origin, paternal and maternal, and each individual is made up of a mosaic of inherited characters, some of which may be dominant, others recessive. The idea of unit characters capable of being inherited independently of one another is one of the most important conceptions which has been added to our knowledge of heredity. We now know from the phenomenon of segregation what constitutes purity in a strain of animals or plants; that is, purity does not depend upon the length of time during which a race has exhibited a constant

³⁰ Thomas Hunt Morgan, *The Physical Basis of Heredity*, J. B. Lippincott Co., 1919.

character, for a strain of absolute purity may arise from the second generation of a cross. Mendel's law has not only explained many facts in heredity, but also has important practical bearing in the improvement of the breeds of cultivated plants and domestic animals.

A few *simple* Mendelian traits have been found in man; these are eye color; curliness of hair; additional fingers and toes, which act as a dominant character; also, various defects of the eye, such as cataract; certain types of deafness, various abnormalities of the skin, hair and nails. Other traits are due to *multiple factors*, such as stature and build and proportion of parts, pigmentation of skin and hair (see p. 574).

THE CELL IN HEREDITY

Each parent (male and female) is composed biologically of somatic or body cells, which are mortal, and germ-plasm, which is distinct, continuous, immortal. The development and embryology of the germ and sperm cells are of particular interest to the student of heredity.

The nucleus is the chief or exclusive bearer of the hereditary characters; that is, the female nuclear material transmits the characters of the mother and her forebears and the male nucleus those of the father and his forebears to the offspring.

Cells divide and multiply in two ways: (1) by direct division or amitosis, and (2) by indirect division or mitosis. Indirect division or mitosis appears to be the natural mode of cell development. The chromatin, which is the deeply staining matter in the nucleus, rearranges itself from its "resting" stage. After a complicated process the nuclear matter forms itself into a long cylindrical thread known as the linene thread. This then divides into links or chromosomes.³¹ The chromosomes are of special interest, for they are believed to carry the genes.

Each chromosome is divided in halves longitudinally, as a stick might be split up the middle, and after a very complex process the halves of each split chromosome migrate to opposite poles. Each centrosome attracts a group of chromosomes consisting of just one-half of the original chromatin material. Each group then, in orderly fashion, rounds itself into a new nucleus, and the body of the cell (the cytoplasm) constricts across the equatorial plane, and two cells are formed.

Every species of plant or animal has a fixed and characteristic number of chromosomes which regularly recurs in the division of all of its cells and in all forms arising by sexual reproduction the number is even. Thus, in some of the sharks the number of chromosomes is thirty-six, in certain gastropods it is thirty-two; in the mouse and salamander, the trout, the lily, twenty-four; in the worm *Sagitta*, eighteen; in the ox and guinea-pig, sixteen; in man

³¹ For a full understanding of cell division the student is referred to one of the standard textbooks upon cytology, or Minot's *Embryology*; also, to E. B. Wilson's *The Cell in Development and Inheritance*, 2d Edition, 1900.

the number is forty-eight. In crustaceans the number of chromosomes may be as high as 168. There may be an unpaired chromosome which determines sex; or the sex chromosomes may be the X-Y combination of Morgan.

One of the most secure generalizations of modern work on the cell is that every cell of the individual contains a *constant number* of self-perpetuating bodies (called chromosomes), half of which are traceable to the father and half to the mother of the individual.

Van Beneden in 1885 discovered the equally important fact that the nucleus of the ovum and the nucleus of the spermatozoön which unite in fertilization contain each one-half of the number of chromosomes characteristic of the body cells.

As both the ovum and sperm cells contain only half the number of chromosomes, a reduction must take place in the history of these cells; in fact, alike in the history of the ovum cell and in the history of the sperm cell, there is a parallel reduction in the number of chromosomes to one-half. This reduction appears to be a preparation of the reproductive cells for their subsequent union, and a means by which the number of chromosomes is held constant in the species.

The gist and meaning of the whole process to the student of heredity is the precisely equal partition of the maternal and paternal contributions, so that each of the zygote cells that is to form a new individual has a nucleus half from the mother and half from the father. In other words, there is convincing evidence that the reduced number of chromosomes is brought about as the result of a separation of such a kind that each mature germ cell gets only a paternal or maternal member of each chromosome pair.

Although the ovum is much larger than the spermatozoön, each contributes equally so far as the amount of nuclear matter is concerned; the new individual is dual in its origin, and the offspring is a double creature and retains its duality to its dying day, and transmits it to succeeding generations.

The ovum may be stimulated to segmentation without the sperm cell (parthenogenesis). When this happens individuals are produced similar to, but not as vigorous as, the normal types. The sperm cell similarly is able to develop without the nuclear matter of the egg. In other words, the ovum and the sperm each contains potential factors for the new individual.

CHAPTER II

THE HEREDITARY TRANSMISSION OF DISEASE

We are now prepared to discuss more in detail the hereditary transmission of disease. The question whether disease is ever transmitted hereditarily rests somewhat upon our conception of disease; that is, whether it is an entity, a reaction, or a "unit character." The process itself, of course, cannot be transmitted, but the potentiality of it may be involved in some peculiarity in the organization of the germ-plasm. This may be, and often is, transmitted through successive generations. In the specific sense in which the word "heredity" is used in biology and in the limited sense in which the word "disease" is used in pathology, there may be no inherited diseases, but this appears to be a quibble of words or a matter of definitions. While we are not familiar with the intimate processes concerned, we are certain that many abnormal conditions of mind and body are transmitted. Some of them follow the Mendelian principles.

Formerly a large number of diseases were regarded as transmissible, but the list has been revised and restricted as a result of recent studies. The reappearance of a diseased condition in successive generations does not prove that it has been transmitted or even that it is transmissible. This mistake has been made with tuberculosis and other infections. Bad hygienic habits, such as faulty diets, are common family and even national traits, and lead to diseased states which seem to be inherited.

Lack of completeness and want of controls vitiate most of the statistics bearing on heredity in relation to human diseases. Even in the case of clearly inherited diseases there are very few pedigrees sufficiently complete for the study of the applicability of Mendelian and other laws of heredity. The problem needs study and critical judgment.

Sometimes the disease itself is not transmitted, but a tendency to the disease is transmitted. This will be discussed again.

Some unit characters as well as certain diseases are transmitted hereditarily, but limited to one sex; that is, the disease or condition appears in one sex only, although transmitted by the other. This remarkable sort of inheritance, known as sex-linked inheritance, occurs when the male parent is characterized by the absence of some character of which the gene is typically lodged in the sex (x) chromosome. A striking feature of this sort of heredity is that the trait appears only in males of the family, but is not transmitted by them to their sons; it is transmitted, however, through normal females of the family. Occasionally sex-linked defects occur in females, but only when duplex; that is, when both parents have the character. Examples of this sort

of heredity are hemophilia, color-blindness, also night-blindness and muscular atrophy. The explanation is the same in all cases of sex-linked heredity.

The diseases, defects, and conditions believed to be transmitted hereditarily are discussed in the following pages. Some of these diseases, malformations, and defects of organization follow Mendel's law. It is probable that other diseases, tendencies, and characters are transmissible, but the subject has only recently been placed upon a scientific basis, and it will require careful and prolonged observation to establish the facts. It is often difficult to determine whether the disease itself or a tendency to the disease has been transmitted in any particular case, and, further, it is often difficult to decide whether an individual has inherited or acquired his affliction.

The transmissible defects which are of principal concern to the human species are the defects of the central nervous system. It is important to remember that the defects of the nervous system do not necessarily propagate just the same defects in the succeeding generations. Thus, an epileptic does not necessarily beget epileptics; epilepsy, insanity, degeneracy, feeble-mindedness, and other stigmata may arise as the result of deficiencies of various kinds in forebears. In other words, these important groups often do not run true to type.

Defects such as harelip, cleft-palate, cervical fistula, spina bifida, etc., are not true instances of hereditary transmission of specific characters. They rather represent an inherited deficiency in developmental vigor. These defects for the most part represent the failure of parts to unite during embryological development; in other words, the failure of embryological clefts to close normally. Such deformities, as well as club-foot and other acquired or congenital deformities or disfigurements, are not, as a rule, transmitted.

Some practical problems of great importance arise from our knowledge of the hereditary transmission of disease and defects. A man or woman who intends marrying is now more than justified in carefully examining the personal and medical histories of the families of his or her intended mate. It is not only possible to foretell, within limits, the color of the eyes, the nature of the hair, and other Mendelian characters in the future offspring, but it is also possible to foretell, with mathematical precision, the chances of transmitting defects, such as insanity, epilepsy, degeneracy, feeble-mindedness, deaf-mutism, color-blindness, migraine, and other nervous disorders, as well as hemophilia, polydactylism, brachydactylism, albinism, and other stigmata. In any doubtful case it may be well to consult a student of heredity, for it is possible to foretell with precision in certain cases which characters will and which will not be transmitted.

To illustrate the precision with which the characters of offspring may be predicted in the best studied cases, we need only refer to the color of the eyes. Two parents with pure blue eyes will have only blue-eyed offspring, for they both lack the brown pigment which determines the color of the iris. Similarly, if the hair of parents be flaxen, this may be taken as evidence of the absence of a hair-pigment-determiner in the germ-plasm, and the offspring will have

flaxen hair. For the same reason parents with lack of curliness or waviness will have only straight-haired children (see table following).

INHERITED CHARACTERS IN MAN

(From Castle, *Genetics and Eugenics*)

1. BLENDING

General body size, stature, weight, skin-color, hair-form (in cross-section, correlated with straightness, curliness, etc.), shape of head and proportions of its parts (features).

2. MENDELIAN

	<i>Dominant</i>	<i>Recessive</i>
Skin and hair	Dark	Blonde or albino (probably multiple allelomorphs)
	Spotted with white Tylosis and ichthyosis (thickened or scaly skin)	Uniformly colored Normal skin
	Epidermolysis (excessive formation of blisters)	Normal skin
	Hair beaded (diameter not uniform)	Normal hair
Eyes	Front of iris pigmented (eye black, brown, etc.)	Only back of iris pigmented (eye blue)
	Hereditary cataract	Normal
	Night-blindness (when not sex-linked)	Normal
	Normal	Pigmentary degeneration of retina
Skeleton	Brachydactyly (short digits and limbs)	Normal
	Polydactyly (extra digits)	Normal
	Syndactyly (fused, webbed, or reduced number of digits)	Normal
	Symphalangy (fused joints of digits, stiff digits)	Normal
	Exostoses (abnormal outgrowths of long bones)	
	Hereditary fragility of bones	Normal
Kidneys	Diabetes insipidus (excessive production of urine)	Normal
	Normal	Alkaptonuria (urine black on oxidation)
Nervous System ...	Huntington's chorea	Normal
	Normal	Hereditary feeble-mindedness

3. MENDELIAN AND SEX-LINKED

(Appearing in males when simplex, but in females only when duplex.)

<i>Dominant</i>	<i>Recessive</i>
Normal	Gower's muscular atrophy
Normal	Hemophilia (bleeding)
Normal	Color-blindness (inability to distinguish red from green)
Normal	Night-blindness (inability to see in faint light)

4. PROBABLY MENDELIAN BUT DOMINANCE UNCERTAIN OR IMPERFECT

Defective hair and teeth or teeth alone, extra teeth, a double set of permanent teeth, harelip, cryptorchism and hypospadias (imperfectly developed male organs), tendency to produce twins (in some families determined by the father, in others by the mother), left-handedness, otosclerosis (hardness of hearing owing to thickened tympanum).

5. SUBJECT TO HEREDITY, BUT TO WHAT EXTENT OR HOW INHERITED UNCERTAIN

General mental ability, memory, temperament, musical ability, literary ability, artistic ability, mathematical ability, mechanical ability, congenital deafness, liability to abdominal hernia, cretinism (due to defective or diseased thyroids), defective heart, some forms of epilepsy and insanity, longevity.

The Microbic Diseases.—It seems a confusion of thought to the student of heredity to speak of the inheritance of any microbic disease. At one time the hereditary transmission of microbic diseases was generally believed. Now we know that, in the true sense of the term, no infectious disease is transmitted hereditarily—for even in the case of syphilis the *Treponema pallidum* is carried in the ovum or sperm as a foreign body. Tuberculosis at one time was considered as transmitted, but we now know that this occurs so seldom and then congenitally that the popular pamphlets are entirely justified in denying it entirely.

Children are sometimes born with smallpox, measles, and other infections; these are not true instances of heredity, but cases of congenital transmission.

Congenital Transmission.—Prenatal infection is not a true instance of inheritance. Microbic diseases may be acquired by infection through the placenta during the fetal period. The placenta is a better filter for some infections than for others. Thus, anthrax and tuberculosis of the mother are rarely transmitted to the fetus, while there is great liability in the case of syphilis. The fetus *in utero* may take smallpox, measles, and other infections, but these instances are more properly spoken of as congenital than inherited.

We must remember that to be inherited on the part of the offspring or transmitted on the part of the parents, students of genetics include only those genes of the characters which were contained in the germ-plasm of the parental sex cells (Martius); or, as Verco says, "what operates on the germ after the fusion of the sex nuclei, modifying the embryo, or even inducing an actual deviation in the development, cannot be spoken of as inherited. It belongs to the category of early acquired deviations which are, therefore, frequently congenital."

Foreign bodies carried along by either the germ or sperm cells are examples of congenital transmission and not instances of true heredity; therefore, in the present-day conception of heredity it is not possible for a microbic disease to be transmitted hereditarily, even though the microörganism is contained in either the germ or the sperm. Thus, hens fed with anilin dyes lay colored eggs. Babies born with smallpox, measles or syphilis contract these infections congenitally. In the case of syphilis and other infections, the parasite may even be carried in the egg or sperm as a foreign body but not as a unit charac-

ter. Poisons and toxins, however, may injure the egg or the sperm cells so as to cause defects in the progeny, which defects are transmitted. While syphilis itself is not transmitted "hereditarily" the effects of it may be.

Hereditary Transmission of a Tendency to a Disease.—While the disease itself may not be transmitted, a tendency to a disease, known as a diathesis, may be transmitted through successive generations. A person may inherit a small bony structure, a poor musculature, "weak" lungs, susceptible mucous membranes, or an abnormal amount, distribution, or development of lymphoid structures, etc. In fact, we are not all born equal, and most persons have some vulnerable structure or organ which is commonly spoken of as their "weak point." In many cases this *locus minoris resistentiæ* is transmitted as a defect in structure or function. An interesting example of a tendency towards constitutional inferiority of the respiratory system is given by Pearl.¹

Davenport has collected the health records and other characteristics furnished for over two hundred families by members of the families concerned. He finds certain definite facts in the behavior of some of the commoner diseases. As an example of the inheritance of a general weakness in an organ he cites the case of the mucous membranes. Thus, in one family the principal diseases to which there was liability were located in the mucous membranes of the nose, throat, and bronchi. In another family the center of susceptibility was more specific, being nearly confined to the nose and throat. In another family the weakness was in the ear; in another the lungs; in another the skin; in one family the kidneys were the seat of incidence, etc.

The examination of the health pedigrees of a number of families impresses one by the fact that the incidence of disease is not always haphazard, for in any large family the various causes of death do not occur in the proportions given in the census table for the population as a whole.

Immunity and Heredity.—Immunity to disease is either natural or acquired. Natural immunity is inherited through successive generations of a species or a race. Acquired immunity, like other acquired characters, is not inherited as a "unit character" in the sense of Mendel. Thus, there has been little variation in our natural power to resist most infections, such as tuberculosis, yellow fever, plague, smallpox, cholera, tetanus, measles, scarlet fever, diphtheria, and so on through a long list, although these diseases have doubtless afflicted the human species through untold ages. The fluctuating virulence of some infections is a matter of common knowledge, and is doubtless due to many variable factors. In a few well-known instances a certain amount of tolerance or resistance has been gained and perhaps transmitted through succeeding generations by a process of the survival of the fittest. Thus, syphilis is much less virulent now than it was during the great pandemic of the sixteenth century. The resistance which the natives enjoy to malaria in badly infected quarters of the globe is largely acquired as a result of early infections, and this increased resistance perhaps becomes more general by a weeding out of the very susceptible.

¹ *Proc. Soc. Exper. Biol. & Med.*, 1926, 23: 573.

Natural immunity to certain diseases as scarlet fever and diphtheria are family traits and are transmitted. Antitoxins and other antibodies are transmitted in two ways, (1) through the placenta and milk, a congenital transmission, or (2) as a tendency to autogenous development which appears to be an hereditary trait. Blood grouping, agglutinins, complement fixing substances and other qualities which determine susceptibility or resistance are transmitted, but just how is not yet known in most cases.

The most direct experimental evidence relating to the inheritance of resistance to infection has been reported by botanists. Biffen's experiments with wheat,² those of Barrus and of McRostie with beans,³ Parker's with oats,⁴ and Salaman and Lesley's work with potatoes⁵ constitute part of the evidence which leads Brooks to say in a general article concerning disease resistance in plants:⁶ "It is a matter of common observation to cultivators that different varieties of the same cultivated plant show marked differences in the power of resisting parasitic diseases. . . . There must clearly be some constitutional or genetic difference between these closely related plants to account for such phenomena." Evidence for the inheritance of resistance in laboratory animals is not abundant, but convincing. Leo Loeb,⁷ in one of his studies on the inheritance of cancer in mice, makes the statement that "the resistance to disease in general is also hereditarily transmitted." Guyer and Smith⁸ and Learmonth⁹ have recently published experiments which indicate that the ability of rabbits or guinea-pigs to form typhoid agglutinins may be increased by selective breeding. Wright and Lewis¹⁰ in their papers, "Factors in the Resistance of Guinea-Pigs to Tuberculosis, with Especial Regard to Inbreeding and Heredity," state that "marked differences in resistance were found among the number of inbred families of guinea-pigs. The high resistance of one of these families was transmitted by each sex to the offspring of each sex in crosses with other inbred families." Finally, there exists a great deal of empirical evidence and tradition relating to this question as it affects man and his reaction to infectious disease. The susceptibility of the savage to the ailments of civilized man, and the greater severity of epidemics among isolated peoples have been explained on the basis of natural selection and breeding of survivors.¹¹ Cummins¹² and Bushnell¹³ have attempted to explain such variations in susceptibility to tuberculosis on the basis of a specific acquired immunity, but the ultimate solution of the problem must await the accumulation of more experimental facts. The experiments reported by Webster¹⁴ were performed under conditions which reduced the possibility of spe-

² *J. Agric. Soc.*, 1912, 4: 421.

⁴ *Am. Soc. Agron.*, 1920, 12: 23.

⁶ *Brit. M. J.*, 1922, 2: 964.

⁸ *J. Infect. Dis.*, 1923, 33: 498.

¹⁰ *Am. Naturalist*, 1921, 55: 20.

¹¹ *Biometrika*, 1913, 9: 320; *Diekonstitutionelle Disposition zu inneren Krankheiten*, Berlin, 1917.

¹² *Internat. J. Pub. Health*, 1920, 1: 137.

¹³ *A Study in the Epidemiology of Tuberculosis with Especial Reference to Tuberculosis of the Tropics and of the Negro Race*, New York, 1920.

¹⁴ *J. Exper. M.*, 1924, 39: 879.

³ *Phytopath.*, 1918, 8: 589; 1919, 9: 141.

⁵ *Genetics*, 1923, 13: 177.

⁷ *Am. Naturalist*, 1921, 55: 510.

⁹ *J. Hyg.*, 1923, 22: 100.

cific acquired immunity to a minimum and indicate, therefore, that in mice, host resistance contains important hereditary factors both specific and non-specific in nature and that the degree of this resistance in a given population may be enhanced by selective breeding. If mice surviving a lethal dose of mouse typhoid bacilli are inbred consecutively for a number of generations, the resulting offspring become progressively more resistant to mouse typhoid infection and to mercury bichlorid intoxication than similar control mice not so selected.

HEREDITY OF CERTAIN DISEASES

The following is a list of "diseases" or morbid states with reference to their hereditary transmission. Some of them are only anatomical or physiological anomalies or defects.

Tuberculosis.—We know that tuberculosis is never transmitted hereditarily, and is seldom contracted congenitally. The reason that tuberculosis runs in a family is twofold: (1) an inherited predisposition to the disease, and (2) increased chances of infection, especially during infancy. Just what the tendency or predisposition is, is not well understood. We do know, however, that the predisposition is not so great but that it may be overcome; the infection may be avoided and the disease prevented. No one is born doomed to die of tuberculosis.

It is now perfectly plain that the principal reason why tuberculosis runs in families is the close association between the infected and well members of the family, which increases the chances of infection and reinfection.

All babies are susceptible to tuberculosis at birth—whether born of tuberculous or healthy parents. Immunity is acquired in time by small repeated infections. The protection thus gained is variable and never great; in some individuals it is quite feeble. This acquired immunity is not transmitted. The border line between immunity and susceptibility to tuberculosis in the human species is delicately balanced and may readily be overturned (see page 172).

Syphilis.—Syphilis and the consequences of syphilis are transmitted from parent to offspring—"even unto the third and fourth generation." The transmission of the infection itself is *congenital*; the transmission of the injurious consequences of syphilis, such as defectives, etc., may follow the law of heredity.

The methods of transmission may be briefly summarized as follows: (a) The husband has syphilis. He infects his wife; and she infects the embryo. Experience indicates that this is the usual method of transmission, and that the wife is often infected soon after marriage. The treponemata circulating in the blood stream of the mother apparently penetrate the placenta with ease, and enter the blood stream of the child. The child naturally has no trace of a chancre, so that we have here another indication of the possibility of syphilis d'emblée.

It is now generally believed that paternal transmission directly to the fetus is impossible. Treponemata have been demonstrated in the seminal fluid of syphilitics, and numerous innocent marital infections have been transmitted by means of infected spermatic fluid by husbands who thought they were healthy.

(b) The wife alone has syphilis and infects the child in the same manner as in (a). It is fairly obvious that in any case of congenital syphilis, the mother is certainly infected, and the father is usually, but not necessarily infected.

A syphilitic father may beget an apparently healthy child, even when the disease is fresh and full-blown. On the other hand, in very rare instances a man may have syphilis when young, undergo treatment, and for years present no signs of disease, and yet his first-born may show very characteristic lesions. The closer the begetting to the primary sore the greater the chance of infection. A man with tertiary lesions may beget healthy children. As a general rule, it may be said that with judicious treatment the transmissive power seldom exceeds three or four years.

Colles' and Profeta's Laws.—Colles, in 1837, stated that apparently normal women bearing syphilitic children do not contract syphilis when exposed to that infection. That is, the mother may nurse her syphilitic baby with impunity. (This is also called Baumes' law.) Profeta's law states that a child showing no taint but born of a woman suffering from syphilis will not become infected even though suckled by its mother. Exceptions to both Colles' and Profeta's laws are recorded. The explanation of these so-called laws was that the mother was immune in Colles' law and the baby in Profeta's law. We now know, however, that both the mother and the baby under these circumstances are infected with spirochetes but do not manifest clinical symptoms of the disease. The apparent immunity in both instances is due to the fact that superinfection does not occur (see page 59).

Concerning the results of congenital syphilis, we have the following possibilities: (1) The infection causes a cessation of development and abortion. (2) The fetus grows, but is born before the normal expiration of intra-uterine life (premature births). (3) The fetus goes to term, but is born dead (stillbirths). (4) The child is born at term living, but with unmistakable signs of syphilis, and dies shortly. (5) The child may show no symptoms of syphilis at birth, but a few weeks later develop typical symptoms. It may die, or as the result of treatment, may live. Syphilis causes death in 80 per cent of those congenitally infected. It is a still greater tragedy in the 20 per cent who survive. (6) The child shows no symptoms of syphilis for weeks, months or possibly years, the disease being latent, and becomes manifest in some cases as late as twenty-eight years in the tertiary form (so-called syphilis hereditaria tarda). (7) The child may be puny, weak, susceptible to infections, underweight, and lack stamina—in short, a runt. This may occur without obvious manifestations of the disease.

See article on Syphilis, pages 51 to 63.

Cancer.—We are indebted to the zeal of Maude Slye¹⁵ whose twelve years of untiring work have given convincing evidence upon the difficult problem of the heritability of cancer. She worked upon the spontaneous development of cancer in mice, and repeatedly demonstrated that cancer can be bred in and out of mice at will. Her studies led to the conclusion that (1) there is an inherited tendency to spontaneous cancer and (2) there is strong evidence against the probability of cancer being a specific germ disease.

Maude Slye is convinced that cancer tendency acts as a unit character. Cancer tendency is recessive to non-cancer, and the first hybrid generation shows none of it, but it reappears in the second hybrid generation and in the same organs which show the ancestral tumor. Cancer tendencies then segregate out and are transmitted as unit characters. It is possible by selective breeding to implant cancer indelibly in mice, or to eliminate it permanently. If this is a general law for all species, we must agree with Maude Slye that there is in man a "genetic method of escape from cancer." Tyzzer and Little have also shown that a tendency to cancer is transmissible in experimental animals. The factor of irritation, of growth control and other features with reference to cancer are discussed on page 426.

Many students of the disease have noted a pronounced hereditary disposition in human families, as mentioned by Warren, Broca, etc.

Warthin,¹⁶ from studies of a long series of cases, believes that a marked susceptibility to carcinoma exists in the case of certain family generations and family groups. This susceptibility, he thinks, is frequently associated with a marked susceptibility to tuberculosis and also with reduced fertility. The multiple occurrence of carcinoma in a family generation practically always means its occurrence in a preceding generation. The family tendency is usually more marked when carcinoma occurs in both maternal and paternal lines. Family susceptibility to carcinoma is shown particularly in the case of carcinoma of the mouth, lip, breast, stomach, intestines, and uterus. In a family showing the occurrence of carcinoma in several generations there is a decided tendency for the tumor to develop in the breast of the youngest generations. In this case the tumors often show an increased malignancy. Levin's study of cancerous fraternities leads him to believe that the cancerous members correspond very closely to the Mendelian percentage of members with recessive unit characters in a hybrid generation. Levin concludes that resistance to cancer is a dominant character whose absence creates a susceptibility to cancer. While some of Warthin's cases show a family history suggesting this form of inheritance, others indicate a progressive degenerative inheritance, that is, the running out of a family line through the gradual development of an inferior stock, particularly as far as resistance to tuberculosis is concerned.

Leprosy.—Leprosy is not transmitted hereditarily, although formerly so regarded. The infection is contracted after birth, as tuberculosis and other

¹⁵ *J. Cancer Research*, 1922, 7: 2, in which this work and bibliography is summed up to date.

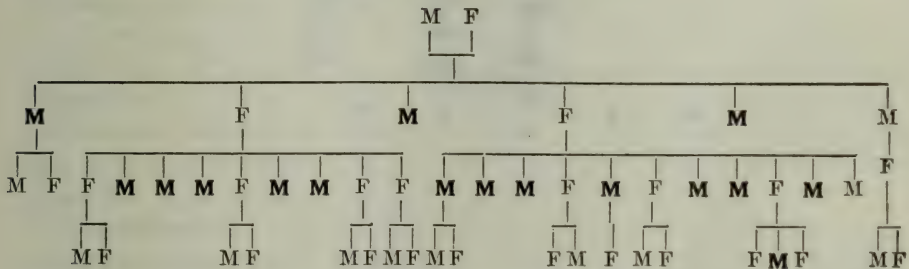
¹⁶ *Arch. Int. Med.*, 1913, 12: 485.

microbic diseases are contracted. The disease is rarely, if ever, transmitted congenitally and the children of lepers remain clean if removed from their infective environment.

Deaf-Mutism.—Deaf-mutism is due to a great variety of causes, but in different individuals of the same family the chances are large that it is due to the same defect. This defect is frequently recessive; that is, hidden in the normal children. Two such normal children who are cousins but from deaf-mute stock tend to have about one-fourth of their offspring deaf-mutes. The proportion of deaf offspring is thrice as great among cousin marriages as among others. The conclusions of Fay, based on extensive statistics, deserve to be widely known. "Under all circumstances it is exceedingly dangerous for a deaf person to marry a blood relative, no matter whether the relative is deaf or hearing, nor whether the deafness of either or both or neither of the partners is congenital, nor whether either or both or neither have other deaf relatives besides the other partner."

Albinism.—Albinism is an extreme case of blondness, all, or nearly all, pigment being absent from skin, hair, and eyes. The method of inheritance resembles that of eye color. This is like a simple recessive unit character. When both parents lack pigment, all offspring are likewise devoid of pigment. When one parent only is an albino and the other is unrelated, then the children are all pigmented. Whenever pigmented parents have albino children, the proportion of the albinos approaches the ideal and expected Mendelian proportions—25 per cent. Albinos may avoid albinism in their offspring by marrying unrelated pigmented persons. Pigmented persons belonging to albinic strains must avoid marrying cousins, even pigmented ones, because both parents might, in that case, have albinic germ cells and produce one child in four albinic. Albino communities, of which there are several in the United States, are inbred communities, but not all inbred communities contain albinos.

Hemophilia.—Hemophilia is a condition in which the blood does not coagulate properly, and those having this condition may bleed to death from minute wounds. It is transmitted hereditarily and is largely confined to males, although transmitted by normal females. It is one of the best instances of an hereditary character, sex-linked.



(Bold-faced type indicates bleeders.)

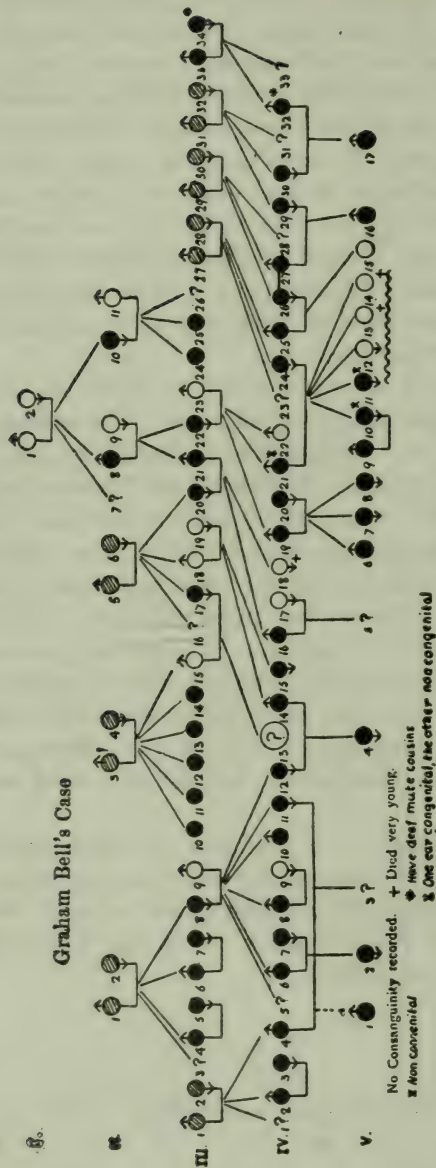


FIG. 51.—FAMILY HISTORY SHOWING DEAF-MUTISM.
(From *Treasury of Human Inheritance*.)

The foregoing case, given by Klebbs, is instructive in showing how the tendency, though transmitted through daughters, finds expression only in the males, and in illustrating first a diffusion and then a waning of the peculiarity (Thompson).

Families of bleeders have been traced back to the eighteenth century and have been written up in the medical archives. Formerly, only 11 per cent of the young with hemophilia lived to the age of twenty-one years. The prognosis has been transformed in late years by serum treatment. Normal horse serum or human blood-serum will stop bleeding and also prevent the condition. The subcutaneous injection of 20 cubic centimeters of horse serum every second month for fifteen injections has arrested the tendency to hemorrhage (Weil). Wolf reports favorable results with repeated subcutaneous injection of 5 to 10 cubic centimeters of a 5 per cent solution of peptone. Time will tell how long the results last.

Weil adds that thyroid, ovarian and suprarenal treatment, calcium chlorid, and gelatin are ineffectual in familial hemophilia.

Gout.—It is known that gout runs in families, but just what the predisposition is that favors this condition of deranged metabolism is not known. During four centuries one family history showed that out of 535 gouty subjects 309 had a family taint—about 60 per cent. In another family out of 156 cases 140 had a family taint, about 90 per cent. Statistics show that in from 50 to 60 per cent of all cases the disease existed in the parents or grandparents. It seems clear that some predisposing factor may be transmitted hereditarily, but in any individual case it is not always plain how much is due to heredity and how much is acquired. With a better understanding of regimen gout is a disappearing disease.

Brachydactylism.—A typical example of an abnormality transmitted hereditarily is that of brachydactylism, or short-fingeredness, a condition in which each digit comprises only two phalanges—the fingers are all thumbs. This condition seems to be due to an inhibition of the normal growth process; that is, normality implies entire absence of the determiner that stops the growth of the fingers in the brachydactyl. Thus, a brachydactyl person married even to a normal person will beget 100 per cent or 50 per cent abnormals, according to circumstances; but two parents who, though derived from brachydactyl strains, although lacking the determiner which inhibits the growth of the fingers may have only normal children.

According to Punnett, brachydactylism is a good example of a simple Mendelian case. It behaves as a simple dominant to the normal; that is, it depends upon a factor which the normal does not contain. The recessive normals cannot transmit the affected condition whatever their ancestry. Once free, they always remain free, and can marry other normals with full confidence that none of their children will show the deformity.

Polydactylism.—Polydactylism is a condition in which there are supernumerary fingers or toes. This is a defect which may be transmitted through successive generations. Polydactylism is endemic in certain secluded villages

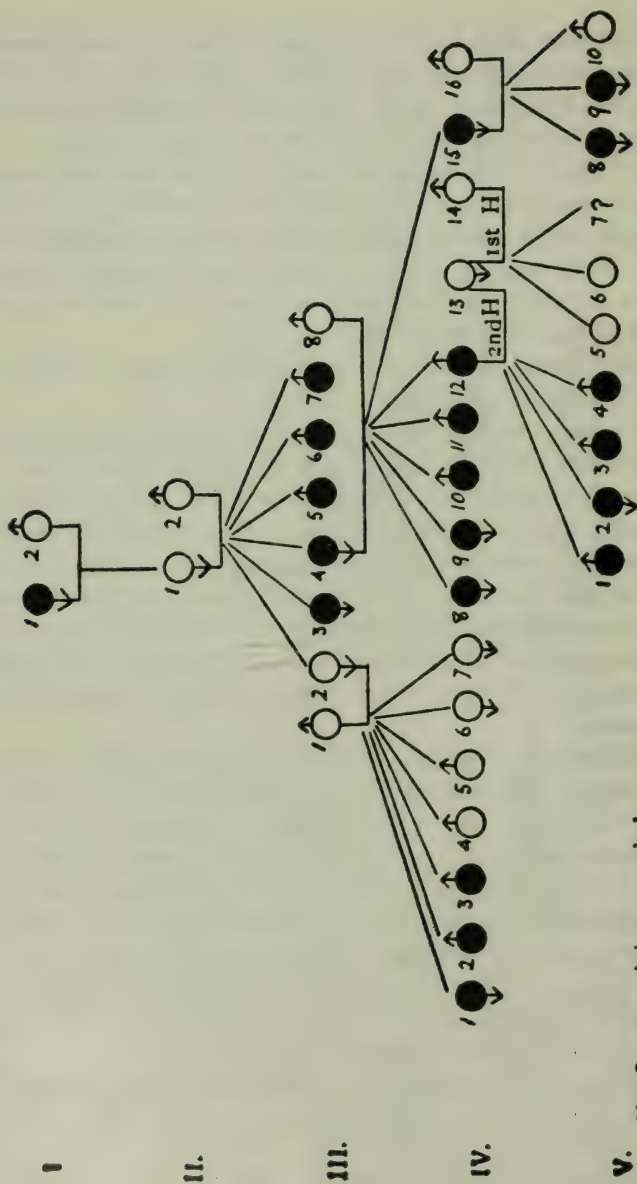


FIG. 52.—FAMILY HISTORY SHOWING POLYDACTYLISM.
Smith and Norwell's case. (From *Treasury of Human Inheritance*.)

in which consanguineous marriages are frequent. Other defects of the fingers and toes are transmitted in accordance with the Mendelian expectation.

Syndactylism.—Syndactylism, or webbed toes or fingers, is sometimes transmitted. A good instance is reported by Murphy¹⁷ of a family in which the defect ran through five generations.

Chemical malformations, such as alkaptonuria, cystinuria and pentosuria, represent types of inborn errors of metabolism. These faults of function are usually not serious and only betray themselves now and then through chance accidents. There are doubtless many other transmissible defects, for we have only scratched the surface of this subject.

Glandular and Tissue Defects.—*The Endocrine System.*—Anomalies of structure and formation of the various glands and tissues of the body are common and some of them seem to owe their origin to heredity. Rewardful results in this field await further studies upon the transmission of defect of the endocrine system. We already have evidence that some of these may be transmitted. This is the case with the thyroid and perhaps with the suprarenal, the pituitary and other glands of internal secretion. Obesity and slender build are family traits, dependent upon metabolism and growth factors, which in turn are largely controlled by endocrine activity and influenced by heredity. Several of the complex qualities of the blood are transmitted and seem to be inherited.

The relation of the glands of internal secretion to human development and human behavior is becoming daily more obvious. The endocrine secretions influence stature, build, proportions, details of development of bone, teeth, nails, hair and skin; intelligence and emotional control. These glands secrete "hormones" which regulate our physical, mental and temperamental constitution. Personality and behavior are determined to a large extent by the activity of the glands of internal secretion which in turn are conditioned largely by inheritance.

Fragile Bones and Blue Sclerotics.—Fragilitas ossium, or osteopsathyrosis, a weakness of the long bones, may arise from a number of pathological conditions affecting the bones. But the typical fragilitas ossium or brittle bones run through families. Davenport and Conrad¹⁸ have shown that the factor which determines the deficient bone formation is a dominant one. Different degrees of the condition are transmitted true to type. Thus in some families the slightest pressure results in fracture; in other families the bones are fairly resistant. The association of blue sclerotics with brittle bones has often been pointed out. Stobie¹⁹ studied a family in which fragile bones, blue sclerotics and deafness were transmitted through five generations; out of sixty-six persons, eighteen were affected. The deafness is due to otosclerosis and appears in the second and third decade.

¹⁷ *J. Am. Ass.*, 1925, 84: 576.

¹⁸ Davenport, C. B., and Conrad, H. S., "Hereditary Fragility of Bone," *Proc. Nat. Acad. Sc.*, 1915, 1: 537.

¹⁹ *Quart. J. Med.*, 1924, 17: 274.

Myopia.—Myopia can hardly be called a disease in the strict sense, being a structural defect in the focusing power of the optical apparatus. It seems that the structural peculiarity which leads to short-sightedness is transmitted. Myopia runs in families (see page 460).

Cataract.—Bateson and others have collected pedigrees in which cataracts run in families. Presenile cataract especially appears to be transmitted hereditarily. The transmission is as a rule direct, the skipping of a generation being unusual. Cataract is also favored by various constitutional diseases, such as diabetes, Bright's disease, and diseases of the vascular system. Exposure to intense light and heat from glowing coals or molten glass or flashes of electricity may produce opacities of the lens (see page 471).

Retinitis pigmentosa.—Retinitis pigmentosa is a degenerative disease of the retina which is transmitted hereditarily. It is widely distributed and appears at various periods of life and in varying degrees of intensity. Normals may carry the disease, so that two normal cousins from retinitis stock may have offspring with retinitis. A large percentage of cases of retinitis come from consanguineous marriages.

Color-Blindness, or Daltonism.—Color-blindness, or daltonism, is a condition probably not localized in the eyes, but due to some defect in the central nervous structure. It is transmitted hereditarily. Color-blindness is much commoner in men than in women. A color-blind man, however, does not transmit color-blindness to his sons; the daughters, also, are themselves normal, provided the mother was, yet the daughters transmit color-blindness to half their sons. A color-blind daughter could be produced apparently only by the marriage of a color-blind man with a woman who transmits color-blindness, since the daughter, to be color-blind, must have received this unit character from both parents, whereas the color-blind son receives the character only from his mother; that is, the condition is sex-linked.

Color-blindness is apparently due to a defect in the germ cell—absence of something normally associated there, with an X-structure which is represented twice in women, once in men.

Color-blindness was discovered by Dalton in 1794. Normally, we see violet, indigo, blue, green, yellow, orange and red, all the colors of the spectrum. Blue, yellow and orange are often diminished, that is only violet, green and red are distinguished. Such persons are color-blind. When only blue and yellow are seen, red and green cannot be distinguished and this form of color-blindness may be dangerous. Total color-blindness occurs and in such cases the world looks like various tones of gray. Those who see only three colors—red, green and violet—are markedly color-blind; those who see only two colors—yellow and blue—are especially dangerous, because they cannot distinguish red from green.

Total color-blindness is rare, but congenital partial color defect is common, occurring in about 4 per cent of all males, but only in about 0.2 per cent of females. The percentage is higher among Quakers. Color-blindness of a degree dangerous in occupations requiring recognition of colored signal lights

occurs in about 3.1 per cent of men and in about 0.7 per cent of women. In total color-blindness consanguinity in the parents has been traced in 12.5 per cent of the cases. The condition is usually inherited. The most common cause of acquired color-blindness is the immoderate use of tobacco and alcohol (page 470).

The prevention of the inherited defect is a problem of eugenics. The prevention of acquired color-blindness consists in avoiding or the disuse of the poisons that cause it.

The occupations in which the color sense is important are: All naval and marine officers, pilots and certain classes of seamen, locomotive engineers, and all occupations in the arts requiring mixing pigments or matching colors.

The Holmgren test consists in matching various colors from a confused mass of skeins of colored yarns. The three chief test colors are pale green, light pink and bright red. The Jennings self-recording worsted test is an improved modification of this skein test, but results in the rejection of a large percentage of subjects who should be accepted for sailors or trainmen, and it therefore should be supplemented with the Eldridge-Green, or the Williams lantern test.²⁰

Diabetes mellitus.—Hereditary influences seem to play an important rôle in diabetes mellitus, for cases are on record of its occurrence in many members of the same family. Thus, out of 104 cases of diabetes mellitus twenty-two had a family taint—about 20 per cent. Naunyn obtained a history of diabetes in thirty-five of 201 private cases, but in only seven of 157 hospital cases.

The prevention of diabetes consists in guarding against overweight in persons who have a history of the disease in their family and especially if they have a high blood-pressure. Starchy and sugary food must be kept within the limits of sugar tolerance. Ease, prosperity and indulgence predispose to diabetes.

Orthostatic Albuminuria.—Orthostatic albuminuria occurs in boys more commonly than girls. These are often the children of neurotic parents, and have well-marked vasomotor instability. Defects or peculiarities in the filtering apparatus in the kidneys may arise as a germinal variation and be handed on from generation to generation. Under conditions which may mean nothing to normal subjects this defect in the kidney may find expression in active disease. In this case, as in gout, it may not be proper to speak of the disease itself being transmitted hereditarily, but the tendency to deviate is so transmitted.

Anaphylaxis, or Food Idiosyncrasies.—Experimental evidence, as well as family histories, clearly indicates that hypersusceptibility to certain foods, such as egg, shellfish, strawberries, tomatoes, etc., is transmitted hereditarily through several generations. The transmission is sometimes specific and limited to one particular food.

Hay fever and other anaphylactic conditions such as asthma, angioneurotic edema, etc., usually show a family history of "idiosyncrasies" of various sorts,

²⁰ *U. S. Pub. Health Bull.*, 1918, 93.

Epilepsy.—Brown-Séquard believed that artificially induced epilepsy in the guinea-pig is transmissible. The statistics collected for man give from 9 to over 40 per cent of cases in which heredity is an important predisposing cause. Gowers gives 35 per cent for his cases. In the Elwyn cases thirty-two of the 126 gave a family history of nervous derangement of some sort, either paralysis, epilepsy, marked hysteria, or insanity. Thom's ²¹ study of 1,536 epileptics at the Monson State Hospital (Mass.) would make it appear that epilepsy is transmitted directly from parents to offspring less frequently than we have heretofore been led to believe. Epilepsy and other mental disorders if transmitted are not necessarily transmitted true to type. There may be an underlying defect which expresses itself in various ways.

Chronic alcoholism in the parents is also regarded as a potent predisposing factor in the production of epilepsy. Echeherria has analyzed 572 cases bearing upon this point, and divided them into three classes, of which 257 cases could be traced directly to alcohol as the cause, 126 cases in which there were associated conditions, such as syphilis and traumatism, 189 cases in which alcoholism was probably the result of the epilepsy. Figures equally strong are given by Martin, who, in 150 insane epileptics, found eighty-three with a marked history of paternal intemperance. Of the 126 Elwyn cases in which the family history of this point was carefully investigated, a definite statement was found in only four of the cases (Osler).

Epilepsy of the ordinary dementing type seems to be due, like feeble-mindedness, to a single developmental defect. Dementia præcox has also been found by several investigators to be due to a similar cause (Davenport). Both may crop out of good soil.

Alcoholism.—Alcohol as well as lead and other poisons can damage the germ cell of the male in such a way as to express itself by defective offspring (*Rauschkinder*, or Jagchildren). It is a common observation that among the offspring of drunkards are many cases of unhealthy, insane, and criminal types. The disastrous results may be manifested by nervous disorders, varying from hyperexcitability to dementia; or as debility and lack of developmental vigor expressed, for instance, in infantilism, want of control, imbecility, or as structural abnormalities, especially of the head and brain. The results are so varied they suggest that what is inherited is general rather than specific. Thus, the offspring of alcoholic parents are not necessarily predisposed in any one particular direction, except that the nervous system is most liable to be affected. They may be epileptic, idiotic, insane, etc. On the other hand, it is necessary to recognize that what may be inherited is not the result of alcoholism, but rather the predisposition which led the parent to become alcoholic. This is clearly illustrated in cases where the parent did not acquire the alcoholic habit until after the children were born. Clouston observes that "it is not the craving for alcohol that was inherited, but a general psychopathic constitution in which the alcoholic stimulus is an undue stimulus and the mental control deficient" (see page 516).

²¹ *Boston M. & S. J.*, 1916, 174: 573.

Migraine.—That migraine is transmitted hereditarily is indicated from the family histories of those suffering with this affection.

Buchanan²² found from a study of 1,300 persons suffering with migraine that it is transmitted in the Mendelian ratio. His studies establish the hereditary nature of the affection.

Huntington's Chorea.—Huntington's chorea is clearly inherited. The disease is known as chronic hereditary chorea. It was described by Lyon in 1863, who traced the disease through five generations. Huntington in 1872 gave the three salient points in connection with the disease, viz.: (1) its hereditary nature; (2) association with psychical troubles; and (3) late onset between the thirtieth and fortieth year.

Huntington's chorea is a typical dominant trait. The normal condition is recessive; in other words, the disease is due to some positive determiner. Persons with this dire disease should not have children, but the members of normal branches derived from the affected strain are immune from the disease. This disease forms a striking illustration of the principle that many of the rarer diseases of this country can be traced back to a few foci, even to a single focus; certainly in this case many of the older families with Huntington's chorea trace back to the New Haven colony and its dependencies and subsequent offshoots (Davenport).²³

Friedreich's Disease—Hereditary Ataxia.—This disease resembles locomotor ataxia, although differing from it in several essential particulars. It begins in childhood and usually occurs in a family having other members of the family affected with the same disease. There are curious forms of incoordination and loss of knee-jerk, early talipes equinus, scoliosis, nystagmus, and scanning speech. The affection lasts for many years and is incurable. In 1861 Friedreich reported six cases of this form of ataxia in one family. Since then it has usually been observed to be a family disease, and is, therefore, assumed to be transmitted hereditarily. The eugenic teaching in this affection, according to Davenport, is that normal members of the affected fraternities should marry only outside the strain. Whether all cases of ataxic offspring of one normal parent are derived from consanguineous marriage is still uncertain and warrants hesitation in advising the marriage of any ataxic person.

Feeble-Mindedness.—The hereditary factor in feeble-mindedness is evident in a large but not exactly defined percentage of cases. This question is discussed in detail on pages 552 to 557.

Insanity.—The hereditary factor is clear in dementia præcox, manic depressive psychosis and in the involution psychoses. It is generally believed that heredity is responsible for more cases of mental disease than any other single cause. It sometimes crops out in good soil. Insanity, however, is too general and vague a term to generalize. Each psychosis must be considered separately with reference to heredity.

²² *Med. Rec.*, Nov. 13, 1920, 807.

²³ *Am. J. Insan.*, 1916, 73: 195.

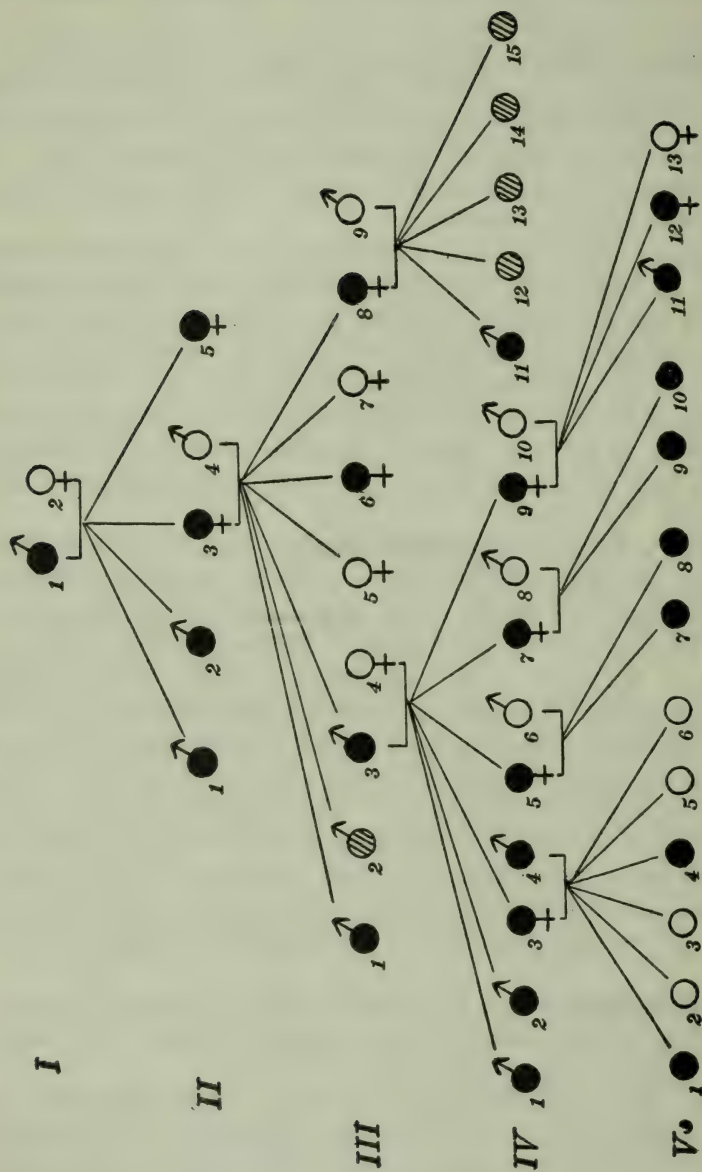


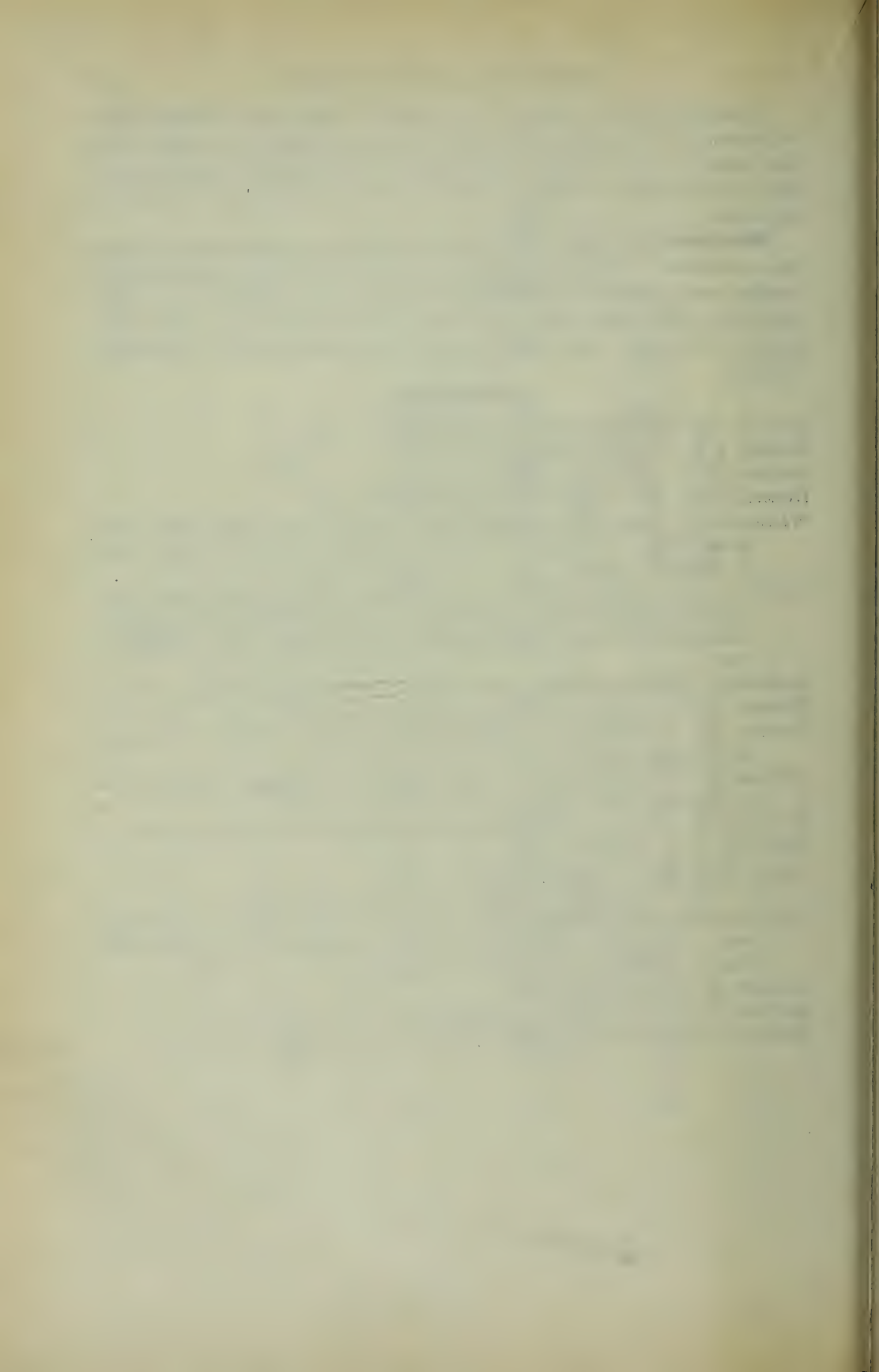
FIG. 53.—FAMILY HISTORY SHOWING HUNTINGTON'S CHOREA.
Last generation incomplete. (Data from Hamilton.)

Practically all the statistics accumulated on insanity have limited value to the student of heredity, because they do not give numerical records of the sane members of the families of the insane. The difficulty of obtaining controls for comparison is evident. The subject is fully discussed in Section II, page 433.

Miscellaneous.—Many other conditions seem to be transmissible, for they run in families. The following have been observed: ichthyosis, muscular dystrophies, nevi, hematuria, hemorrhagic telangiectasia, synostosis of the proximal ends of the radius and ulna, called congenital pronation. There also appears to be a life factor which seems to be hereditary, and determines longevity.

REFERENCES

- LOCK, R. H. *Variation, Heredity, and Evolution*, 1910.
- HUXLEY, T. H. *Collected Essays*, 1899, Vol. II.
- LAMARCK, J. B. *Philosophie Zoologique*, 1809.
- DARWIN, CHAS. *The Origin of Species*, 6th Edition, 1872.
- WEISMANN, A. *Essays Upon Heredity*, 1889; *The Variation Theory*, 1906; *The Germ Plasm: A Theory of Heredity* (translated by W. N. Parker and H. Rönnefeld, 1893).
- GALTON, F. *Natural Inheritance*, 1889; *Hereditary Genius*, 1869; *English Men of Science*, 1874; *Inquiries Into Human Faculty and Its Development*, 1883; *Natural Inheritance*, 1889; *Eugenics: Its Definition, Scope, and Aims*, 1905.
- DAVENPORT. *Statistical Methods*, 1904; *Heredity in Relation to Eugenics*, 1911.
- PEARSON, K. *The Grammar of Science*, 2d Edition, 1900.
- BATESON, W. *Materials for the Study of Variation*, 1894; *Mendel's Principles of Heredity*, 1909.
- DEVRIES, H. *Die Mutationstheorie*, 1901; *Species and Varieties: Their Origin by Mutation*, 1905.
- WILSON, E. B. *The Cell in Development and Inheritance*, 3d Edition, 1925.
- THOMPSON, J. A. *Heredity*, 1908.
- CASTLE, WILLIAM E. *Heredity*, 1911.
- *Genetics and Eugenics*. Harvard University Press, 1916.
- The Proceedings of the Royal Society of Medicine*, 1909, Vol. II: "The Influence of Heredity on Disease, with Special Reference to Tuberculosis, Cancer and Diseases of the Nervous System."
- PUNNETT, R. C. *Mendelism*, 1911.
- EAST and JONES. *Inbreeding and Outbreeding*. J. B. Lippincott Co.
- MORGAN, T. H. *The Physical Basis of Heredity*. J. B. Lippincott Co.



SECTION VI

FOOD

CHAPTER I

GENERAL CONSIDERATIONS

The proper amount, variety, and quality of food is one of our most important preventive measures; in fact, the vigor and success of a nation rests fundamentally upon its diet. Diet is influenced by many circumstances, such as availability of foods, economic status, habits and prejudice, knowledge and education, etc. Instinct is not always a safe guide. Finally, diet may lower the standard of public health in subtle ways, for there are probably many degrees of malnutrition not recognizable except in their effects on the individual over a long period of time. Diet may make or mar public health.

Foodstuffs fall naturally into three great divisions: (1) those derived from the animal kingdom, (2) from the plant kingdom, and (3) inorganic substances from the mineral world. The animal foods are much more apt to convey infections or to possess injurious properties than foods derived from plant life. Of the animal foods meat and milk are the chief offenders. Water ordinarily is not classed as a food because it passes unchanged through the body and hence does not furnish energy. Water is discussed in Section IX.

It is often said that there is "death in the pot." This is a clever adage, but a gross exaggeration. The statement is misleading, for it implies frequent and serious injury due to food, whereas food poisoning is relatively rare and seldom fatal. There is small danger in clean food, little in fresh food, and still less in thoroughly cooked food.

The hygienic conscience of the people has been aroused, and a demand is being established for clean, fresh, wholesome foodstuffs. The separation of the producer and the consumer and the demands of large cities have made these sanitary reforms eminently necessary. The pure food laws, the meat inspection act, the milk ordinances, and the local surveillance over markets, provision shops, dairies, food-handlers, etc., are all part of the general movement to obtain a reasonably decent and safe food supply.

People should be educated to demand flesh from healthy animals, cut up and handled in a careful manner by butchers free from disease, and to demand garden truck grown in clean dirt and not in soil polluted with human excrement. Food must be guarded in transportation and purveyed in markets and shops so as to be protected from flies, rats, dust, and unnecessary human contact.

The health of food handlers is an important factor. This applies espe-

cially to those who handle milk and milk products. Meat and other foods may be infected from cases or carriers who have been responsible for epidemics of typhoid and paratyphoid fevers, diphtheria, scarlet fever, septic sore throat, dysentery and other infections. Harris and Dublin¹ examined 1,748 food handlers and found ten cases of active tuberculosis and forty-one with evidence of syphilis, of which thirty-seven occurred among waiters.

The prophylactic and therapeutic uses of food are growing subjects. It is only necessary to point out the importance of diet in the prevention and treatment of beriberi, scurvy, pellagra, rickets, tuberculosis, diabetes, acidosis, nephritis, arteriosclerosis, gout, rheumatic affections, disorders of metabolism, dyspepsia, gastric ulcer, infantile diarrheas, and many other affections. Some of the best medicine is bought in the market rather than in the drug store.

Food directly affects growth, nutrition and well-being, and even influences reproduction. The food we eat may increase our resistance or lower our immunity to certain infections. This is clear in the case of tuberculosis, but generalization from this experience leads into error, for it seems diet has little effect on the immunity mechanism of most infectious processes. So far as we know, diet neither raises nor lowers our protection against measles, scarlet fever, diphtheria, whooping-cough, mumps, anthrax, smallpox, yellow fever, rabies, tetanus, and many others. On the other hand, a defective diet which produces scurvy or rickets in turn reduces immunity to a number of infectious diseases and inflammatory states, particularly of the respiratory and intestinal tracts. Our knowledge of this subject is fragmentary and quite incomplete.

Health and efficiency depend more, perhaps, upon the food we eat than upon any other single factor in hygiene. It is therefore a matter of concern that our food sometimes causes injury. This chapter deals with those injuries and their prevention.

How Foods May Be Injurious to Health.—Food may affect health in a great variety of ways:

1. *Natural Poisons.*—Foods may be naturally poisonous, as in the case of certain mushrooms, some fish, or the alkaloids in various species of plants. Small amounts of poison have been extracted from potatoes and other wholesome foods.

2. *Animal Parasites.*—Foods may convey animal parasites, such as trichinæ and tapeworms. These parasites, as a rule, occur as antemortem infections in the flesh of the food animal. Plant foods, too, may carry the eggs or larvæ of various animal parasites, some of which are capable of developing in the human body.

3. *Plant Parasites.*—Foods usually contain bacteria. Both animal and vegetable foods may convey bacteria pathogenic for man. The harmful varieties are more often found in animal foods than upon plant foods. Milk often

¹N. Y. C. Dept. of Health, Mono. Series, No. 17.

contains tubercle bacilli, or streptococci; sometimes diphtheria bacilli, or other pathogenic microorganisms. Typhoid bacilli may be conveyed in oysters, or on celery, water cresses, etc.

The common cause of food infection, or "food poisoning" as it is called, is the Gärtner bacillus (*B. enteritidis*) or one of the other closely allied members of the colon-typhoid group.

4. *Toxins*.—Poisonous substances may develop in the food as a result of bacterial activity. The only example we have in this class is botulism.

5. *Putrefactive Poisons*.—This includes the class of so-called ptomains, or decomposition products of protein. "Ptomain poisoning" is hypothetical, and has not been demonstrated. The term is a misnomer, and its popularity unfortunate. *Infected* food rather than *decomposed* food is apt to cause acute gastro-intestinal attacks.

6. *Special Poisons*.—Foods may contain special poisons, as, for example, solanin in sprouted potatoes, or ergot in rye. There is a substance in buckwheat, activated by light, which may cause serious injury owing to photodynamic action.

7. *Accidental Poisons*.—A great variety of substances may find their way into food, either through accident or intent. They include arsenic, lead, caustic alkalies, acids, alkaloids, adulterants, insect powders, etc.

8. *Amount*.—Injury is caused by eating too much or too little food; thus, an excess of food predisposes to obesity and perhaps to arteriosclerosis and degenerative lesions of the liver and kidneys; an insufficient amount undermines health. Undernutrition in children is common. War edema is due to underfeeding.

9. *Composition*.—An unbalanced diet affects growth, vigor and health. Certain faulty diets lead to beriberi, scurvy, rickets, pellagra and other deficiency diseases. Goiter is due to lack of iodine in water, food, or both. An excessive fatty diet in infants leads to acidosis. An excess of protein favors putrefactive changes. An excess of carbohydrate food favors fermentation. Highly spiced and stimulating diets are irritating both to the digestive tube and organs of excretion. Drinking too little water is a common dietetic error.

10. *Digestion and Metabolism*.—Foods otherwise wholesome may be injurious on account of faulty digestion or disturbances of metabolism. The common causes of such troubles are eating too fast, improper mastication, eating when fatigued or overheated, injudicious combinations, especially unripe fruits or vegetables, containing raw or partly cooked starch, etc.

11. *Anaphylaxis*.—Certain persons have an idiosyncrasy to particular foods. This occurs most commonly with sea food, but also takes place with strawberries, eggs, tomatoes, milk, oatmeal, and a great variety of substances. Many forms of protein in foods produce symptoms resembling anaphylaxis in persons who are sensitized or hypersusceptible. The trouble is not due to any fault with the food, but to a defect in the person. One man's meat may be another man's poison.

THE USES OF FOOD

The two ultimate uses of all food are to supply the body (1) with materials for growth or renewal, and (2) with energy or the capacity for doing work. The potential energy received in a latent form stored in the various chemical combinations in foods is liberated as kinetic or active energy in two chief forms, heat and motion. Force is the manifestation of energy, and the force developed by a healthy man may be measured in foot pounds. A *foot pound* is the amount of energy expended or force required to lift mechanically a weight of one pound to a height of one foot.

The work of an average man is calculated at about 2,000,000 foot pounds per diem. This may exceptionally be increased to 3,000,000 foot pounds. Ordinarily less than one-fifth of the total energy of the body is expended in motion, and more than four-fifths in heat production.

Caloric Value of Food.—From a chemical standpoint foods are oxidized or burned to simple compounds during the process of digestion and metabolism within the body. Food is therefore fuel. A tallow candle produces precisely the same amount of heat whether it burns quickly in the air, or is eaten and burns more slowly in the body. Furthermore, the resulting waste products are similar. Food is good fuel if it fulfills two conditions: easy assimilation and complete combustion. The oxygen to feed the flame is mainly furnished by the air we breathe. In order to get the fullest advantage without injury from food, it must burn without smoking and the combustion must be complete. The best stimulus for complete metabolism is exercise, sunshine, and active breathing of pure fresh air. It is the common experience of all persons that digestion and the utilization of foods are favorably promoted by life in the open air.

Since the discovery of vitamins there has been a tendency to belittle the importance of the caloric value of foods. The maintenance of health and the ability to work and play require food energy, and this is quite as vital to the body as the fuel value of coal is to the engine or gasoline to the automobile (see War Edema). The value of a diet, however, does not depend wholly upon its caloric value and vitamin content.

It is not sufficient to know merely the amount and caloric value of the coal fed to a furnace, and subtract therefrom the amount of unconsumed ash. We must know how much of the heat generated has been *utilized*. The ordinary articles of diet cannot be utilized without vitamins. The importance of these "unknown dietary factors" in promoting the *utilization* of foods is a recent discovery. Complete digestion and metabolism are also important.

Calorie for calorie, foods are not interchangeable so far as nutritive value is concerned. The food calorimeter further gives no indication of digestibility. Concentrated calories may be quite worthless for human physiologic purposes. The comparison of foods on a caloric basis alone may be mis-

leading. The subject is complex; there are many other factors. It even involves the art of preparing, cooking and serving.

Calories Required and Their Estimation.—The total intake of energy into the body is derived from food plus the oxygen of the inspired air. The total output of energy is computed from: (1) the heat of combustion of the unoxidized ingredients of the urine and feces; (2) the energy liberated as body heat; and (3) the energy of external muscular work, or the work of the voluntary muscles.

Two methods may be employed to study the energy-producing power of food in the body: (1) a careful and prolonged study of subjects who are allowed to follow their usual vocations, but whose food and excreta are carefully measured and analyzed; (2) the shorter method of enclosing a man for a brief period, not exceeding a few days, in a cabinet known as a calorimeter.

The unit of measurement is the *calorie*, which is the amount of heat required to raise one kilogram² of water from 15° to 16° C. This equals 3,100 foot pounds, or approximately the heat required to raise the temperature of one pound of water 4° F. *Fuel value* is a term denoting the total number of calories derived from a gram or pound of any given food substance if it is completely burned. The fuel values are calculated for a given food by the factors of Rubner as follows:

4.1 calories per gram of either protein or carbohydrate

9.3 calories per gram of fat

Atwater and Bryant compute the food factors as 4 calories per gram for proteins and carbohydrates and 8.9 for fats, in a mixed diet. C. F. Langworthy gives the fuel value of the three chief classes of nutrients as follows:

1 pound of protein yields.....	1,860 calories
1 pound of fats yields.....	4,220 calories
1 pound of carbohydrates yields.....	1,860 calories

The following are the quantities which are generally accepted at the present time as a sufficient daily diet for a man of average weight, doing a moderate amount of muscular work:

	Weight in Grams	Weight in Ounces	Energy Value in Calories
Protein	100	3½	400
Fat	100	3½	900
Carbohydrates	500	18	2,000
TOTAL			3,300

²This is the large calorie. The small calorie is the amount of heat required to raise one gram, instead of one kilogram of water from 15° to 16° C. The large calorie is the one used in studies of food and metabolism.

Of course the energy value must be greater if more work is done, and conversely, so that we may have such figures as these:

Light work.....	3,000 calories
Moderate work.....	3,500 calories
Heavy work.....	4,000 or more calories

During the World War the United States ration in the field was:

Protein	158 grams
Fat	200 grams
Carbohydrates	514 grams
Energy value, 4,600 calories.	

The minimum caloric requirement (maintenance diet) is about 30 calories per kilogram of body weight.

CLASSIFICATION OF FOODS

Foods are classified in various ways, but no classification is complete. They may be divided into groups, according to their physical properties, their source, their composition, and their function, or the rôle which they perform in the animal body, and their biologic properties.

Physical Properties.—Foods are classed in accordance with their general physical properties first into solid, semisolid, and liquid foods; secondly, into fibrous, gelatinous, starchy, oleaginous, crystalline, and albuminous foods. Foods are also classed as foods, beverages, and condiments. Roughage is cellulose and other indigestible residue.

Sources.—Foods may be classed as to their source primarily into (a) animal, (b) plant, and (c) mineral.

Animal foods consist of meat, fowl, fish, shellfish, crustaceans, insects and their products (honey), eggs, milk and its products, animal fats, gelatin.

The plant foods are subdivided into seeds, roots and tubers, leaves; also cereals, vegetables proper, fruits, sugar, gums, vegetable oils and fats.

The minerals are mainly calcium, potassium, sodium, chlorin, fluorin, iodine, magnesium, phosphorus, iron, sulphur, etc. They are chiefly taken as part of the constituents of animal and plant foods—except sodium chlorid, water and its dissolved salts.

Chemical Composition.—The simplest chemical classification possible is that advocated by Liebig, who was the first to suggest a really scientific definition of foods. He grouped all foods into two classes: nitrogenous and non-nitrogenous. Each of these classes contains food materials from both the animal and vegetable kingdoms, although the majority of the animal substances belong to the nitrogenous and the majority of the vegetable preparations to the non-nitrogenous group.

Nitrogenous foods contain proteins and include gelatinoids and albumi-

noids, substances which resemble albumin. They consist chiefly of the four elements: carbon, oxygen, hydrogen, and nitrogen, to which a small proportion of sulphur and phosphorus is usually joined. The nitrogenous foods were regarded by Liebig as containing plastic elements; that is, they are essentially tissue builders or flesh formers. The non-nitrogenous group Liebig called respiratory or calorifacient foods, because their function in the body is largely to furnish fuel to maintain animal heat. It is now known that the non-nitrogenous foods supply energy for muscular action, hence they are also called force producers, to distinguish them from the nitrogenous or tissue builders. This is a convenient distinction, but it must not be held too absolutely, for the tissue builders are used as force and heat producers as well.

COMPOSITION AND FUNCTION

Food constituents are now ordinarily classed as: (1) proteins, (2) carbohydrates, (3) fats, (4) condiments, (5) inorganic substances, and (6) vitamins.

1. **Proteins.**—Proteins build tissue and repair waste; to a less extent they serve as fuel to yield energy in the forms of heat and muscular power. Meats, milk, eggs, and a few seeds such as the pea and bean are very rich in protein, the cereal grains contain less of this food substance, whereas the tubers and vegetables contain but very little. We now know that not all proteins have equal food value. We have excellent, good or poor food proteins for the formation of body proteins in growth. Hence, the quality as well as the quantity of proteins is important.

Emil Fischer's epoch-marking work between 1899 and 1906 disclosed that proteins, both animal and vegetable, are composed of a series of amino-acids united through their carboxyl and amino groups by elimination of water. When proteins are broken down by hydrolysis, they yield their constituent amino-acids. Most proteins can be resolved by digestion into about seventeen or more amino-acids.³ The proportions in which these are present in the protein molecule varies greatly in proteins from different sources. All or nearly all of these digestion products appear to be indispensable constituents of an adequate diet.

All natural foods contain several proteins, but few of them are complete. The nutritive value of a protein depends upon the number and amount of the amino-acids it yields on artificial hydrolysis or on digestion. Not only must the number of amino-acids be complete, but their relative proportions must correspond to those existing in our body. One protein may supplement the deficiency of another; in other words, adequate protein nutrition may be maintained by mixtures, provided these requirements are met. Proteins are essential elements in the diet because they contain amino-acids which are

³ The following amino-acids were listed by Mathews up to 1921: glycocoll, alanin, valin, isoleucin, leucin, glycoleucin, serin, cystein, aspartic acid, glutamic acid, tyrosin, phenylalanin, tryptophane, prolin, histidin, oxyprolin, arginin and lysin.

the building stones or units out of which the bodily protein structures are put together. The amino-acids must be taken in protein foods, for they cannot be synthesized in the animal organism. According to Mendel, many if not all of these amino-acids are essential for the construction of tissue and the regeneration of cellular losses. Cystin, tryptophane and lysin are indispensable. Even closely related chemical compounds cannot replace lysin. Histidin and arginin are also important.

Casein is a complete protein because it contains all of the seventeen or eighteen amino-acids necessary to rebuild human protein; hence, milk is the best source of protein. Meat protein is also of good quality, but it is not an economical food. The cellular organs such as liver and pancreas are good sources of protein; this is also true of the leafy plants, although the amount they contain is comparatively small. Proteins of seeds are of poor quality because they contain only a few of the amino-acids necessary to rebuild human protein. It is possible by combinations of incomplete proteins to obtain satisfactory dietary results. The great difference in the biologic values of the proteins from different sources is one of the outstanding results of modern research in nutrition.

A diet containing about one hundred grams of protein per day is usually recognized as satisfactory. Voigt and Atwater give 116 to 120 grams of protein per day as the average adult requirement. Chittenden advocated a low protein diet, and got along well on one-third this amount. Experiments prove and experience confirms that a liberal supply of protein, or at least a diet containing a fair margin of safety, serves to maintain vigor throughout the whole span of life. If overdone, however, harm may result, for Newburg's experimental investigations have led him to assume that indulgence in high protein diets will quickly give evidence of renal injury. Bayliss formulated the slogan, "Take care of the calories and the protein will take care of itself."

For a further consideration of the structure of proteins and the relation ofamins to health, see pages 664 to 670.

2. Carbohydrates.—The starchy or carbohydrate foods are represented by the cereals; the tubers, such as potatoes; the sugars of the cane, beet, fruits, etc.; and glycogen in flesh. Cane sugar, beet sugar, starch, glucose, syrup, honey and candy consist almost solely of carbohydrates. They are all lacking in vitamins of every description, with a possible exception of honey.

Carbohydrates are the chief source of heat and energy in our diet. It is a common dietary error to eat too much of this class of food, which favors intestinal fermentation. Carbohydrate foods are for the most part deficient in protein, fat, mineral salts and certain vitamins. The consumption of sugar in this country has increased enormously, and in 1924 amounted to 110 pounds per capita per year. This is believed to be one of the causes of the increase in diabetes.

3. Fats.—Fats and oils are hydrocarbons, and are represented by butter, the fat of meat, the oil of fish and a large number of oils from the plant

world obtained mainly from nuts and seeds, such as olive oil and cottonseed oil. All vegetables contain more or less oily substances. The fats as well as the carbohydrates serve as fuel to yield energy in the form of heat and muscular power.

Certain fats contain a vitamin which is indispensable, whereas other fats do not contain this dietary essential. This vitamin, known as "fat-soluble A," is abundant in the fat of milk, eggs, and is generally found in other fats of animal origin, and also in leafy plants. Little or none is found in lard and oils of plant origin (page 774). The antirachitic vitamin is associated with cod-liver oil. Fats, like proteins, vary in chemical composition, and therefore do not all have the same dietary value.

The chief worth of fat lies in the ease with which it is stored, especially in the connective tissue. Although carbohydrate is also stored, in the form of glycogen, a kind of starch, in the liver and muscles, this takes place only to a limited extent. We notice that both these foodstuffs, fat and carbohydrate, enable a store of reserve material to be kept for use when required to give energy. Fat contains more carbon in the molecule and therefore has a higher fuel value, weight for weight, than carbohydrate.

A very practical value of fat consists in its use in cooking. Very few attractive dishes can be made without it, and it is a physiologic principle that food should be eaten with relish.

The importance of fat in the dietary was demonstrated during the World War. In Germany, the lack of fat was perhaps the cause of the greatest personal discomfort. A diet low in fat does not satisfy. The minimum amount recommended by Armsby, Chittenden, Lusk and Mendel⁴ is given as 75 grams (2 $\frac{5}{8}$ ounces) per day for the average man. This represents about 700 calories, or approximately one-quarter of the average daily energy requirement. The Japanese, however, habitually eat very little fat, and Osborne and Mendel⁵ have succeeded in securing satisfactory growth from an early age to full adult size in experimental animals on rations extremely poor in true fats. In the body fats burn only in the fire of carbohydrates, and therefore derangement of carbohydrate metabolism, as in diabetes, leads to acidosis and serious consequences.

4. **Condiments.**—Among the condiments are classed: spices, such as pepper, mustard, cinnamon, cloves, etc.; also coffee, tea, and alcoholic beverages. Coffee and tea have no nutritive value except for the added sugar and cream. Food flavors have their uses. Attractive and savory dishes promote appetite and digestion by stimulation of the salivary and gastric secretions, sometimes in advance.

5. **Inorganic Substances.**—Mineral matter or ash performs an important service in forming bone and assisting in digestion and metabolism. These substances are ordinarily not classed as foods; however, life cannot be maintained without them.

⁴The Interallied Scientific Food Commission.

⁵*J. Biol. Chem.*, 1920, 45: 145.

Common organic or vegetable acids, such as citric from lemons and oranges, tartaric from grapes, malic from apples, etc., usually exist in combination with the bases, calcium, sodium, potassium, etc., when derived from fresh vegetables and fruits. They are indispensable articles of food, for when absorbed they form carbonates, which aid in maintaining the normal reaction of the blood.

If calcium phosphate is deficient in the food of the young, growing infant, the bones are poorly developed and so soft that they become bent, as occurs in rickets. The ordinary diet of Americans and Europeans is probably more often deficient in calcium than in any other chemical element. Calcium metabolism probably has an important bearing upon the prevention and cure of tuberculosis. In 1909, MacCallum and Voegtlin⁶ showed that the symptoms observed after removal of the parathyroid gland are associated with a deficiency of calcium. This brings about tetany, which may be relieved simply by the administration of calcium. Of all foods, milk is the best source of calcium, as it contains this element in abundance and in utilizable form. Milk, however, is deficient in iron, and this fact must be taken into account when used as a sole article of diet for growing children or adults.

The body demands small but persistent amounts of iodine, iron, phosphorus, sulphur and other elements that enter into its composition. If these are lacking, serious trouble ensues. Insufficient iodine leads to goiter, insufficient iron to anemia, insufficient phosphorus to osteomalacia, etc.

Lack of inorganic salts in the food impoverishes the coloring matter of the red blood-corpuscles on which they depend for their power of carrying oxygen to the tissues, and anemia and other disorders result. An ash-free diet soon causes serious symptoms. The ordinary American diet, consisting of cereals, bread, meat, and potatoes is rather lacking in mineral constituents, especially calcium. Longworthy gives the following as the estimated amount of mineral matter required per man per day:

Phosphoric acid (P_2O_5).....	3	to 5	grams
Sulphuric acid (SO_3).....	2	to 3.5	grams
Potassium oxid.....	2	to 3	grams
Sodium oxid.....	4	to 6	grams
Calcium oxid.....	0.7	to 1.0	grams
Magnesium oxid.....	0.3	to 0.5	grams
Iron	0.0006	to 0.012	grams
Chlorid	6	to 8	grams
Iodin	0.00043		grams

6. **Vitamins.**—Vitamins promote growth and favor the utilization of food. On account of their importance, a brief summary of our knowledge on this subject follows.

⁶ *J. Exper. M.*, 1909, 11: 118.

VITAMINS

Vitamins are "accessory factors" in the diet necessary for growth and metabolism. They are "unknown dietary factors" essential to life. Vitamins are remarkable in that exceedingly small amounts produce extraordinarily great results—in this respect resembling enzymes. Their chemical nature is not known. Seidell⁷ has obtained a crystalline picrate having the antineuritic properties of vitamin B. There was a tendency in the past to regard vitamins as substances comparable to enzymes and toxins in their instability and marked activity of infinitesimal doses. Seidell has shown, however, that the antineuritic vitamin performs its functions in convenient amounts and withstands laboratory manipulations. Vitamins are produced in the plant world even by simple unicellular cells such as yeast. Most of them cannot be elaborated nor stored by man or other mammals. Growth, nutrition and utilization of food therefore depend upon the daily intake of vitamins. In this sense, we live a hand-to-mouth existence, and are parasitic on the plant kingdom. The story of the discovery of vitamins is absorbing.

In 1896, Hopkins, an Englishman, concluded that animals required something in their foods other than the common nutrients, and called these hypothetical substances "accessory food factors." In 1897, Eijkman,⁸ a Dutch chemist, showed that polyneuritis could be induced in fowls by restricting them to a diet of polished rice, and that a diet of uncorticated rice would cure fowls of this condition. In 1907, Fraser and Stanton⁹ showed that the alcohol extracts of rice polishings would relieve experimental polyneuritis.

Osborne and Mendel,¹⁰ in 1909, started a series of experiments involving the study of the effect of feeding purified proteins and mixtures thereof with other purified nutriment. They called attention to the peculiar effect upon growth induced by "protein-free milk." In 1911, Funk¹¹ took up the problem and showed that pressed yeast, hydrolyzed with 20 per cent sulphuric acid for twenty-four hours retained its property of curing polyneuritis when given to birds, from which he concluded that in yeast and in rice polishings there was present a chemical entity of a nitrogenous basic nature, which he called "vitamin." The antineuritic property of this substance was soon confirmed, but its chemical nature was not established. To Funk is due the credit for calling attention to the fact that certain diseases are due to the lack of a vitamin. In 1912, McCollum and Davis¹² showed that butter fat and egg yolk contain something that stimulates growth and is absent in lard and olive oil. Later, McCollum and his associates showed that this principle "fat-soluble A" is present especially in milk, eggs and leafy vegetables. Drummond¹³ suggested that the antiscorbutic vitamin be classified as "water-soluble C." From here the subject grew by leaps and bounds.

⁷ *U. S. Pub. Health Rep.*, 1924, 39: 294.

⁸ *Lancet*, 1909, 1: 451; 1910, 733.

¹¹ *J. Physiol.*, 1911, 43: 395; 1912, 45: 75.

¹³ *Lancet*, 1918, 2: 482.

⁸ *Virchow's Arch.*, 1897, 149: 187.

¹⁰ *Carnegie Inst. Bull.*, 1911, Pt. II, 156.

¹² *J. Biol. Chem.*, 1913, 15: 167.

It soon became evident from the standpoint of chemical nomenclature that the term vitamin was, in some respects, ill chosen. Subsequent studies have shown that vitamins are not amins, but the term is attractive and is now established by usage.

For the most part the vitamins are stable and heat resistant. Vitamins A and C are thermolabile, although it is not the heat so much as oxidation which causes their destruction. Vitamin B is notably resistant to heat at boiling temperature.

Our knowledge of the vitamins to date is contained in McCollum's *Newer Knowledge of Nutrition*, Funk's *The Vitamins*, and Sherman and Smith's *The Vitamins*.

There may be a large number of vitamins, but only five or six are clearly recognized:

1. **Vitamin A**, or "*fat-soluble A*," is associated with certain fats, and is especially abundant in milk, butter, egg yolk, the fat of glandular organs, and also in the leaves of plants. The seeds of plants contain less, and products derived from the endosperm of the seed are very poor in this substance. Thus, such foods as bolted flour, degerminated corn meal, polished rice, starch, glucose and the sugars from milk, cane and beet, are practically free of this vitamin. It is practically absent from lard and the fats and oils of vegetable origin and non-pigmented fats generally.

Milk and the green leaves of plants can therefore be regarded as "protective" foods, and should never be omitted from the diet. Milk is a better protective food than are the leaves, because it contains a larger amount of this vitamin.

The specific result of a lack of a sufficient amount of this vitamin in the diet of experimental animals is the development of xerophthalmia. The eyes become swollen so badly that they are opened with difficulty or not at all. The cornea becomes inflamed, and unless the missing dietary essential is supplied, blindness follows. The condition has been noted in man.

2. **Vitamin B**, the *antineuritic vitamin*, "*water-soluble B*," promotes growth, and its absence induces polyneuritis (beriberi). This vitamin is widely distributed in all sorts of natural foods and can be isolated in a concentrated, but not in a pure form, by extraction with alcohol or water. It is found in animals, seeds, leaves and tubers, but it is never associated with fats or oils of either animal or plant origin. Our ordinary foods contain several times the amount of vitamin B which is necessary for the maintenance of growth and health in animals. Vitamin B is probably a mixture of several vitamins, including a pellagra preventing factor (P.P.).

3. **Vitamin C**, the *antiscorbutic vitamin*, "*water-soluble C*," when deficient is the cause of scurvy. This vitamin is widely distributed in nature, but in variable amounts in different foods. It is particularly abundant in oranges and tomatoes. It is more sensitive to heat, oxidation, drying, etc., than the other vitamins, and is therefore especially associated with fresh foods. The subject is discussed in detail under Scurvy, page 626.

VITAMINS IN FOODS

Source	A	B	C	Source	A	B	C
<i>Grain Products</i>				<i>Meats, Fish</i>			
Barley, whole	+	++	—	Meat (muscle)	—to +	+?	+?
Bread, white (water)	?	+	—	Meat extract	—	—?	—
Bread, white (milk)	+	+	?	Meat, canned	—	slight	—
Bread, whole wheat (water)	+	++	?	Roe, fish	+	++	+?
Bread, whole wheat (milk)	++	++	?	Sweetbreads	+	+	*
Corn (maize), white	—	++	—	<i>Fruits</i>			
Corn (maize), yellow	—	++	—	Apples	+	+	+
Cottonseed meal	+	++	*	Bananas	+?	+?	+
Flour, white	—	+	—	Cloudberries	*	*	+++
Grains, sprouted	+	+++?	++	canned	*	*	+++
Malt, green	+	+++?	++	Cocum (dried)	*	*	+
Millet	++	++	—	Grape juice	*	+	+
Oats	+	++	—	Grapefruit	*	++	++
Rice, polished	—	—	—	Lemon juice	*	++	+++
Rice, whole grain	+	++	—	juice dried	*	++	+++
Rye, whole	+	++	—	Limes	*	+	+
Wheat embryo	++	+++?	—	Mango	*	*	+
endosperm	—	+	—	Mulberries	*	*	+
middlings, commercial	*	+++?	—	Orange juice	+	++	+++
bran	+	+	—	peel	+	+	++
whole	+	++	—	Pears	*	+	—
<i>Sugars, Starches</i>				Prunes	*	*	—
Glucose	—	—	—	Raspberries	*	*	+++
Honey	—	+	—	canned	*	*	+++
Starch	—	—	—	Tamarind, dried	*	*	+
Sugar	—	—	—	Tomatoes, raw	++	+++	+++
<i>Fats, Oils</i>				canned	++	+++	+++
Beef fat	+	—	—	dried	++	+++	++
Butter	+++	—	—	<i>Vegetables</i>			
Cocanut oil	+	—	—	Alfalfa	+++	+++	*
Cod-liver oil	+++	—?	—	Beans, kidney	*	+++	*
Corn oil	+?	—	—	navy	*	+++	—
Cottonseed oil	+?	—	—	soy	+	++	—
Horse fat	+	—	—	sprouted	+	+	++
Lard	+?	—	—	string, fresh	++	+	++
Linseed oil	+	—	—	Beets	*	+	*
Margarine, oleo	+	—	—	Cabbage, fresh raw	+	+++	+++
Margarine, nut	—	—	—	cooked	+	++	++
Mutton fat	+	—	—	dried	+	++	+
Olive oil	—	—	—	green	++	+	+++
Oleo oil	+	—	—	Carrots, fresh raw	++	++	++
Orange peel oil	++	*	*	cooked	++	+	+
Palm oil	+	—	—	Cauliflower	+	++	+
Peanut oil	—	—	—	Celery	*	+	*
Pig kidney fat	++	—	—	Cress	*	*	+
Whale oil	++	*	—	Chard	++	+	*
<i>Meats, Fish</i>				Cucumber	*	+	*
Brains	+	++	+?	Dandelion greens	++	++	+
Fish, lean	—	+	*	Dasheens	—?	+	+
fat	+	+	*	Eggplant (dried)	*	++	*
Heart	+	+	+?	Endive	+	*	+
Kidney	++	++	+	Legumes, sprouted	+	*	++
Liver	++	++	+	Lettuce	++	++	+++
				Onions	*	++	+
				Parsnips	—?	++	*
				Peas	++	++	+?
				sprouted	*	*	++

NOTE:

- + indicates that the food contains the vitamin.
- ++ indicates that the food is a good source of the vitamin.
- +++ indicates that the food is an excellent source of the vitamin.
- indicates that the food contains no appreciable amount of the vitamin.
- ? indicates doubt as to the presence or relative amount.
- * indicates that evidence is lacking or appears insufficient.

VITAMINS IN FOODS (Continued)

Source	A	B	C	Source	A	B	C
<i>Vegetables</i>				<i>Nuts</i>			
Potatoes, sweet ...	++	+	*	Pine nuts	+	+	*
white, raw	+	++	++	Walnuts, black ...	*	++	*
white, boiled (15 min.)	*	++	++	Walnuts, English ..	*	++	*
white, boiled (1 hr.)	*	++	+	<i>Milk</i>			
white, baked ...	*	++	+	Milk	+++	++	+ variable
Radish	*	+	*	condensed	+++	++	+ variable
Rhubarb	*	*	+	evaporated	+++	++	-?
Rutabaga	-?	++	+++?	dried, whole	+++	++	+ variable
Sauerkraut	*	+	-?	dried, skim	+	++	+ variable
Spinach, fresh ...	+++	+++	*	Skimmed milk	+	++	+ variable
dried	+++	++	*	<i>Dairy Products</i>			
Squash, Hubbard ..	++	+	*	Butter	+++	-	-
Swede	*	++	+++?	Buttermilk	+	++	+ variable
Turnips	-?	++	*	Cream	+++	++	+ variable
<i>Nuts</i>				Cheese	++	*	*
Almonds	+	+	*	Cottage cheese ...	+	*	*
Brazil nuts	-?	++	*	<i>Eggs</i>			
Chestnut	*	+	*	Eggs	++	+	+?
Cocoanut	+	++	*	white	*	*	*
Cocoanut press cake	+	++	*	yolk	+++	+	*
Filberts	*	++	*	<i>Yeast</i>			
Hickory nuts	*	++	*	Yeast	-	+++	-
Peanuts	-	++	*	Yeast extract	-	+++	-
Pecans	*	+	*				

4. **Vitamin D**, the *antirachitic vitamin*, the absence of which leads to rickets, is associated especially with oils and fats, particularly the oil of fish. It is most abundant in cod-liver oil. Vitamin D is discussed in detail under Rickets, page 633.

5. **Vitamin E**, the *reproductive vitamin* of Evans and Bishop, is present in lettuce, meat, egg yolk, beef liver, whole wheat, wheat germ, rolled oats, and dried alfalfa. There is very little of it in milk, although large quantities of milk fat will relieve the disturbances of the *astrus* in adult female rats, which is brought about by deficiency in this dietary factor.

6. **Vitamin P.P.**, or the *pellagra preventive vitamin* of Goldberger, is a new dietary factor particularly associated with yeast. See Pellagra.

Our knowledge of the vitamins is still so imperfect that precise statements concerning the vitamin content of many of our common foods cannot be made; in fact, different samples of the same food may vary considerably in their vitamin content. The foregoing table¹⁴ gives the relative values of different articles of food as sources of vitamins so far as known. The vitamin content of each food is indicated roughly, as we are not justified in using numerical expressions of relative values.

It is comparatively easy to prepare from ordinary wholesome foods diets which will contain several times the minimum amount of vitamins on which

¹⁴ Taken from Sherman and Smith's book, *The Vitamins*, and based on Report No. 38 of the British Medical Research Committee, modified by M. S. Rose, *Laboratory Handbook for Dietetics*, 1921.

normal nutrition can be maintained over considerable periods. Investigators are agreed, therefore, that there is no need for commercial vitamin preparations. In view of the incompleteness of our knowledge, the best plan is to eat a varied diet judiciously chosen.

Immunologic Significance of Vitamins.—It is now clear that vitamins are not only essential factors for growth and nutrition, but that they have an important bearing on resistance and immunity. It has long been known that in certain forms of avitaminosis, such as scurvy and rickets, there is increased susceptibility to certain infections, particularly of the respiratory and digestive tracts; in fact, both these diseases, as well as pellagra and beriberi, have long been regarded as bacterial infections. Werkman, Baldwin and Nelson¹⁵ state that rats suffering from the lack of vitamins A or B are more susceptible to diphtheria toxin. Smith, McClosky and Hendrick¹⁶ studied the toxicity of a number of pharmacologic agents in vitamin-deficient rats. They found increased susceptibility to pilocarpin and ergotoxin in vitamin B deficient animals; they also found a lowered resistance to ergotoxin and to morphin in rats on vitamin A deficient diet. There was a definite though slight increase in susceptibility to histamin, ouabain and to the alkaloids strychnin, atropin, cocain, and apocodein.

Irradiated Foods.—It now appears that radiant energy may be stored in food either as it grows in the field or by artificial exposure to rays of short wave length. Steenbock and Daniels¹⁷ and Hess and Weinstock¹⁸ found that foods after radiation became active in preventing rickets. Steenbock and Daniels state that by exposing such food materials as wheat, rolled oats, corn, hominy, cream of wheat, shredded wheat biscuits, corn flakes, patent wheat flour, cornstarch, meat, milk and egg yolk to ultraviolet light, they can be endowed with rickets-preventing properties. Apparently it is the lipoidal constituents which carry this activation. Hess and Weinstock exposed cottonseed oil and linseed oil to the mercury vapor lamp and found they became bottled sunshine; further, that the irradiated oils were able to store the antirachitic factor for considerable periods. Plant foods may or may not contain this stored energy, depending on whether or not they have sunlight during growth. Finally, it has been shown that antirachitic oils have radioactive properties, which are not possessed by inactive oils (see Rickets, page 633).

THE AMOUNT OF FOOD

Excessive Amounts.—The amount of food required varies greatly with conditions. In favored communities, where cooking is a fine art, the number and variety of food preparations are so great that the appetite is often stimulated beyond the requirements of the system, and consequently more food may be eaten than is necessary or desirable to maintain the best bodily health and vigor. Gluttony results in overdevelopment and overwork of

¹⁵ *J. Infect. Dis.*, 1924, 35: 549.

¹⁷ *J. Am. M. Ass.*, 1925, 84: 1093.

¹⁶ *U. S. Pub. Health Rep.*, 1926, 41: 767.

¹⁸ *Am. J. Dis. Child.*, 1924, 28: 517.

the digestive apparatus; the stomach and bowels become enlarged; the liver is engorged, and a disposition is established to obesity followed by degenerative changes, fatty heart, etc. Overeating is also supposed to favor high blood-pressure and arteriosclerosis. The quantity of food required to maintain the body in vigor varies with the climate and season, clothing, occupation, work, and exercise, the state of individual health, age, sex, and body weight.

Both overeating and overdrinking may be temporary or chronic. When chronic they may lead to obesity, gout, oxaluria. It is very certain to cause congestion of the liver and the condition known as "biliousness," in which the stomach and intestines are engorged, constipation results, the tongue is heavily coated, the bodily secretions are altered in composition, the urine especially becomes overloaded with salts, the liver becomes congested, and, finally, the nervous and muscular systems are affected, which result in the production of headache and feelings of fatigue, lassitude, drowsiness, and mental stupor. An excess of protein favors putrefactive changes in the intestinal tract and irritates the kidneys; an excess of carbohydrates tends to fermentation; an excess of fat leads to acidosis, especially in babies. Excessive starch and sugars in the diet may serve as an exciting cause of diabetes, especially in persons who might otherwise go through life without developing the disease. Individuals who have a tendency to obesity, high blood-pressure and low sugar tolerance, especially with a family history of diabetes, should watch their urine for sugar and keep their diet well within their sugar tolerance. The body can successfully burn more sugar with muscular exercise, therefore diabetes is often associated with luxury, ease and indulgence.

Obesity is usually due to errors of metabolism or heredity rather than to bad dietary habits. Overweight of fifteen pounds or more is an increasingly serious condition with advancing age.

Insufficient Food.—*Starvation* or *asitia* is a term which technically applies to the lack of sufficient food for the maintenance of the body, while *inanition* means the lack of the assimilation of food by the tissues. When food is completely withheld, life cannot be prolonged beyond six or ten days in the majority of instances. Professional fasters have gone forty-one days without anything but water. If food is withheld suddenly, the sensation of hunger gradually increases, becomes extreme, lasts for two or three days, and slowly disappears. It is accompanied by a gnawing pain in the epigastrium, which is relieved on pressure. The pain may disappear, but it is followed by a sensation of extreme weakness or faintness, which is both local in the stomach and general throughout the body. Even though the pain disappears, the sensation of hunger may occasionally reassert itself, when all food is withheld, until death, or until the subject becomes insane or unconscious.

Hunger is not always a reliable guide to the need of the system for food. Some dyspeptics are always hungry and eat more than they can digest. A habit of rapid eating does not satisfy the sensation of hunger. More food may be taken than is necessary, because it has not had time to meet the needs of the system before the meal is over. Cannon has shown that the sensations

of hunger come and go rhythmically, appearing synchronously with the contractions of the empty stomach.

A reduced diet causes a lowering of basal metabolism, influences the sex instinct and its manifestations. There results a lessening of sex interest and a diminution in procreative power.¹⁹

Undernourishment.—A large percentage of growing children are underweight for their height. Judged by standard height-weight tables, from 15 to 40 per cent, and even as high as 60 per cent of school children have been found to be undernourished. In a study of 506 children selected as the best specimens of health that could be found among school children, it was found²⁰ that one-fifth of them were underweight according to the standards most frequently used in school health work in the United States. Twenty per cent of these children were more than 10 per cent underweight according to the Wood standard height-weight-age tables; 13 per cent were more than 10 per cent underweight, according to Dreyer's standard stem length and chest circumference tables. Many factors, however, affect the height-weight relationship in addition to nourishment, particularly race and family trend as to body build. Undernourishment has been found to be even more prevalent among the children of the well-to-do than those of the poor. The causes of undernourishment are (1) physical defects and pathological processes, (2) lack of home control, (3) chronic overfatigue, (4) improper health habits, and (5) improper diet and food habits. To remedy the condition requires individual diagnosis of the cause in each instance, and success often depends upon the coöperation of the parent, the teacher, the physician and the child (see also page 1279).

Underfeeding and Growth.—Failure to grow according to normal expectations may result from underfeeding, from inappropriate food, or from pathological defects either inherited or acquired. Inborn errors of growth form a special category which falls outside the sphere of nutrition. Retardation of growth due to diets defective in quality rather than quantity will be discussed under diseases due to vitamin deficiency. Underfeeding per se will retard or suppress growth, but Osborne and Mendel²¹ have shown that the capacity to grow is not entirely lost, but is held in reserve until it is exercised. Even when the period of stunting through underfeeding is prolonged beyond the time when growth usually ceases, the institution of a proper diet may result in prompt resumption of growth and apparent completion of its usual cycle.

Famine and Pestilence.—When starvation occurs upon a large scale, affecting a community with famine, pestilence is sure to accompany it. Famine and fevers go hand in hand; in fact, it is a common belief that famine is the direct cause of pestilence. Thus, disease has often been rampant in Ireland when the potatoes have failed, and in India when the grain supply has given

¹⁹ *J. Nerv. & Ment. Dis.*, 1919, 49: 3.

²⁰ Clark et al., *U. S. Pub. Health Rep.*, 1923, 37: 1239.

²¹ *J. Biol. Chem.*, 1915, 23: 439.

out. Much of the illness which occurred in the early history of the Crimea was coincident with insufficient food, and it is stated that in the Middle Ages the ravages of pestilential diseases, such as typhus, smallpox, plague, etc., were always worse in times of general starvation. We had a recent example in the famine throughout the Volga region of Russia, which was accompanied by typhus fever, cholera and relapsing fevers. The history of epochs of famine in siege or otherwise is always accompanied by outbreaks of violence, for hunger begets ill temper, vice, and crime. This has occurred notably in Athens, Florence, and London, and in Paris during the Commune. There is, however, no direct causal relationship between famine and epidemics. The "depressed vitality" caused by insufficient food is not the real cause of epidemics of plague, smallpox, relapsing fever, typhus fever, and other pestilential diseases, sometimes called famine fevers.

Starvation does not depress immunity to all diseases, but lowers resistance only to certain infections. When food is lacking in quantity, it is also defective in quality, and while this combination undermines vitality, it lowers resistance only to some infections, such as tuberculosis and intestinal diseases. The so-called "famine fevers" are not due to increased susceptibility, but indirectly to factors favoring transmission. Famine is accompanied by lack of sanitation and hygiene, and by misery, squalor and an increase of lice, fleas and other vermin and many other factors that favor the spread of typhus fever, plague, relapsing fevers, etc.

War Edema.—The symptoms of war edema are gravitational dropsy, slow pulse, low blood-pressure and polyuria. The condition is associated with prolonged undernutrition, due to lack of calories over a long period of time. The disease is therefore aggravated by cold and exertion and is improved by rest and warmth. Many persons in the war area had a daily ration of only about 1400 calories, consisting mainly of a coarse food, chiefly carbohydrates, very little protein, and almost no fat. All the symptoms can be explained by lack of function of the thyroid gland. Clinically the condition resembles myxedema.

It occurred especially in Germany, Russia, Rumania, but also in other countries during the Great War. When advanced, the symptoms are edema of legs, thighs and genitalia, with some puffiness of the eyelids. In bad cases there is edema also of the chest and abdominal walls. Patients complain of general weakness and pain in the legs. They are usually very apathetic. There is extreme muscular weakness and marked pallor. The whole appearance resembles the condition found in chronic parenchymatous nephritis. Uncomplicated cases are afebrile. With rest in bed and a better ration, improvement slowly takes place.

War edema is clearly the result of underfeeding. The ration responsible was low in protein and almost fat-free. Thus, in Berlin the poorer classes were reduced to a ration containing only about 50 grams of protein per day, which is about half the standard dietary observed in most civilized countries. The fats were replaced by carbohydrates. The ration was largely vegetable

in origin, rich in cellulose, devoid of stimulating properties, and limited in variety. It contained only about 1400 calories.

In addition to war edema, other results were traceable to the restricted diet. Hernias and displacements of internal organs were a frequent consequence of emaciation. Lactation was unsatisfactory among childbearing women. Gastro-enteric disorders, rickets, and other deficiency diseases occurred, and delayed growth affected the children. Tuberculosis increased its ravages, and decreased resistance to other diseases was noted.

BALANCED DIETS

A balanced diet is necessary for the full enjoyment of health as a positive quality. It is one that furnishes the optimum condition for the maintenance of vigor and the characteristics of youth; it must promote growth, favor utilization of food and provide for normal reproduction. The science of nutrition has emerged from its simpler beginnings into a complex problem of great intricacies. Attention was first called to the importance of the *chemical* composition of foods, and stress was placed upon the importance of the diet containing protein, carbohydrates and fats. To this was later added the necessity of certain inorganic salts. Now we know that some proteins are incomplete and inadequate for nutrition; the fats also have different dietetic values. The *caloric* value of food was next emphasized, but it soon became apparent that many considerations other than fuel value make up a balanced ration. *Biologic* studies then revealed the vitamins, necessary for growth and nutrition.

Some of the factors which make up a balanced diet are: sufficient calories to furnish heat and energy; inorganic salts, and iron, phosphorus and calcium and iodine; adequate quantity and variety of foods containing all the necessary vitamins; some "ballast" or "roughage," that is, undigestible residue such as cellulose. Furthermore, a balanced diet should provide approximately the same fuel value each day. In other words, we should not have a feast and upset our digestion by overeating to-day, and have a famine to-morrow, but stoke the furnace regularly, according to its needs. Certain wholesome and nutritious foods contain small quantities of toxic substances and may cause harm if eaten in inordinate amounts.

A balanced diet must also take into account the acid-base equilibrium in order to avoid acidosis or alkalosis. The urine ordinarily is acid, and this acidity is increased when acids are taken. The feces are alkaline. The action of the bowel is in the main just opposite to that of the kidney. The kidney normally excretes acid, the bowel excretes the alkali in excess. Acid is excreted almost wholly by the kidney; alkali is excreted partly by the kidney and partly by the bowel. The meat and bread portion of our diet when oxidized in metabolism leads to the formation of acid over base forming substances. Fruits and vegetables tend to the establishment of the proper acid-base equilibrium. It is plain, then, that a balanced diet depends upon many

factors, some of them exceedingly complex. Our only safety lies in a generous ration, varied as possible. A wide variety provides a factor of safety. Growing children especially should have a safe margin both as to quantity and quality, for the science of nutrition is in its infancy.

A balanced diet furthermore consists not only of a liberal consumption of all the essential constituents of a normal ration, but also includes prompt digestion and absorption; also normal metabolism and evacuation of the undigested residue. Such a diet can be attained only by supplementing the seed products, tubers, roots and meats, which constitute the bulk of the diet of man, with milk and the leafy vegetables. Milk and the leafy vegetables are called *protective foods*, because they are rich in vitamins. Under the artificial conditions of civilization, instinct is not always a safe guide for the balanced ration.

THE DEFICIENCY DISEASES

Defective diets give rise to the deficiency diseases, due principally to vitamin starvation. The deprivation of each vitamin acts in a rather specific or characteristic way. Thus, the absence of vitamin A induces xerophthalmia; vitamin B, beriberi; vitamin C, scurvy; vitamin P.P., pellagra; and vitamin D is sometimes associated with rickets. There are defective elements in the diet other than vitamin starvation which complicate the pathological picture. Furthermore, deficiency diseases may arise from the lack of dietary elements other than vitamins, as iodine and goiter.

BERIBERI (Polyneuritis)

Our knowledge of beriberi ²² is sufficient to place this scourge of the tropics among the preventable diseases. It is now evident that beriberi is a disease due to an unbalanced diet, deficient in a specific vitamin, vitamin B. It is our best known example of a deficiency disease. Most nations where beriberi prevails subsist on a monotonous and one-sided diet, made up largely of polished rice, that is, rice without the pericarp. The disease may be prevented or cured by the administration of rice bran, or other substances containing the specific vitamin.

Vitamin B and Beriberi.—The particular vitamin associated with beriberi is also known as “antineuritic vitamin,” or “water-soluble B.” A certain amount of this accessory dietary factor must be in the ration in order to permit normal growth of the young and the maintenance of weight and health of the adult animal and man. Its absence from the diet causes polyneuritis and other symptoms of beriberi. This vitamin is very resistant to drying, heat, and other influences, but may be weakened by heating in the presence of an alkali.

²² Vedder's book, *Beriberi*, Wm. Wood & Son, 1913, contains a complete description and bibliography of the disease.

Occurrence.—Beriberi, or kakke, is a specific form of multiple peripheral neuritis occurring endemically, or as an epidemic, in most tropical or sub-tropical climates. The disease is believed to be of great antiquity in China. It is specially prevalent among the Malays, Chinese, Japanese, and during the Russian War more than 50,000 cases occurred in the Japanese army. It is common in the Philippines, and is known in India and Australia. It prevails in parts of South America and the West Indies and is met with among the fishermen of Norway; and off the Newfoundland Banks.

Symptoms.—Beriberi is characterized clinically by disturbances of motion, sensation, dropsy, and affection of the heart. The symptoms are attributable to degenerative changes in many of the peripheral nerves, being a toxic neuritis similar in many respects to that produced by alcohol, arsenic, and other poisons, such as the toxon of diphtheria. The pneumogastric nerve is also involved, which accounts for the heart symptoms. Three types of the disease are recognized: (1) the paraplegic, or dry, (2) the dropsical, or wet, and (3) the mixed. The course of the disease is uncertain; sudden death owing to involvement of the heart is a common termination. Recovery is frequent and may be complete; it is promoted by change of climate and improvement in the sanitary surroundings, but is dependent upon change to a wholesome diet containing antineuritic vitamin.

Rice and Beriberi.—Many physicians who have studied the subject in Japan, Java, the Philippines, and other countries have long regarded rice as the important cause of the disease. In the prisons of Java the proportion of cases was 1 to 39 when rice was eaten completely shelled; 1 to 10,000 when the grain was eaten with its pericarp. In many places the disease has disappeared when the unshelled rice has been substituted for the shelled.

Eijkman, in 1897, showed that a disease resembling beriberi, characterized by degeneration of the peripheral nerves, may be produced in fowl by feeding them on white or polished rice. These results were later confirmed by Grijns (1900) and Halshoff Pol (1904), but a great impetus was given to the study of the disease by Fraser and Stanton²³ who, in 1909 to 1911, clearly demonstrated that the disease is brought about by a one-sided diet of white or polished rice. These investigators took three hundred Japanese laborers into a virgin jungle, where they occupied new and sanitary quarters. After excluding the existence of beriberi by a careful examination of each person, they were divided into two parties of equal numbers. One party received polished rice as the staple article of diet, while the other party received undermilled rice with pericarp. In three months beriberi appeared among the members of the party receiving polished rice. When a certain number of cases had been noted, polished rice was discontinued, and thereafter no cases occurred. No sign of the disease appeared among the party receiving undermilled rice. The conditions were then reversed. The party hitherto on undermilled rice were given polished rice, and after a somewhat longer interval

²³ *Studies from the Institute of Medical Research, Federated Malay States, 1909, No. 10.*

beriberi broke out in this group also. This outbreak also ceased on discontinuing the issue of polished rice. Again no sign of the disease appeared among the control party receiving undermilled rice. Place infection and communicability were excluded by transferring individuals suffering from beriberi from one group to the other from time to time. This experiment was later repeated by Strong and Crowell²⁴ in the Philippines with a similar result.

The same changes in diet which avoid or cure beriberi in man act in a similar manner with respect to polyneuritis in fowl. It has now been established that polished rice causes beriberi if the diet is based almost exclusively on this foodstuff, but that, if a sufficient amount of other things, such as fresh meat and vegetables, are taken with it, the disease is not produced. In the polishing of rice the pericarp or cortical portion of the grain is removed and the embryo is discarded. It is evident that these discarded portions contain some substance (vitamin) essential to a well-balanced ration. It has been found that most of the phosphorus is contained in the pericarp. The amount of phosphorus is a good guide in the selection of a beriberi-preventing rice. In the East, rice is regarded as unsafe if it contains upon analysis a content of less than 0.35 per cent of phosphorus pentoxid. It is not, however, the absence of the phosphorus which induces beriberi, but the amount of phosphorus, as phosphorus pentoxid (P_2O_5) may be taken as an index of the degree to which the rice has been polished.

Funk,²⁵ in 1911, isolated a substance from rice polishings that prevents and cures polyneuritis gallinarum. Funk obtained a crystalline organic base ($C_{17}H_{20}N_2O_7$) for which he invented the term "vit-amin." The chemical nature of this accessory dietary factor remains undetermined. Seidell²⁶ obtained from yeast a crystalline substance which appears to have the properties of vitamin B.

The work of Fraser and Aron, Breaudat and Denier, Dehaan, Heiser, and others leaves little doubt concerning the relation of polished rice to beriberi. From 15 to 40 grams of unpolished rice in the daily diet prevents and cures the disease. The results in the Philippines based upon the rice theory are little short of marvelous, and equally good results of this character are reported by Van Leent,²⁷ Vorderman,²⁸ Fletcher,²⁹ Hightet,³⁰ Theze,³¹ Chamberlain,³² and others.

Despite the fact that beriberi is the best example we have of a specific avitaminosis, some students of the disease from time to time insist that the

²⁴ *Philippine J. Sc.*, 1912, 7: 271.

²⁵ *J. Physiol.*, 1911, 43: 26; also Casimir Funk, *Die Vitamine*, Wiesbaden, 1914.

²⁶ *U. S. Pub. Health Rep.*, 1924, 39: 294.

²⁷ *Arch. de méd. nav.*, Oct. 1897, 241. *Cong. internat. d. sc. méd.*, Amst., 1880, 6: 170; etc.

²⁸ Onderzoek, etc., of Java en Madoera, van Beriberi, Batavia, 1897.

²⁹ *J. Trop. Med. & Hyg.*, 1909, 12: 127; also *Lancet*, London, 1907, 1: 1776.

³⁰ *Philippine J. Sc.*, 1910, 5: 73.

³¹ *Ann. d'hyg. et de méd. colm.*, 1910, 13: 16.

³² *Philippine J. Sc.*, 1911, 6: 133.

etiology is not so simple and that there are other factors involved. Thus, McCarrison,³³ who studied beriberi in India for five years, believes that vitamin deficiency is rarely the sole agent in the cause of the disease. It was long considered an infectious disease and this theory still persists. The same is true of rickets, scurvy and other deficiency diseases.

Infantile beriberi is one of the causes of high infantile mortality in the Philippines and in some other places. The disease is brought on by the baby's being suckled at the breast of a mother suffering from beriberi. The lines to be followed in prevention and cure are the same as in adult beriberi.

Rice.—A grain of rice, after removal from the husk, consists of three parts: (1) An outer layer called the pericarp, which is a very thin membrane. The color of the pericarp varies in different species of rice, from white

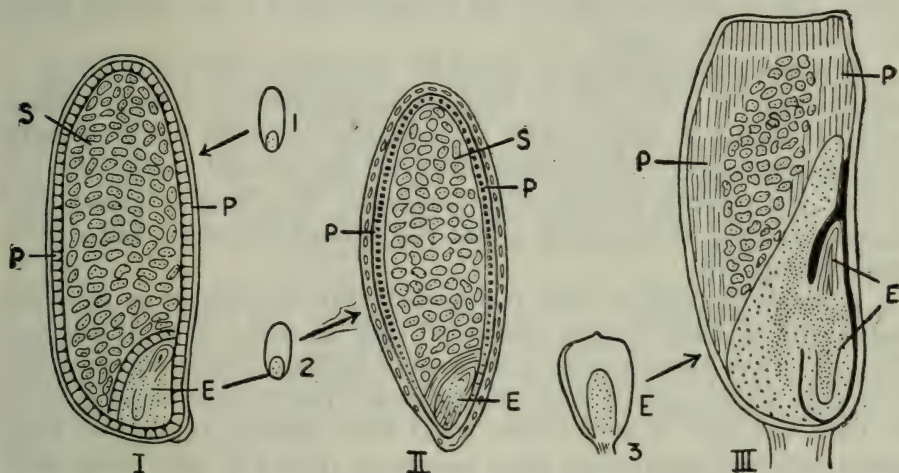


FIG. 54.—SECTIONS THROUGH SEEDS OF RICE (I), WHEAT (II), AND CORN (III), SHOWING THE PROTEIN (P) AND STARCH (S) OF THE SEEDS AND THEIR GERM (E); 1, 2 AND 3, THE SEEDS AS SEEN FROM THE OUTSIDE, NATURAL SIZE.

to yellow, through the browns and reds, to almost black. (2) The middle layer, called the subpericarpal or aleurone layer, which is composed of cubical cells filled with aleurone and fat; it contains very few starch grains. Practically all of the fat, and the greater part of the protein of the grain is confined to this middle layer; the pericarp and subpericarp contain practically all the phosphorus, and also all the vitamins, and organic nitrogenous bases. (3) The bulk of the grain, which consists of the innermost portion is filled with starch granules. The embryo is absent in milled rice.

Rice is first husked between large horizontal revolving stone disks. The chaff is winnowed out. This part of the process chips off only a small part of the pericarp. The grain is then milled by means of a vertical, revolving conical stone, around which is a close-fitting metal gauze case. Friction between the stone and the case rubs off the pericarp and the aleurone layer.

³³ *Brit. M. J.*, 1924, 1: 414.

The dust escaping is called rice polishing or rice bran, which is used in the prevention and treatment of beriberi. In the so-called "highly milled grades" of rice, such as are commonly seen in the markets of the world, all of the pericarp and most of the aleurone layer have been removed. The last process in the treatment of rice is the polishing with buffers covered with long-wooled sheepskin. This removes the dust, and leaves a clean, white grain. Talcum and glucose are often added to give a shiny surface. By undermilled rice is understood rice retaining a large share of the pericarp and aleurone layers.

Prevention.—The prevention of beriberi consists in substituting the use of whole rice for the polished grain; also in improving the general quality of the food and in providing for better balanced dietaries, especially adding articles containing vitamins, such as fresh meat, vegetables, milk, eggs and fruit. The prophylactic value of rice polishings added to the ordinary native diet must be borne in mind.

It seems a simple thing to substitute undermilled for highly milled or polished rice, but it will require a long and patient campaign to make a change which is utterly at variance with the economic and social habits of entire nations who have for many years considered the polished rice as the best quality, and generally purchase it by preference. Much may be accomplished through taxation of the highly milled rice, through education, and also, in part, through law.

It should be borne in mind that beriberi may be produced by an unbalanced diet of other starchy substances, such as wheat flour (Little and Strong). Cases have occurred on the coast of Labrador from a restricted diet, consisting largely of wheat flour. Wellmann and Bass ³⁴ produced polyneuritis of fowls with sago in twenty days; boiled white potatoes, twenty-four days; boiled mill rice, twenty-seven days; cornstarch, thirty-two days; white flour, thirty-four days; corn grits, thirty-six days; boiled sweet potatoes, thirty-eight days; cream of wheat, thirty-nine days; puffed rice, thirty-nine days; macaroni, forty days. There is no danger of contracting beriberi with the varied diet customary in this country and Europe.

There are certain accessory factors believed to favor beriberi: The disease occurs especially in overcrowded places, such as ships, jails, and asylums; during the hot and moist seasons; and following exposure to wet. These are to be avoided. Europeans living under good hygienic conditions, and enjoying a well-balanced diet, do not contract the disease.

SCURVY (*Scorbutus*)

Scurvy is a deficiency disease due to the lack of a specific food factor, vitamin C. It is caused by a prolonged defect in diet, particularly the lack of fresh food in the ration. It is characterized by debility, anemia, a spongy con-

³⁴ *Am. J. Trop. Dis. & Prev. Med.*, 1913, 1: 129.

dition of the gums and a tendency to hemorrhages. The lesions occur especially in the bones and the blood-vessels.

Scurvy occurs independently of age, sex and general vitality, if the diet is deficient. It may develop in vigorous adults and even in the aged, but it is especially prevalent in infants because of the monotonous one-sided diet of cow's milk or some proprietary food lacking in the specific vitamin. The incidence among infants varies in different localities. It is now less common than formerly since the cause and prevention are understood.

Scurvy is a nutritional disease caused by a diet deficient in fresh fruit and fresh vegetables. Dried fruits have practically no protective power. Dried vegetables likewise have lost their antiscorbutic value. Much of the adult scurvy that occurred among the civil population of Europe during the World War was due to a mistaken reliance on dried vegetables. The potato is the mainstay against scurvy during the winter months.

History and Prevalence.—As early as 1776, Captain Cook discovered that, "in order to preserve the health of his crew on long voyages, it was necessary for them to take every opportunity of obtaining fresh food." We first hear of scurvy in the literature as a plague infesting armies and besieged towns; later, as a disease which decimated sailors of the navy and of the mercantile marine. The disease has long been a scourge of sailors and soldiers. Fifty-five of the 143 epidemics analyzed by Hirsch were during hostilities. It affected prisoners and troops in the World War, especially on the eastern front. In Mesopotamia the British troops suffered owing to lack of fresh food. The extent to which scurvy prevailed during the World War is summed up by Hess.³⁵ It also occurs in the civilian population, sometime sporadically, often in endemic form.

Scurvy is endemic in northern Russia and in some parts of the tropics. It occurs in Norway and Ireland when the potato crop fails, and in other places when the supply of fresh food is deficient. In the United States scurvy is a rare disease except in infants. Frank cases in adults are uncommon and it seldom appears in the mortality statistics.

Experimental Scurvy.—*Etiology.*—Typical scurvy can be produced and cured at will by so regulating the diet that it is either deficient or well supplied with antiscorbutic vitamin. Whether this accessory food factor is utilized by the body as a food, or whether it acts indirectly is still unknown. Furthermore, it has not been definitely established whether the lesions and symptoms are due entirely to the lack of vitamin or whether the deficiency leads to secondary toxic states. The scorbutic condition, once established, predisposes to infections.

Theobald Smith³⁶ as early as 1895-96 noted that "when guinea-pigs are fed with cereals (it has been observed for some years in this laboratory), with bran and oats mixed, without any grass, clover or succulent vegetables, such as

³⁵ *Internat. J. Pub. Health*, 1920, 1: 302; also *Scurvy, Past and Present*, J. B. Lip-pincott Co., 1920.

³⁶ *Bur. An. Ind., Ann. Rept.*, U. S. Dept. Agric., 1895-6, 172.

cabbage, a peculiar disease, chiefly recognizable by subcutaneous extravasation of blood, carries them off in from four to eight weeks."

The classic experiments of Holst and Froelich³⁷ in 1912 demonstrated the symptoms and lesions of scurvy in guinea-pigs by means of a diet of cereal grains. These observations have been confirmed by Baumann,³⁸ Jackson, and many others. It is easy to produce or prevent scurvy in guinea-pigs, and to cure it when not too far advanced, simply by the use of antiscorbutic food.

While it seems clear that scurvy is due to the deficiency in the diet of a specific food factor of the vitamin type, other explanations of the disease deserve mention. A favorite theory is that the faulty diet undermines health and thus permits infection. Many outbreaks of a disease resembling scurvy during the World War seemed to have an infectious nature. The diagnosis in such cases is not clear, for every weakening disease with a tendency to hemorrhages is not scurvy. Furthermore, we must remember that scurvy lowers resistance and favors infection. The theory that scurvy is an auto-intoxication will not die. Thus, McCollum and Pitz³⁹ found in the guinea-pigs which had died of scurvy, that the cecum, which is a very large and very delicate pouch through which the food must pass in going from the small to the large intestine, was always packed with putrefying feces. This was soon disproved by Hart, Hess, Mendel, Steenbock, Chick, Harden and others who again substantiated the earlier view that the disease is the result of a deficiency of some nutritive factor in the diet.

It takes about six months for symptoms to appear in man on a diet deprived of vitamin C. The time depends upon numerous secondary factors, such as individual susceptibility and the amount of vitamin which has been previously stored in the body. During the prodromal period there may be impaired vitality or even lowered resistance.

Antiscorbutic vitamin C has not been isolated in a state of chemical purity, yet it can be measured quantitatively by graded tests on animals. It is soluble in water and in alcohol; it loses its potency on alkalinization, heating, drying or aging. Hess has shown that heat in itself causes little destruction of this vitamin, unless accompanied by oxidation. The rate of destruction of vitamin C depends upon a variety of conditions, especially the duration of heating, oxidation and reaction of the medium. Gradual oxidation and destruction of the antiscorbutic vitamin is the reason that foods lose their antiscorbutic vitamin on aging, that stale milk is not as valuable in this respect as fresh milk, and that vegetables fresh from the farm are superior to those which have been stored for long periods.

Antiscorbutic vitamin has the attribute of all known vitamins in that it cannot be synthesized by animals, which must depend upon the vegetable world for their supply. This principle is of practical significance in the prevention of scurvy. It means that cow's milk may be rich or poor in vitamin,

³⁷ *Ztschr. f. Hyg. u. Infektionskrankh.*, 1912, 72.

³⁸ *Am. J. M. Sc.*, 1917, 153: 650.

³⁹ *J. Biol. Chem.*, 1917, 32: 229.

according to the nature of the fodder; and that the quality of the milk which the baby obtains at the breast depends upon the food of its mother.

Vitamin C is found in fruits, vegetables, green grass and other forage. Cooked foods, with certain exceptions, such as tomatoes, have little antiscorbutic value, and dried foods none (see Prevention, page 628).

Infantile scurvy (Barlow's disease) occurs in babies, mostly between the sixth and tenth month, and particularly in those not breast fed. Every infant raised on sterilized or proprietary foods for several months, without any fresh or "natural" food, is menaced with scurvy. The disease rarely occurs in breast-fed babies. After the second year it is exceptional, as by this time the diet is enlarged. Latent and subacute cases that do not reach the threshold of clinical symptoms occur frequently and undermine health. Scurvy occurs in the babies of the rich, owing to the strict diet of heated milk and nothing else. All babies should have orange juice, tomato juice, or some other antiscorbutic. It is customary to start breast-fed babies with an antiscorbutic at the third month.

The early symptoms of infantile scurvy are failure to gain weight, increasing pallor and irritability. When the disease is well developed, it is characterized by subperiosteal hemorrhages, painful swellings and ecchymoses about the joints, especially the ankles and knees; hyperesthesia; pseudoparalysis of the lower extremities, due to the pain and hemorrhages; spongy, bleeding gums; hemorrhages from the nose, and occasionally other mucous membranes. The slightest bruise causes hemorrhage into the injured part. In advanced cases, there is also general weakness and marked anemia. Infantile scurvy is often mistaken for rheumatism, infantile paralysis, etc.

When we reflect that it usually requires about six months before a case of scurvy reaches the threshold where it can be recognized clinically, it is clear that during this phase there must be a certain undermining of health. This same condition doubtless occurs in the preclinical period of other deficiency diseases.

The prompt reaction of a patient to antiscorbutic treatment is magical. A cessation of pain often takes place in one or two days, and within a week all the symptoms may have disappeared.

Scurvy of *adults* is similar to that of infants. The main difference is in the subjective symptoms in the adult. Pains occur in various parts of the body, the gums are frequently the site of infection and there is ulceration as well as hemorrhage.

Complications.—The common complications of scurvy usually result either from hemorrhages or from infection. The hemorrhage may be in the orbit, causing proptosis, or in the brain or abdominal cavity. Edema is not uncommon. Infants suffering from scurvy even in mild degree are peculiarly susceptible to infections. The respiratory tract is particularly vulnerable. Pneumonia is the most common cause of death. Scurvy also predisposes to and is frequently associated with frequent attacks of "grip," extensive nasal diph-

theria, furunculosis, torpid ulcers of the skin, pyelitis, otitis, adenitis, gingival pyorrhea, gastro-intestinal disturbances, etc.

Relation of Milk to Scurvy.—Milk, at best, is not rich in antiscorbutic vitamin. Its original potency depends upon the vitamin content in the diet of the mother or in the fodder of the cow. Scurvy is very rare among breast-fed infants because the mother's diet is seldom completely deficient in this particular vitamin. It is otherwise with infants fed on cow's milk. The amount of antiscorbutic vitamin is variable in cow's milk and may even be absent in stall-fed cows in winter, receiving only dried fodder and silage.

The potency of the antiscorbutic vitamin in milk decreases with age, drying, heating and oxidation. Heated milk may induce scurvy in infants, owing to the duration of the heating process and the exposure to oxidation rather than the degree of heat. The heat of ordinary pasteurization (142-145° F. for 30 minutes) decreases the antiscorbutic property of milk about one-half. Homogenization of milk destroys this specific food factor. A very small percentage of hydrogen peroxid will destroy vitamin C in milk. This and other oxidizing preservatives therefore should not be used.

Prevention.—The prevention of scurvy is plain and may be summed up in two words: *fresh food*, especially fresh fruits and fresh vegetables. To prevent scurvy the diet must include an adequate amount of antiscorbutic food from day to day. The amount of this specific dietary principle varies greatly in different foods and is absent in many.

The best antiscorbutics are the citrous fruits and fresh raw apples and bananas; among the vegetables, tomatoes, cabbages, turnips and potatoes, and also onions, spinach and lettuce. Drying, heating and oxidation cause a deterioration of the antiscorbutic principle in some of these foods. Dried vegetables are not protective; canned tomatoes, processed at a high temperature, retain their prophylactic properties; orange juice may be boiled without injury. Raw cow's milk from pasture-fed cattle contains antiscorbutic vitamin. This property is diminished by about one-half through age or pasteurization.

Every baby which is not being nursed must receive a daily quota of antiscorbutic food in addition to cow's milk, whether pasteurized or raw. This should be begun at one month of age. At first, one teaspoonful of orange juice is given, diluted with water and sugar if it is tart, and this is gradually increased until one tablespoonful is fed at three months of age. Tomato juice is a good substitute, but the amount should be doubled. Canned tomato, strained, serves the purpose as well as raw tomato juice and does not need cooking. No reliance should be placed on dried prunes or on beef juice or on small quantities of vegetables weak in antiscorbutic power, such as carrots or peas, especially if they are old and have been cooked for long periods.

Meat (that is, muscle) lacks antiscorbutic power.⁴⁰ On the other hand, the liver and other glandular organs, as the lungs, spleen, pancreas and brain,

⁴⁰ E. B. Vedder, *Mil. Surg.*, 1922, 50: 534.

contain vitamin C. This explains why scurvy is not particularly prevalent among the peoples of the Arctic, who live for the most part on animal food and who rarely have fresh fruit and vegetables. Carnivorous animals escape because they are especially fond of the liver of their quarry. Vedder states that when the Indians of the plains had been without game over a considerable period, they were accustomed to open the freshly killed bison and eat handfuls of raw liver.

In contradistinction to rickets, ultraviolet irradiation neither prevents nor cures scurvy; in fact, it aggravates the disease.

The same measures prevent and cure scurvy. Antiscorbutic food is not only protective but is a specific remedy and, when taken early, produces complete and permanent cure with no sequelæ. Education in good dietary habits is one of the public health requirements in prevention.

Our knowledge of scurvy emphasizes the importance of fresh food as well as variety in the diet. The antiscorbutic property becomes impaired with age, is lost on drying and largely destroyed by cooking. There is also a serious loss when food is kept hot for a long time between cooking and serving. This vitamin is better preserved in cold storage than at ordinary temperatures.

The prevention of scurvy is a public health problem everywhere with regard to infants, but is not a problem of adults living in prosperous countries in peace times in the temperate zones. Adults living on a well-selected diet of ordinary staple foods require no special antiscorbutic measures. Under these conditions, there is no need to purchase vitamins in any other form.

REFERENCES

- American Pediatric Society. "Collective Investigation on Infantile Scurvy in North America," *Arch. Pediat.*, 1898, 15: 481.
- BARLOW, R. "On Cases Described as Acute Rickets," *Med. Chir. Trans.*, 1883, 66: 159.
- British Medical Research Committee. "Report on the Present State of Knowledge Concerning Accessory Food Factors (Vitamins)," Special Rep. Ser., No. 38.
- HESS, A. F. *Scurvy, Past and Present*. J. B. Lippincott, 1920.
- HOLST, H., and FROELICH, T. "Ueber experimentallen Skorbut," *Ztschr. f. Hyg. u. Infektionskrankh.*, 1919, 72: 1.

RICKETS

(*Rachitis*)

Rickets is a nutritional disorder due to lack of sunshine and faulty diet. It is a disease of civilization brought on by artificial living. The disease is characterized by an alteration in the structure and growth of bones, which become enlarged at the extremities and so soft that they bend under the weight of the body. It occurs during the first two years of life, when growth is rapid. The symptoms develop gradually, and when well advanced the disease is characterized by restlessness and night sweating, delayed dentition, softening of the skull bones and flabby muscles. Rickets is responsible for

bowed legs, knock knees, flat feet and saber legs; and is associated with the rickety rosary, pigeon breast, square head and pot belly, a strange medley that find their most exaggerated expression in misshapen dwarfs.

Rickets constitutes one of the important diseases of infancy on account of its prevalence, its serious complications and sequelæ, and the fact that it is readily preventable. Deformities of the bones of the pelvis produced by rickets in childhood is one of the causes of difficult labor, and may result in fetal or maternal death.

Rickets first manifested itself in Europe in the sixteenth century. The reason is obvious. Populations, particularly in England—a country never noted for its abundance of sunlight—were attracted to city life with its impoverished diet and bad housing. Rickets became so prevalent by 1650 as to call forth Glisson's famous monograph on the subject.⁴¹ It was not until 1885 that Pommer⁴² established its pathology.

Rickets is one of the penalties of living in houses on a denatured diet. Primitive man lives in the sunshine and on natural food. Tropical races are spared; the Eskimos, deprived of the sun's rays for long periods, are protected by a diet rich in fish oils and liver.

Prevalence and Geographic Distribution.—Rickets occurs mostly in cities. It began in the cities of Europe and later appeared in North America and then in India. In India it is found among the upper classes due to "purdah," or the custom of keeping women with their children permanently in darkened houses; the lower classes, even with a restricted diet, are spared because they live so much in the sun. It does not occur in the native parts of Africa, and is rare in China and Japan, where, regardless of the density of population, much of the life is out-of-doors. Savages may starve and may become victims of pestilence, but do not develop rickets. It occurs among all domestic animals except the cat, which refuses to be entirely housed. Schmorl⁴³ found histological evidence of rickets in 90 per cent of infants between the ages of four and six months in a large series of routine autopsies.

Hess⁴⁴ finds rickets more prevalent among well-nourished than among poorly nourished infants, which suggests the importance of the growth factor as an exciting or concomitant cause of the development of the condition. Atrophic or marantic infants are notably free of the disease.

The poor suffer most, although the children of the well-to-do are not spared. Rickets is much more prevalent than is indicated by the meager figures found in vital statistics. For various reasons it is more often overlooked than recognized. It does not appear in the mortality tables because it is seldom a direct cause of death.

Rickets is the most common chronic nutritional disorder occurring among infants of the temperate zones; it is mild or absent in the tropics. It is

⁴¹ *De Rachitide*, London, 1650.

⁴² *Untersuchungen über Osteomalacia und Rachitis*, Leipzig, 1885.

⁴³ *Ergebn. d. inn. Med. u. Kinderh.*, 1903, 4: 403.

⁴⁴ *Abvs Pediatrics*, Vol. II, 907.

more particularly a disorder of cities and of the large industrial centers, although it is by no means absent among the rural population. Its frequency is roughly proportional to the density of population.

Seasonal Prevalence.—Rickets begins to increase in the fall, becomes more marked in the winter, and reaches its peak in March; it then declines steadily, and new cases rarely develop after June. Hess and Lundagen⁴⁵ came to the conclusion that 50 per cent of breast-fed infants and probably most of those fed on the bottle are victims of rickets by the end of March. Refinements in diagnosis and a better understanding of the disease in recent years has demonstrated that in winter and early spring rickets is to be expected in about 90 per cent of our infants who are artificially fed, provided they are kept indoors. The seasonal variation offers striking evidence in favor of the part that sunlight plays in rickets.⁴⁶

Age and Race Incidence.—Rickets is most frequent during the second half of the first year of life or the first half of the second year. It may occur earlier, but is not congenital. Premature infants are notably predisposed to rickets, probably on account of their exceptionally rapid rate of growth. To a less extent this is true of twins. There is a late form called "rachitis tarda" which appears about the time of puberty, but which is very rare except under extreme privation such as occurred in the Central Empires during the World War.

In the temperate zones rickets is more frequent among colored and dark-skinned races than white on account of the pigment armor against the effect of sunshine. As is well known, Italian and Negro babies in our large cities are especially prone to develop this disorder.

The Disease.—The earliest symptoms are restlessness, irritability and head sweating—signs which are suggestive rather than indicative. Soon thereafter, enlargement of the costochondral junctions ("the rachitic rosary") develops. This is the most reliable early sign of rickets. The head becomes somewhat square in shape, the fontanel is too widely open, and areas of softness may be felt along the lower part of the occipitoparietal suture. The epiphyses at the wrist may be enlarged and radiographs disclose changes in the ulna typical of rickets. A test of the blood will show a deficiency of inorganic phosphates. All these signs are not present in every case.

The well-developed or advanced case presents a picture which is evident at a glance. The head is square, the thorax deformed, showing two lateral rows of visibly enlarged costochondral junctions and a groove traversing its lower part (Harrison's groove). The abdomen is large and protuberant—the well-known "pot belly"; constipation rather than diarrhea is the rule. The legs are bowed or knock-kneed and the ligaments lax. The infant may no longer be well-nourished, for the extreme cases are associated with retardation in growth and weight.

⁴⁵ *J. Am. M. Ass.*, 1922, 79: 2210.

⁴⁶ *J. Am. M. Ass.*, 1922, 79: 2210; *Proc. Soc. Exper. Biol. & Med.*, 1922, 19: 380.

The majority of cases are very mild, so mild indeed that many pass unrecognized and leave no local deformity or systemic disturbance. Recovery comes about spontaneously as the result of the fortuitous advent of spring or summer, or the decrease in rate of growth and the enlargement of the diet. Bowed legs, beading of the ribs and enlarged epiphyses gradually disappear and the bones regain their normal contour in most cases. However, these signs may persist, as well as the square head, the deformed chest, the scoliosis, kyphosis, flat-foot, etc. The pelvic deformity is the most important, as it may lead to varying degrees of dystocia, rendering cesarean section necessary.

Osteoporosis is a condition that has doubtless frequently been confused with rickets since very early craniotabes is unquestionably often osteoporotic rather than rachitic in nature.

It is an observation of long standing that rachitic children are predisposed to infections of the respiratory tract; in fact, this is one of the main dangers of the disorder. Bronchitis and pneumonia are frequent complications. Italian infants in the large cities are especially susceptible to both rickets and the respiratory diseases.

Tetany is frequently associated with rickets and has a similar seasonal incidence. Park suggests that tetany should be regarded as a form of rickets involving the calcium rather than the phosphorus metabolism. When rickets is complicated with tetany, treatment should always be supplemented by calcium chlorid or hydrochloric acid for the immediate relief of symptoms. Like rickets, the prevention and cure of tetany depends upon sunshine, ultra-violet irradiation or cod-liver oil.

Etiology.—The etiology of rickets involves a number of factors, the chief of which is lack of sunshine, or absence of specific food in the diet, or both; also lack of balance between calcium and phosphorus. But even all this does not lead to the disease unless it occurs at the age of rapid growth.

Until recently there was a sharp difference of opinion as to whether rickets came about as the result of improper diet or faulty hygiene. To-day we know that both factors are involved. In regard to the diet there is general agreement that bottle-fed babies develop rickets with far greater frequency than those which are breast fed. The purity of the cow's milk plays no rôle in the etiology; rickets will develop even when the best milk is fed. Many have associated rickets with a diet poor in fat, especially with condensed milk or with the various proprietary cereal foods. According to Hess there is little connection between the two. It is probable that the rickets-producing effect of carbohydrate is largely due to the fact that it leads to rapid growth. Hess found that about 75 per cent of the cases developed among the well-nourished and but 25 per cent among the poorly nourished infants. This is quite contrary to what occurs in infantile scurvy.

Many other theories of causation have their advocates: overeating (Glisson), acidosis (Heitzmann), congenital syphilis (Klose, Vogt and Prichard), lack of fresh air and exercise (Findley and Galbraith), and lack of vitamin A (Mellanby). It is also claimed that rickets is inherited, that it is the

result of disturbance of the endocrine glands, or that it is the result of infection. None of these hypotheses has been established. It is probable that there is a prenatal factor, in that the diet and regimen of the mother plays an etiologic rôle.

Relationship has been suggested between rickets and syphilis, tuberculosis, malaria and epilepsy, but has not been established clinically or experimentally. Neither is there any relationship between rickets and scurvy. The conception of "scurvy rickets" may be compared to the equally fallacious conception of "typhoid malaria."

The Hygienic Factor.—The greatest recent advance in our knowledge of rickets has been an understanding of the precise nature of the hygienic factor. This has been shown to be *sunlight* rather than fresh air, exercise, cleanliness, etc. Infants do not develop rickets in the summer on account of the protective effect of the sun's rays. This is due to the ultraviolet radiations and not to the visible rays. These radiations have very slight power of penetration, and are filtered out by ordinary window glass, so that a baby will receive little, if any of their protective properties if it is kept in a room with the windows closed, notwithstanding the fact that it is in a flood of light.

The Dietary Factor.—Dietary factors may assume a determining rôle, but it is equally evident that rickets will develop when there is lack of sunshine, even if the food supply is satisfactory. No single dietary fault can be found that is common to all. The defects appear to be of various kinds and of such nature as rather to predispose to the development of rickets than actually to cause it.

The antirachitic factor is present in large amounts in the oil of the cod and in considerable amounts in the oils of the shark and burbot. It is present in small amount in butter, which cannot be considered as protective. Vegetable oils do not possess this property, except cocoanut oil. It has also been found in alfalfa and clover blossoms. It therefore seems that the antirachitic factor occurs naturally in the vegetable world and is stored in certain animal oils and fats. Egg yolk is protective and curative although less so than cod-liver oil.

Almost from the moment that vitamins were discovered, it was assumed that rickets belonged to the class of vitamin deficiency diseases. Mellanby suggested fat-soluble vitamin A or one closely associated with this factor. When this was shown to be untenable, an unknown vitamin (X) was accused by Park but acquitted. The chemical nature of the defect in the diet which predisposes to rickets is as yet unknown. It seems probable that certain foods contain stored radiant energy which may take the place of sunshine. Hess and Weinstock⁴⁷ rendered cottonseed oil and linseed oil antirachitic by irradiating them with ultraviolet ray. This perhaps explains the common meeting ground for two such apparently dissimilar agencies as radiant energy and cod-liver oil.

⁴⁷ *J. Am. M. Ass.*, 1924, 83: 1845.

Phosphorus and Calcium.—Recent investigations seem to point to a defect of absorption in the intestinal tract, due to a faulty chemical reaction and an unfavorable interrelationship between the salts of calcium and phosphorus.

Schabad showed some years ago that cod-liver oil, when given in rickets, is able to change a negative to a positive calcium balance. Sunlight or artificial ultraviolet radiation will bring about a similar result and raise the inorganic phosphate of the blood to normal.

It has been found recently that there is a characteristic chemical alteration of the blood in rickets. The percentage of inorganic phosphate is diminished. The normal content of the blood in infants is about 4.0 to 4.5 milligrams per 100 cubic centimeters of plasma. In active rickets the percentage falls to 3.0 or 3.5 per cent or even lower in the severe forms. This observation is in harmony with that of Hess and Lundagen that there is a seasonal tide of inorganic phosphate in the blood of infants; an ebb in the winter and early spring and a flood in the summer, a wave which runs parallel with the seasonal curve of the incidence of rickets. The calcium is normal or but slightly diminished.

McCollum and coworkers⁴⁸ have demonstrated that diets producing rickets in rats must have a disturbed calcium-phosphorus ration; that is, they must be deficient either in calcium or in phosphorus and that the antirachitic factor and sunlight must be lacking. Shipley, Park, McCollum and Simmonds⁴⁹ suggest the probability of two main kinds of rickets: a low phosphorus rickets with approximately normal calcium and a low calcium with approximately normal phosphorus. Tetany appears to be an expression on the part of the nervous tissue of an insufficiency of the calcium ion. Rickets is an expression on the part of the skeletal tissues of a disturbed relationship between the calcium and phosphorus ions of the body fluids.

Prevention.—The prevention of rickets depends upon sunshine and diet. The key to the practical prevention is found in the fact that artificially fed, city babies furnish the vast majority of cases. Rickets is a disease of civilization. It is a warning against departures from the inexorable laws of nature. The story of rickets helps emphasize the importance of hygiene and a well-balanced diet.

Rickets is a public health problem of considerable magnitude. It deserves efforts at community control. The prevention of the disease should be made a special point of attack in the crowded portions of cities through baby hygiene work and education. The facts that the disease is common and serious and can be prevented by sunshine and by cod-liver oil should be broadcast until they become common knowledge. Gastro-intestinal disorders or respiratory infections in babies can in part be avoided by the prevention of rickets. Rickets itself is not a direct cause of death. It is one of the underlying causes of excessive infant morbidity and mortality.

⁴⁸ *J. Biol. Chem.*, 1922, 54: 249.

⁴⁹ *Am. J. Dis., Child.*, 1922, 23: 91.

Rickets is unique in the fact that it is the only disorder for which we possess two specific therapeutic agents: cod-liver oil and ultraviolet irradiation. Sunlight and its substitutes and cod-liver oil and its substitutes will prevent rickets and cure the disease, but an ounce of prevention is worth a pound of cure.

Sunlight; Irradiation.—*Hygiene* plays an important rôle in prophylaxis; this refers especially to sunlight, and to a far less extent, to fresh air, exercise and cleanliness. Infants should be out of doors as much as possible and not shielded from the rays of the sun; their arms and legs may be exposed while their bodies are protected against the cold.

A substitute for sunshine is ultraviolet radiations, as furnished by the mercury vapor lamp (air cooled), carbon arc lamp, etc. This agent is of great value in the winter months and may be given in addition to cod-liver oil or as a substitute in cases where the oil is not well tolerated. The mercury vapor lamp is placed at a distance of three feet from the baby and irradiations given every other day in increasing doses, from three to twenty minutes. The skin should become tanned but not blistered; different parts of the baby are selected for successive treatments. The carbon arc lamp brings about equally good results. Ultraviolet therapy is likewise of specific value in the treatment of tetany.

Hess⁵⁰ finds that the effectiveness of radiation depends on three factors: diet, the rate of growth, and the intensity of the pigmentation of the skin. Black rats are much more difficult to protect than white rats and the same holds true with colored and white infants. Hess and Weinstock⁵¹ found that the specific rays are in the ultraviolet zone of the solar spectrum and are 300 millimicrons in length or shorter. Pyrex glass is markedly permeable to the short ultraviolet rays and does not interfere greatly with the protective action of the rays. Wearing apparel filters the rays and prevents protection according to its thickness and color. Black clothing cuts off the ultraviolet rays entirely.

The high incidence of rickets in children during the late winter months is due at least in part to their mothers not receiving enough ultraviolet rays either during pregnancy or while in lactation. Cows prevented from receiving ultraviolet rays are not able to secrete this antirachitic substance in sufficient quantity to cure or allay the progress of clinical rickets.

The mercury vapor quartz lamp has been found one of the most satisfactory substitutes for the direct rays of the sun. The white flame carbon arc lamp was found by Hess and Unger⁵² to more nearly approach the sun's spectrum. Roentgen rays have no curative or protective effect on rickets.

Protective Diet.—In view of the prevalence of rickets, it seems advisable to give every baby cod-liver oil as a routine prophylactic, on the same principle that we give orange juice to prevent scurvy. Cod-liver oil is an excellent

⁵⁰ *J. Am. M. Ass.*, 1922, 79: 1177; 1921, 77: 39.

⁵¹ *Lancet*, 1922, 2: 367; *J. Am. M. Ass.*, 1923, 80: 687.

⁵² *J. Am. M. Ass.*, 1922, 78: 1596.

specific and should be given in small doses from October to May, beginning with 5 drops of the pure refined oil three times a day at the age of one month, or 15 drops mixed with the day's total feeding. The amount should be increased until the baby receives one and one-half teaspoonfuls daily at three months. Few babies do not tolerate cod-liver oil. Standard emulsions may be used but the absolute amount of oil must not be thereby decreased. Phosphorus may be given but should not be considered a substitute for the oil.

One of the best prophylactic measures is maternal nursing or woman's milk. This is, however, not always a safeguard even if the mother's diet has been adequate during pregnancy and lactation. A food which has proved of protective value is yolk of egg. Infants one or two months of age are given one-half of a yolk of raw egg mixed with the daily formula. At three months an entire yolk is given. Cow's milk contains antirachitic property, and this is not destroyed by drying, but neither woman's milk nor cow's milk can be depended upon to protect growing infants against rickets.

The modern teaching of the prophylaxis of rickets is to give every baby and growing child, as well as all adults, a sufficiency of both sunshine and antirachitic substances in the diet.

Rickets is the price we pay for abandonment of a life out-of-doors and a natural diet for a life in houses and a diet of denatured foodstuffs. We have not yet learned how to live in the city under artificial conditions of modern civilization.

COLLATERAL READING

- MELLANBY, E. *Medical Research Committee Report*, No. 38, London, 1919.
 PARK, E. A. *Phys. Rev.*, 1923, 3: 106.
 FINDLAY, L. *Lancet*, 1922, 1: 825.
 HESS, A. F. *Lancet*, 1922, 2: 367.
 MARFAN, P. *Maladies des Os*, Paris, Baillière et Fils, 1912.
 SCHMORL, G. *Ergebn., d. inn. Med. u. Kinderh.*, 1903, 4: 403.
 WIELAND, E. *Ergebn., d. inn. Med. u. Kinderh.*, 1910, 6: 64.
 HESS, A. F. *J. Am. M. Ass.*, 1922, 78: 1177.

PELLAGRA

Pellagra is a disease due to a defective diet, and characterized by digestive, nervous and cutaneous symptoms. Localized outbreaks resembling epidemics occur, but sporadic cases have a world-wide distribution. A marked feature is its seasonal prevalence in the springtime, following the restricted winter diet. Under unfavorable economic and dietary conditions in endemic localities pellagra recurs year after year, giving a false impression of chronicity. A disease in dogs known as black tongue seems to be a pellagrous affection.

The digestive symptoms are dyspeptic disturbances and later stomatitis. Nervous manifestations begin with weakness, vertigo and insomnia; then pains and neurasthenia. About 2 per cent of pellagrins develop mental disturbances requiring institutional care. The skin eruption is the most characteristic sign. It begins as an erythema, bilateral and symmetrical, affecting especially the

backs of the hands and forearms, the face and neck, feet and genitalia. The symptoms vary greatly in severity. Fully developed cases present a characteristic picture.

History.—The earliest observations of the disease date from 1735 and were made by Gaspar Casal in the neighborhood of Oveido, Spain, but were not published until 1762, three years after his death. Nine years later, but without knowledge of it, Francesco Frapolli reported the disease from Italy. It was reported by Hameau from France in 1829. In 1858, Theodori found it in Rumania and in 1874, Pruner-Bey mentions it as occurring in Egypt.⁵³

The first recorded observation of pellagra in the New World appears to be the report of a sporadic case by Gray from Utica, New York, in 1864. It has been suggested by Babcock that the disease probably occurred as far back as 1828 in the South Carolina State Hospital for Insane. Knowledge of it as endemic in the Western Hemisphere dates from about 1893, when it was described under the name of *psilosis pigmentosa* by Bowen from Barbados. Since then the disease has been reported from nearly all the countries of North, Central and South America. The recognition of its endemicity in the United States dates from 1907 or 1908 and the names of Searcey, Babcock, Watson, Wood and Lavinder are closely associated with this important episode in the history of its recognition.⁵⁴ The disease (sporadic or endemic) has now been reported from nearly all parts of the civilized world.

Occurrence.⁵⁵—In Europe the disease has long prevailed in Portugal, Spain, Italy and Rumania. Italy and Rumania have suffered severely, while the northern European countries have been very slightly if at all affected. The distribution of the disease in America is also uneven, prevailing especially in the southern part of the United States and some of the West Indies; while Canada, Mexico and South America have remained well-nigh free. The areas most affected are the northern part of Italy, Rumania, lower Egypt and in the United States south of the Ohio and Potomac Rivers.

Sporadic cases may crop out anywhere but usually the disease prevails in endemic areas, and at times outbreaks occur in the manner of an epidemic. It prevails under widely contrasting conditions of climate and soil, so that clearly these factors are but secondarily, if at all related to pellagra prevalence.

One of the most striking features about pellagra is that it appears during the spring and early summer, in this respect resembling the seasonal prevalence of endemic scurvy of temperate climates.

There is no special race susceptibility. In some localities the white and in others the colored pellagrins predominate.

The disease may develop at any age, but is rarely seen during the first year of life. It attacks both sexes about equally during childhood and adolescence, but many more cases occur in adult women than in men.

⁵³ Roussel, *Traité de la pellagra*, Paris, 1866; Rossi, "On the Etiology of Pellagra and Its Relation to Psychiatry," *Am. J. Insan.*, 1913, 69: 939.

⁵⁴ Marie, *Pellagra*, Columbia, S. C., 1910.

⁵⁵ Goldberger, "Pellagra," *Tice's Practice of Medicine*, 1920, Vol. IX, p. 205.

Economic conditions have a marked influence upon the incidence of the endemic disease. The earliest students of the disease commented on the association of pellagra with poverty and poor hygienic conditions. Pellagra, however, occurs both sporadically and endemically under excellent sanitary surroundings. The amount of the disease varies from year to year with fluctuations in economic conditions. There is a direct correlation between pellagra and family income and the availability of food supplies.

In Europe, the disease attacks almost exclusively the peasant farm laborer. In the endemic area in the United States, it has not so marked a rural distribution, nor is it restricted to the agricultural population. Indeed, with us, pellagra has prevailed extensively in certain types of industrial communities, such as cotton mill villages, mining and sawmill camps; also in orphanages and asylums for the insane.

Before the World War, in notable contrast to scurvy and beriberi, pellagra did not occur in the army, navy or merchant marine, even of those countries in which the disease was highly prevalent. During the World War, a notable outbreak occurred among Armenian refugees at Port Said and among Turkish and German prisoners of war in Egypt.

Prevalence and Fatality.—Goldberger estimates that in 1916, which was by no means a year of exceptionally high incidence, approximately 5,500 deaths from pellagra occurred in the southern states. Of these, 840 were in Mississippi, 729 in South Carolina, 677 in Alabama, and 607 in Tennessee. There has been a notable reduction in prevalence in more recent years as a result of prosperity and better knowledge of the importance of a balanced ration. The public health importance of the disease cannot be evaluated entirely from the death rate, for the case fatality rate is not high. Goldberger estimates that in 1916 upwards of 100,000 individuals in our southern states suffered an attack of the disease.

Etiology.—The cause of pellagra has long been a bone of contention, and even to-day there is a conflict of views with ardent supporters of theories. It was early suggested that the disease arose from bad food. This vague idea underwent various modifications, until the theory was evolved that pellagra is a poisoning resulting from spoiled corn (maize). This *zeistic theory*, which long held sway, was particularly developed by Lombroso.

The Insect-borne Theory.—Sambon, in 1905, and more elaborately in 1910, advanced the view that pellagra was an insect-borne infection conveyed by a species of *Simulium* or buffalo gnat. Studies by all the students of the disease in the United States disproved both the maize theory and the insect-borne theory.

The Excreta-borne Theory.—The Illinois Pellagra Commission⁵⁶ believed the disease was due to infection with possible location in the intestinal tract, and that deficient animal protein in the diet may constitute a predisposing factor. These views received support from the Thompson-MacFadden Pel-

⁵⁶ *Report of the Pellagra Commission of the State of Illinois*, Springfield, 1912.

lagra Commission⁵⁷ (Siler, Garrison and MacNeal), who soon concluded that pellagra is in all probability a specific infectious disease, communicable from person to person, and particularly in association with insanitary methods of excreta disposal. These conclusions were apparently supported by the observations of Smith, Pollitzer and Mustard⁵⁸ (1916) in South Carolina, who believed that "bad hygienic and overcrowded conditions play some part in the propagation of this disease." Jobling and his coworkers,⁵⁹ in Nashville, Tennessee, noted that an overwhelming majority of pellagrins developed the disease while residing in houses provided with crude, insanitary methods of excreta disposal.

Various writers have from time to time also argued in support of infection on the basis of one or more of the following epidemiological features of the disease: (1) its marked seasonal prevalence in the spring; (2) its peculiar geographic distribution in the United States; that is, its preponderating prevalence in the southern states; (3) its seemingly abrupt appearance with the subsequent apparently rapid extension and progressive increase in the United States; (4) its much higher incidence in adult women than in men; (5) its occasional occurrence in the seemingly well-nourished (including nursing infants) and, at times, in individuals economically well situated and thus presumed to have had a liberal diet available. None of these arguments, however, is inconsistent with the dietary origin of the disease.

Goldberger explains the occurrence of the sporadic case in well-to-do families as the result of eccentricities of diet, especially in women, and points out that well-to-do people may be food faddists, that they may be parsimonious, or that they may be unfavorably situated with respect to sources of household food supply, and thus that their diet may become one-sided, restricted and monotonous and possibly faulty in one or more respects.

Goldberger and fifteen of his associates⁶⁰ tried in a number of ways to transmit pellagra to themselves by series of inoculations with blood, with nasopharyngeal secretions and by feeding with dermal scales, urine and feces from cases of pellagra in various stages and of varying degrees of severity. The results of this heroic experiment were entirely negative.

The Faulty Diet Theory.—Just about the time (1914) when some of the most persuasive evidence in support of infection began to appear, Goldberger and his coworkers of the United States Public Health Service began studies of pellagra which have completely disproved the theory of infection and point unmistakably to faulty diet as the essential primary factor in the causation of the disease.

It was shown by Goldberger and Wheeler in 1915⁶¹ that the disease may be

⁵⁷ *Am. J. M. Sc.*, 1913, 146: 238; *Second Progress Report of the Thompson-McFadden Pellagra Commission*; *Third Report of the Robert M. Thompson Pellagra Commission*.

⁵⁸ *South. M. J.*, 1916, 9: 786.

⁵⁹ *J. Infect. Dis.*, 1916, 18: 501; 1917, 21: 109.

⁶⁰ *U. S. Pub. Health Rep.*, 1916, 31: 3159.

⁶¹ *U. S. Hyg. Lab. Bull.* No. 120, 1920.

experimentally produced in the human subject through a restricted diet. At a prison farm near Jackson, Mississippi, eleven convicts who volunteered for the experiment were carefully isolated and put on a diet consisting principally of wheat, maize and rice, with pork fat and some fresh vegetables (sweet potatoes, turnips, cabbage, greens). At least six of the volunteers developed evidence which experienced observers recognized as pellagra, while of forty-eight controls, none developed any recognizable indications of the disease.

Goldberger, Waring and Willets⁶² then demonstrated experimentally that pellagra may be completely prevented by diet without the intervention of any other factor, hygienic or sanitary. These workers conducted studies at two orphanages at Jackson, Mississippi, and at the Georgia State Sanitarium at Milledgeville, institutions which had been endemic foci of the disease for some years. Aside from a modification in the diet and increased watchfulness over the individual eating, all administrative routine and hygienic and sanitary conditions remained unchanged, and no restrictions were imposed on new admissions by reason of actual manifestations of pellagra or a history of an attack of the disease. At the end of the first year following the inauguration of the modified diet, it was found that at the orphanages, of an aggregate of 172 pellagrins under observation, only one had shown any evidence of a recurrence, and not a single case had developed among an aggregate of 168 non-pellagrins who had been continuously under observation; and at the sanitarium, of an aggregate of seventy-two pellagrins, not one presented recognizable evidence of a recurrence, although at the same time 4 per cent of a comparable group of thirty-two pellagrins not receiving the modified diet had recurrent attacks of the disease.

The Dietary Factor.—While it is clear that defective diet plays the dominant rôle in pellagra, the dietary factors concerned are not yet fully disclosed. Vitamins were at first accused. Sandwith⁶³ suggested the possibility of a tryptophan deficiency. McCollum, Simmonds and Parsons⁶⁴ first thought that pellagra was primarily associated with three dietary factors, namely vitamin A, mineral elements and protein mixture. More recently, McCollum and Simmonds⁶⁵ express the view that "it is abundantly established that pellagra is in some manner caused by faulty diet."

In 1918, Goldberger, Wheeler and Sydenstricker⁶⁶ reported that their studies suggested that the pellagra-producing dietary fault is the result of some one or more of the following factors: (1) a physiologically defective protein supply; (2) a low or inadequate supply of fat-soluble vitamin; (3) a low or inadequate supply of water-soluble vitamin; and (4) a defective mineral supply. In 1920, Goldberger and Wheeler studied the pellagra-producing diet and found it deficient in amino-acids, mineral elements, possibly but doubtfully

⁶² *U. S. Pub. Health Rep.*, 1915, 30: 3117.

⁶³ *Tr. Soc. Trop. Med. & Hyg.*, 1913, 6: 143; *Tr. Nat. Ass. for Study of Pellagra*, 1914, 97.

⁶⁴ *J. Biol. Chem.*, 1918, 33: 421.

⁶⁵ *The Newer Knowledge of Nutrition*, 3rd Ed., Macmillan, 1925.

⁶⁶ *J. Am. M. Ass.*, 1918, 71: 944.

a deficiency in fat-soluble vitamin, and, perhaps, some as yet unknown factor. In a later report,⁶⁷ Goldberger, Wheeler and Sydenstricker concluded that the pellagra-producing dietary fault is the result of some one or of a combination of the following factors: (1) a physiologically defective (amino-acid) supply; (2) a defective or inadequate mineral supply; (3) a deficiency in an as yet unknown dietary essential (vitamin?) none of the known vitamins being regarded as necessary factors.

Early in 1925, these workers reported feeding studies of the pellagra-preventive power of casein and of dried brewers' yeast.⁶⁸ Casein did not completely prevent though it appeared to modify the clinical picture of the disease, but the yeast in a daily dose of about 30 grams completely prevented its development.

These studies of Goldberger and his associates give us the evidence needed to establish a solid foundation for a dietary etiology. They show that none of the recognized dietary essentials, with the doubtful exception of the protein factor, are necessarily concerned as etiological factors in producing pellagra, thus proving the existence of a heretofore unrecognized or unappreciated dietary essential, which they have designated as factor or vitamin P.P., a deficiency of which is involved in the causation of disease. The contributions of Goldberger to pellagra are one of the notable achievements in medical science. He has produced and prevented the disease and even cured it by dietary changes alone. Finally, he has narrowed the food factor down to a new dietary essential, vitamin P.P.

Prevention.—The rational prophylaxis of pellagra is based on the theory that a faulty, deficient diet is the primary etiological factor. Theoretically, all that is necessary for the control and eradication of the disease is the substitution of an adequate diet for the faulty diet of the affected population. Practically, however, in attempting to accomplish this, one encounters difficulties of two general categories; first, that represented by the meagerness of our knowledge of the distribution in our foods of the unknown dietary factor. Until more is known about this dietary factor, advice as to diet must be of a broad, general character; that is, that the diet be made a normal, adequate one, as varied as possible. Since the available evidence indicates that fresh milk and fresh meat contain the pellagra-preventive factor or factors, special emphasis may be laid on these as pellagra-preventing foods.

We thus come to the second difficulty—the economic situation. It is not very helpful to urge people to improve their diet without at the same time giving serious thought to the correction of the economic factors involved. It is necessary in each instance to study carefully the type of community in order to determine the factors that may be there particularly operative. Encouragement of family cow ownership or improvement in marketing conditions, as, for instance, the establishment of an all-year-round meat market, may be the means to the desired end.

⁶⁷ *U. S. Pub. Health Rep.*, 1920, 35: 648. ⁶⁸ *U. S. Pub. Health Rep.*, 1925, 40: 54.

In attempting to deal with the disease in institutions or among those that are rationed, probably the first need is education of those in charge of the rationing. In institutions having but meager funds available, notably in hospitals for the insane, where economic pressure is accentuated by difficulties in individual feeding, it may be practicable to include dried yeast as a regular part of the daily diet. An allowance of 0.5 gram per patient per day would suffice, and thus go a long way and at small cost towards the complete prevention of pellagra.

Pellagra afflicts the people of the South mainly because their food supply is too restricted, and during the latter part of the winter consists too largely of milled cereals, tubers, molasses, syrup and fat pork. Experiments on animals have shown that this list of foods will not promote well-being over a prolonged period. On such a diet, ill health and inefficiency are to be expected, and experience abundantly shows that this result is being realized where such dietary practices prevail.

FOOD POISONING

By "food poisoning" is meant acute attacks of illness due to some injurious property in food. The term is not a good one, for many of these attacks are not poisoning at all, but infections; many are derangements of function due to a variety of causes. The term food poisoning, however, is much better than "ptomain poisoning," which is unscientific, misleading and incorrect. Mistakes in diagnosis are exceedingly common and can be avoided only by careful study of the case and circumstances.

General Considerations.—There are two well-recognized causes of food poisoning: (1) *food infection* and (2) *food toxemia*.

Food infection is caused by *Bacillus enteritidis* or closely allied organisms of the Gärtner group. Other bacilli are suspected, but their relationship has not been established. The first important landmark in clearing up the cause of food infection was the isolation by Gärtner, in 1888, of *B. enteritidis* from an outbreak at Frankenhausen. The trouble was caused by eating the meat of a cow which was killed on account of enteritis, and the specific bacillus was found both in the organs of the cow and the fatal case.

Food toxemia is due to toxins preformed in the food. Botulism is the only known example in this class. Other bacteria may produce toxic products in food, but none has been demonstrated to be poisonous by the mouth.

There is a great clinical difference between these two classes of food poisoning. Food infection is an acute disease characterized by nausea, vomiting, cramps and diarrhea, and fever. Botulism is characterized by nervous symptoms, paralysis, constipation and no fever. The symptoms vary greatly in severity. The case fatality rate in food infections is rarely over 2 per cent; botulism is fatal in from 50 to 100 per cent of those attacked.

The period of incubation is usually eight to twenty-eight hours, often longer. Frequently several meals intervene between the time the infected

food is eaten and the onset of the disease. Vomiting is often the first symptom and suspicion at once points to the food vomited, whereas it may have been due to something eaten at a prior meal. This mistake is common.

Various classifications of food poisoning have been attempted; none is satisfactory unless based on etiology. The commonest mistake is to classify food poisoning according to the food responsible, as meat poisoning, cheese poisoning, potato poisoning, milk poisoning, etc., etc. As a matter of fact, food infection is the same disease, whether the bacteria are conveyed in meat, milk, or mixtures. Botulism is the same whether it comes from sausage, brawn, pork, spinach, olives or beans.

Most instances of food poisoning are from food that is inadequately processed or imperfectly preserved. There is little danger in fresh food, whereas food that is prepared hours before it is eaten gives opportunity for the growth of bacteria. The chief offenders in food infection are meat, milk, and mixtures containing these products; the foods responsible for botulism are chiefly sausage, pork, and also a number of vegetables preserved in some way.

FOOD INFECTIONS

Food infection is almost always associated with Gärtner's bacillus (*B. enteritidis*), or a closely allied species, occasionally streptococci and other bacteria. This form of food infection is also called food poisoning, and commonly miscalled "ptomain poisoning." Since meat is the chief vehicle for this infection, it was formerly called "meat poisoning," but milk and milk products, as well as vegetables, may be the vehicles of these pathogenic bacilli.

Incubation Period.—Usually from six to twelve hours elapse between the ingestion of the food and the onset of symptoms. Occasionally the period of incubation is said to be four hours or less; it may be seventy-two hours or more. The incubation period varies in different outbreaks, and even in the same outbreak.

Symptoms.—The symptoms are essentially those of an acute gastro-intestinal irritation, namely, nausea, vomiting, and diarrhea. The onset is usually sudden. The attack may be ushered in with headache and a chill. The abdominal pain is frequently the first symptom and may be gripping and severe. The diarrhea usually consists of repeated bowel actions, which as a rule are offensive. Later in the attack, the stools become more watery and frequently of a green color. Faintness, muscular weakness and prostration may be quite marked. There is almost always a moderate rise of temperature, seldom above 102°-103° F. Various nervous manifestations, such as restlessness, muscular twitchings, and drowsiness, may occur, but these symptoms are not constant or marked. Patients are usually thirsty and secrete less urine. Herpes and other skin rashes have been noted.

The severity of the symptoms varies greatly in different outbreaks, and even in the same outbreak. All degrees are met with, from fulminating cases, fatal within twenty-four hours, to those of slight diarrhea and malaise, insuffi-

cient to keep the patient from work. Usually the attack is over in a day or two, with prompt recovery; although occasionally marked prostration may persist. The severity doubtless depends upon the virulence of the particular strain of bacilli concerned, the dose, and also with the susceptibility of the individual, which plays an important rôle in this as in other infections.

Occurrence.—The *infectivity* rate is generally high. Sometimes all those who eat the infected food are made ill; in some instances less than half suffer.

The case *fatality* rate varies greatly in different outbreaks. In the 112 British outbreaks studied by Savage, there were some 6,190 cases with ninety-four deaths, a case fatality rate of 1.5 per cent. In the United States deaths from this cause are rare.

The age and sex *distribution* depends entirely upon the accidental age and sex distribution of those who eat the infected food.

Most cases occur in the summer time, corresponding to the seasonal prevalence of typhoid fever, cholera, dysentery, infantile diarrheas (summer complaint), and other intestinal infections. The bacilli responsible for food infection grow in the food before it is eaten, and therefore temperature is an important factor. The greater multiplication of these bacteria in hot weather also increases the opportunities for infection and its transmission. Secondary infections rarely occur. There is the same potential possibility of contact transmission as in typhoid fever, but case-to-case infection is not observed. The Gärtner bacillus and its congeners do not readily adapt themselves to the intestinal tract of man, and therefore carriers are almost unknown (except in the case of paratyphoid bacilli).

The number of persons involved in outbreaks varies from one to several hundred. The larger outbreaks are almost all caused by infected milk. Thus, at Newcastle-on-Tyne, in October and November, 1913, 523 persons were affected by milk containing *B. enteritidis*, and a similar outbreak occurred in the same place in 1914, affecting 468 persons from the same cause. Usually the number of individuals affected is limited to one or two families, or to the participants of a meal or banquet.

Prevalence.—There is a common opinion that food infection is exceedingly frequent. This is a mistake, certainly in this country, although no satisfactory figures are available. Outbreaks apparently occur much more frequently on the continent of Europe than in England, and more frequently in England than in the United States. Savage tabulates only 112 British outbreaks of bacterial origin, studied from 1878 to 1918. Savage and White⁶⁹ studied one hundred recent outbreaks (1921-1923) of the less severe evanescent nature, and due mostly to *Bacillus ærtrycke*. Mayer presents a list of forty-eight food poisoning outbreaks which occurred in Germany between 1888 and 1911, and which were attributed to *B. enteritidis*. Bainbridge states that in Germany between 1898 and 1908 there were at least 261 outbreaks due either to *B. suispestifer* or *B. enteritidis*. Ostertag, in 1902, was able to collect records

⁶⁹ Spec. Rep. No. 91, Medical Research Council, 1925.

of but 85 epidemics in the period 1880 to 1900, mostly of German sources. Very few outbreaks appear in the literature from the United States. Failure to report cases accounts for our incomplete knowledge of the prevalence of the disease.

*An Illustrative Outbreak.*⁷⁰—The following outbreak, investigated by Savage in 1908, is given as a typical illustration, and points out as well the difficulties which confuse us in most studies of this disease.

On Friday, May 8, 1908, in Murrow, a village in Cambridgeshire, a woman purchased some pork bones from a local butcher and that evening used them to make some brawn. The following morning the brawn was emptied out of the saucepan in which it had been made and, without cleansing the vessel, potatoes and asparagus were cooked in it. These vegetables were eaten for midday dinner by four persons and all were subsequently attacked with vomiting, diarrhea, and the other symptoms of food poisoning, two in the night and two the next morning. The husband, who was away at midday, remained well and unaffected.

On Monday, two days later, the brawn made up into pork cheeses (a local name for brawn) was given away to three different neighbors and was consumed by a further fourteen persons, all of whom were attacked with similar symptoms after an incubation period varying from twelve to forty-eight hours. Three of the eighteen attacked died. No one eating the brawn escaped.

None of the brawn was available for examination, but from the only fatal case investigated a Gärtner group bacillus (*B. ærtrycke*) was isolated and its connection with the outbreak was further proved by the fact that it was agglutinated in high dilution by the serum of three survivors.

The brawn was home prepared, and the materials were slowly heated for several hours with a short boil at the finish, but obviously actual boiling temperature was not reached. That the Gärtner bacilli were present before preparation and survived cooking is evident from the infection imparted to the vegetables through the uncleansed saucepan. Further inquiries elicited that the pig which supplied the bones for the brawn had suffered from local injury or disease of one leg, no doubt due to infection by this food-poisoning bacillus.

Here are all the commonly present features of such outbreaks: a typical group of symptoms, a number of cases geographically separated but linked by a particular food consumed in common, a special bacillus demonstrated to be the pathological cause, and lastly (unlike most outbreaks) with definite evidence connecting it with disease in the animal supplying the incriminated food.

Kind of Food Responsible.—The great majority of outbreaks are due to meat or meat mixtures. Of the 112 British outbreaks, in twenty-one the vehicle was a non-flesh food; that is, milk, one; cream, one; ice cream, six; potatoes, two; pineapple jelly, one; canned peaches, one; rice cooked in fat, one. The remaining ninety were all due to flesh food, mainly brawn, meat

⁷⁰ See also M. J. Rosenau and H. Weiss, "Food Infections with an Illustrative Outbreak," *J. Am. M. Ass.*, 1921, 77: 1948.

pies, pork, ham, beef, etc. The meat of the pig or ox accounts for 68 per cent of the British and 61 per cent of the continental outbreaks. The almost complete absence of outbreaks due to the meat of the sheep is striking. The number of cases ascribed to fish is small.

Most outbreaks are due to some form of prepared meat foods, such as brawn, meat pies, sausage, chopped meat, etc. When the nature of the infection is considered, the more the food is handled and the longer it lies around, the greater the opportunity for it to become contaminated and for the bacteria to grow and multiply. A prodigious overgrowth of bacteria occurs in milk and meat mixtures prepared in advance and allowed to stand at room temperature.

Taste, Odor and Appearance.—It cannot be too strongly emphasized that in the vast majority of outbreaks of food infection the food affected is not noticeably altered in appearance, taste or smell. The prevalent idea that "poisonous" food must be tainted still persists, although long exploded. The presence of bacilli of the Gärtner group is not disclosed any more than typhoid, dysentery or cholera infection can be detected in meat, milk or water by our unaided senses.

In the Ghent outbreak, investigated by van Ermengem in 1895, a veterinary surgeon, in his office as slaughterhouse inspector, was so certain that the suspected meat (saveloy), in the absence of any abnormal signs, could have no connection with the trouble, that he ate two or three pieces of it to demonstrate its harmlessness. He was attacked with severe cholera-like symptoms, and died five days later, the Gärtner bacillus being recovered postmortem. We admire his confidence, but learn a lesson from his tragic story.

In some outbreaks, peculiarities of the food have been noted, such as objectionable flavor, heavy odor, moist or soft condition, etc. However, it is not so much decomposed as infected food that may be harmful.

Diagnosis.—Diagnosis of food infection depends upon: history of exposure to the suspected food; symptoms suggestive of food poisoning; isolation of the infecting organisms from the suspected food, and also from the blood, urine, feces, or viscera of the patient; specific identification of the causative organism by agglutination tests; demonstration of agglutinins in the blood-serum of patients. Agglutinins appear six or eight days after the onset of symptoms. Agglutination in comparatively low dilutions is usually accepted as diagnostic owing to the fact that it is unusual to find a positive reaction for *B. enteritidis* or *B. suispestifer* in normal individuals. Bacilli appear in the blood early during the fever. They disappear quickly from the feces, rarely persisting more than seven to ten days.

Acute attacks with gastro-enteric symptoms are not necessarily due to harmful foods. This is a common mistake in diagnosis. Nausea, vomiting, cramps or diarrhea may be due to indigestion, indiscretions in diet, eating when fatigued; also to exposure. The gastro-intestinal tract is exceedingly sensitive to reflex nervous influences; thus, emotion may cause vomiting or diarrhea. Nervous exhaustion is a frequent cause of gastro-enteric disturbance.

THE COLON-TYPHOID GROUP

BACILLUS	LACTOSE		DEXTROSE		MANNITE		MALTOSE		SACCHAROSE		INDOL	MO- TILITY	REMARKS
	Acid	Gas	Acid	Gas	Acid	Gas	Acid	Gas	Acid	Gas			
<i>B. cloacæ</i> , Jordan	+	+	+	+	+	+	+	+	+	+	+	+	liquefies gelatin
<i>B. coli</i> communior, Durham	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. lactis aërogenes</i> , Escherich	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. coli</i> communis, Escherich	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. acidii lactici</i> , Huppe	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. coli</i> anaërogenes, Lemblee	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. paratyphosus α</i> Schottmüller	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. paratyphosus β</i> Schottmüller	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. enteritidis</i> Gaertner	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. cholerae</i> suis, Salmon & Th. Smith ..	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. typhi</i> murium, Loeffler	+	+	+	+	+	+	+	+	+	+	+	+	indol variable
<i>B. psittacosis</i> , Nocard	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. icteroides</i> , Sanarelli	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. dysenteriae</i> , Shiga	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. dysenteriae</i> , Hiss "Y"	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. dysenteriae</i> , Flexner	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. dysenteriae</i> , Strong	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. dysenteriae</i> , Rosen	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. Morgan</i> No. 1	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. typhosus</i> , Eberth	+	+	+	+	+	+	+	+	+	+	+	+	
<i>B. fecalis</i> alkaligenes, Petruschky	+	+	+	+	+	+	+	+	+	+	+	+	

All the members of this group are Gram-negative bacilli. They all have their normal habitat in the intestinal tract. As a rule, those that are most active in fermenting sugars are the least pathogenic, and vice versa.

Organic diseases of the heart or kidneys are often associated with gastric symptoms. Nausea and vomiting often usher in acute infectious diseases, especially in children. The importance of a careful differential diagnosis is evident. This has been emphasized by our studies at Harvard, in which all of the above mistakes were observed.

THE COLON-TYPHOID GROUP

It is important to have a clear knowledge of the colon-typhoid group of bacteria in order to have a better understanding not only of food infection, but of many other phases of preventive medicine. Therefore, the essential features of these bacilli are given.

The colon-typhoid group is a long chain of important organisms with the highly specific typhoid bacillus at the upper end, and the lowly and common colon bacillus at the other. Some of the important links in this chain are the paratyphoid bacilli, the dysentery bacilli, the hog-cholera bacilli, the *Bacillus psittacosis* (a disease of parrots communicable to man), the *Bacillus icteroides* (once associated with yellow fever), the *Bacillus typhi murium* (the bacillus of mouse typhoid, the type of the bacterial rat viruses), the *Bacillus enteritidis* of Gärtner (associated with food infection and diarrheal diseases), and many others.

All the bacilli in this group are short rods with rounded ends, with a tendency to form threads; they have no spore; are Gram-negative; grow readily on ordinary culture media and do not liquefy gelatin. Their normal habitat appears to be the intestinal tract of man and animals, but they are widely distributed in nature.

The organisms comprising this group are closely related. Intermediate strains exist, and therefore it is difficult to determine where specific differences begin and end. Some of these intermediate strains may be missing links. A study of these closely related organisms excites the imagination to the belief that we may here see evolution in the making.

The differentiation of the non-lactose fermenters based upon fermentation tests is shown in the following diagram from Savage.⁷¹

THE GÄRTNER GROUP

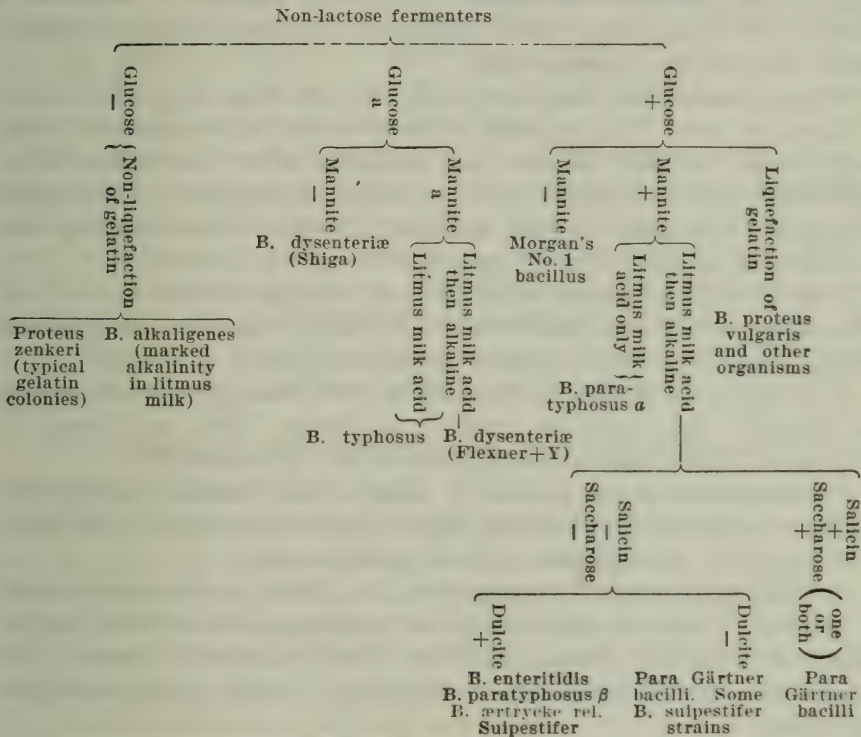
The Gärtner group, of which *B. enteritidis* is the type, occupies an intermediate position between the colon bacillus on the one hand, and the typhoid bacillus on the other. It is also called the "intermediate group," the "hog-cholera group," the "enteritidis group," the "paratyphoid group," and the "Salmonella group."

The classification of the members of this group is quite involved, and there is still a lack of agreement among different investigators concerning some of the details. The important members are as follows:

⁷¹ *Food Poisoning and Food Infections*, Cambridge University Press, 1920.

B. Enteritidis.—Gärtner, in 1888, brought forth the first definite evidence which incriminated bacteria as an etiologic factor in food poisoning. At Frankenhausen, fifty-seven individuals became ill after eating the flesh of a cow which had been slaughtered on account of enteritis; one case resulted fatally. Gärtner isolated the bacillus from the organs of the cow and also from the spleen of the man who died. He called the organism *B. enteritidis*. It has since been shown to be the common etiologic factor in food poisoning. It is the type of the group.

DIFFERENTIATION OF THE TYPHOID COLON GROUP



+ = fermentation with production of acid + gas.

a = fermentation with production of acid only.

- = no acid or gas production.

B. morbificans bovis was isolated by Basenau in 1893 from the muscles and viscera of a cow killed on account of puerperal metritis. There is little doubt but that this organism is identical with *B. enteritidis* of Gärtner.

B. suipestifer, also called *B. cholera suis*, or the hog-cholera bacillus, was isolated by Salmon and Theobald Smith in 1885. For a long time this was believed to be the cause of hog-cholera, but it is now known to be a secondary invader. It is often used as a group type. Man is not susceptible to hog-cholera, which is due to a filtrable virus. There are doubtless many strains of the hog-cholera bacillus, only some of which are pathogenic

for man, for no relationship has been found between hog-cholera and food poisoning in man; in fact, pork has been eaten many times from hogs with hog-cholera without ill effect. This is an illustration of the difficulties in distinguishing between closely allied strains.

B. ærtrycke was isolated in 1898 in Hatton, England, and by De Nobele in Ærtrycke, Belgium. This organism is known to be identical with *B. suipestifer*. Most of the English outbreaks are associated with *B. ærtrycke*.

B. psittacosis was isolated by Nocard in 1893 from the bone marrow of parrots which had died en route from Buenos Aires to Paris. Several persons in Paris contracted this infection from parrots, and were stricken with a highly fatal pneumonia-like disease. Bainbridge regards *B. psittacosis* as identical with *B. suipestifer*. The organism is now only of historical interest, because there has been no recurrence.

B. typhi murium was isolated by Löffler in 1893 from an epizootic among his laboratory mice. This organism is closely related to *B. suipestifer*. Bainbridge pointed out that the name was applied to either pure cultures of *B. enteritidis*, or to mixed cultures of *B. enteritidis*, *B. suipestifer*, and *B. paratyphosus* β . Shibayama reported instances of human illness resulting from infection with the cultures of *B. typhi murium* used as a rat virus.

Danzs's rat virus consists of a culture of a bacillus isolated from an epizootic among harvest mice, the virulence of which is raised for rats by animal passage. Bainbridge believes that it is identical with *B. enteritidis*.

B. icteroides was isolated by Sanarelli in 1898 from yellow fever patients, and believed by him to be the cause of the disease. Reed and Carroll, in 1899, showed that it was culturally identical with *B. suipestifer*.

B. paratyphosus β was isolated by Acharde and Bensaude in 1896, but carefully studied by Schottmüller in 1900. Brion and Kayser, in 1902, named the two types *B. paratyphosus* α and *B. paratyphosus* β .

In England, the term *B. paratyphosus* β is limited to those strains which are identical, by culture, agglutination and absorption tests, with the original strain of Schottmüller. Such a restriction is not yet generally accepted, however, and consequently some confusion has arisen. See also paratyphoid fever, page 125.

The English, due chiefly to the careful observations and absorption tests, of Bainbridge, Bainbridge and O'Brien, and the studies of Savage, believe that only three of these organisms are involved in cases of food infection: namely, *B. paratyphosus* β , *B. suipestifer* and *B. enteritidis*. The Germans, however, regard *B. paratyphosus* β and *B. suipestifer* as one and the same organism. If the strain comes from an animal they call it *B. suipestifer*; if from man, *B. paratyphosus* β .

It is evident to the student of bacteriology that the confusion in this and other groups is due first of all to the fact that we have no clear-cut criteria of genera, species and strains among bacteria; second, to the fact that we are dealing with biologic variables. Thus, a strain may be cultivated so that certain cultural and biologic properties are entirely changed. These differences

are probably always variants, rarely if ever of the magnitude of mutants. It is true that sometimes the changes thus produced seem fixed in that they breed true to type, but usually they revert when placed under proper conditions. Furthermore, we do not know the fundamental significance of the differences between the closely allied members of the group.

Toxin Production.—True toxins have not been demonstrated with any organism belonging to the colon-typhoid group. Almost all students of this subject have found that filtered cultures of *B. enteritidis* and other organisms of the Gärtner group are toxic when injected into experimental animals. There is a great difference of opinion concerning the properties of the toxic substance. Some have emphasized the inconstancy of production and their instability, while others have found them constant and their toxic properties to persist indefinitely and to be heat resistant. Ecker⁷² reviewed the literature on the subject up to 1917, and Branham⁷³ summarized in convenient and tabular form all the investigations to 1925.

There seems to be a well-grounded suspicion that poisons of some sort are formed in the food before it is eaten. This supposition is used especially to explain instances of food poisoning with a short period of incubation. This theory is entirely conjectural. Endotoxins have been invented to account for the facts. Laboratory experiments with filtrates show a toxic property when injected into experimental animals, but not when given by the mouth. How bacilli belonging to the Gärtner group injure the host is part of the problem of the pathogenesis of infectious diseases, which is still far from solution.

SOURCES OF INFECTION WITH THE GÄRTNER GROUP

Diseased Animals.—*Antemortem Infection.*—*B. enteritidis* and its congeners are pathogenic for some of our food animals, especially cattle and hogs, as well as for man. Cattle suffering during life from puerperal fever, uterine inflammations, navel infection (in calves), septicemia, septic pyemia, diarrhea, and local suppurations are apt to furnish meat containing the Gärtner bacillus or closely related bacilli. Such meat has given rise to food infection. Hence, emergency slaughter (Nothschlacht), unless intelligently supervised, furnishes meat that may be a menace. The meat of such animals presents no warning signs of its danger. This fact was well proved by the meat inspector at Ghent who lost his life because he was so sure that meat must be tainted to be harmful. The story of this tragedy has become classic (page 646).

Postmortem Infection.—The meat may come from healthy animals, but become infected after slaughter. This may take place through the hands or instruments of the butcher who has just handled a diseased carcass.

Human Carriers.—Human carriers of *B. enteritidis* do not occur. Bainbridge states that "infection of meat by human carriers of *B. suispestifer* is unknown." Human carriers of *B. paratyphosus* β occur, and when meat is

⁷² *J. Infect. Dis.*, 1917, 21: 541.

⁷³ *J. Infect. Dis.*, 1925, 37: 291.

infected by a carrier it is assumed always to be one of the paratyphoid group. In 4,154 specimens of human feces from healthy individuals examined in my laboratory in 1917-18, not a single carrier of any of these non-lactose fermenters was found.

The English observers believe that all cases of food poisoning traced to human carriers are in reality cases of paratyphoid fever, but the Germans, who do not differentiate between *B. paratyphosus* β and *B. suispestifer*, hold that the latter organism also occurs in normal, healthy human intestines, and may thus become a source of food infection. Such instances must be exceedingly rare in the United States, for in our studies of the subject we did not find a single instance in three years.

Rats and Mice.—The common gray rat and mice may harbor *B. suispestifer* and *B. enteritidis* as carriers. Zwick and Weichel examined 177 mice and found that twenty-eight were acting as carriers of Gärtner group bacilli. Hence, food may become infected by contamination with rat or mice feces. There is abundant opportunity for such contact in the slaughterhouse, in butcher shops, in refrigerator plants, in transportation, and in the home.

Rat Viruses.—Food has occasionally become contaminated by contact with bacterial viruses used against rats. These viruses are pure cultures of *B. typhi murium*, closely allied to *B. enteritidis*. Shibayama reported instances of human illness resulting from the free use of such cultures, which contaminated a bowl of food.

Food Poisoning of Non-Specific Bacterial Origin.—A number of other bacteria have been associated with food poisoning, such as *B. proteus*, *B. faecalis alcaligenes*, *B. prodigiosus*, and even the colon bacillus itself. There is slight evidence that any of these are ever responsible for the trouble in question. It is not to be denied that massive growths of bacteria that ordinarily are harmless may cause gastro-intestinal irritation.

That this is unlikely, or at least unusual, is evidenced by the enormous numbers of bacteria, including fecal bacteria, taken with milk, cream, butter and other milk products; also in cheese, sauerkraut, sausage, etc., etc. Food very massively infected with *B. coli* and other intestinal bacteria and given to animals to eat does not cause illness. Linden^{73a} traced an outbreak of food poisoning to streptococci in cheese.

Prevention.—The prevention of food infection starts with the meat inspector (see page 743). Animals suffering with any septic or pyemic lesions, whether local or generalized, should be condemned as unfit for food purposes, unless bacteriologic examination excludes the Gärtner group bacilli. These infections may be discovered either antemortem or postmortem. Food animals suffering with puerperal sepsis or enteritis are especially suspicious. The greatest care should be taken with sick animals killed under the provisions of emergency slaughter (Nothschlacht), especially cattle and hogs.

Pains must be taken to disinfect hands, tools and other contaminated objects and surfaces in case such an animal comes to the slaughterhouse, to avoid

^{73a} U. S. Pub. Health Rep., 1926, 41: 1647.

spreading the infection to other carcasses. The bacteria grow well in meat. Cleanliness must be exercised in butchering, handling, storing and transporting. Every care must be exercised to guard food against defilement with rat and mice feces.

Food should not be handled more than necessary. The health of food handlers is obviously important. It is especially prepared mixtures, such as chopped meats, sausage, meat pies, brawn and salads, that cause trouble. Such fussy and fingered foods are not necessary and are to be avoided. Food mixtures prepared hours before the meal and allowed to stand at room temperature permit bacteria to grow and multiply.

It should be remembered that articles other than meat are sometimes infected, especially milk, cream, ice cream—the list is long and comprehensive. Cleanliness and freshness are the watchwords.

Food infections are especially apt to take place from foodstuffs that have not been properly refrigerated. *B. enteritidis* may grow and multiply at temperatures as low as 10° C. Foods that have been handled, chopped, fixed or prepared some hours before the meal are most apt to give trouble; hence, the frequency with which food poisoning is associated with picnic lunches, fraternity spreads and banquets.

Cooking is our ultimate safeguard. These non-spore-bearing bacilli are readily killed. The cooking must be thorough and should be recent, that is, just before serving (see page 691).

BOTULISM (Food Toxemia)

Botulism is a specific toxemia caused by the toxin of *Clostridium botulinum* (formerly called *Bacillus botulinus*). The bacillus grows in a great variety of foodstuffs, both of plant and animal origin, and produces its poison in the food before it is eaten. The name botulism (from *botulus*, a sausage) has lost its original significance.

Botulism stands alone as a type of food poisoning. It is well understood and is the only known instance in its class. The bacillus itself is essentially a harmless saprophyte. Botulism further differs from food infection in that the attack is chiefly upon the central nervous system. Acute gastro-intestinal disturbances usually do not occur. There is no fever.

A complete history and a thorough review of the literature is found in Dickson's monograph.⁷⁴

Etiology.—The cause of botulism was demonstrated by van Ermengem⁷⁵ who studied a series of cases which occurred at Ellezelles, in Belgium, in 1895. In the first of these outbreaks, twenty-three persons became ill and three died after eating ham which had been preserved in brine. From portions

⁷⁴ *Monogr. Rockefeller Inst. M. Research*, No. 8, July 31, 1918.

⁷⁵ *Arch. Pharmacol.*, 1897, 3: 213; *Centralbl. f. Bakteriol., Ite Abt.*, 1896, 19: 442; *tschr. Hyg. u. Infektionskrankh.*, 1897, 26: 1.

of the ham and from the spleen and intestinal contents of one of the victims, van Ermengem succeeded in isolating a Gram-positive, spore-bearing anaërobic bacillus, to which he gave the name *Bacillus botulinus*. He found that infusions of the macerated ham, and bouillon cultures of the bacillus produced the typical symptoms of botulism in guinea-pigs, rabbits, cats, pigeons and monkeys. The bacillus itself he believed to be a saprophyte, and the poisoning to be due to a toxin which is formed when it grows in food under anaërobic conditions.

Prevalence.—Botulism has been recognized by German clinicians since 1735, when the first authenticated case was recorded. The outbreak which first attracted the attention of the medical profession occurred in 1793 in Wildbad in Württemberg, where thirteen persons became ill and six died after eating sausage packed in the stomach of a hog, and which contained a great deal of blood. The number of cases reported in Germany is as follows:

YEARS	CASES	DEATHS
From 1793 to 1820	76	37
From 1820 to 1822	98	34
From 1822 to 1886	238	94
From 1886 to 1913	about 800	about 200

Dickson ⁷⁶ was able to tabulate, during the twenty years prior to 1917, twenty-two outbreaks of botulism in the United States. Of these, eighteen occurred on the Pacific Coast, seventeen in California and one in Oregon. These outbreaks involved eighty-one individuals, fifty-five of whom died—a mortality of 67.9 per cent.

There have been reported in the United States and Canada from 1899 to 1926, and including one outbreak from England, 147 outbreaks, or a total of 504 cases with 337 deaths, giving a case mortality of 67 per cent; fifty-four outbreaks have been proved toxicologically and bacteriologically. An average of about thirteen outbreaks occur annually in the United States. These figures are given in full to emphasize the fact that botulism is comparatively a rare disease.

Botulism is more common in Europe than in this country. The first and only outbreak known in the British Isles occurred in August, 1922, at Loch Maree, causing eight deaths. The outbreak was traced to wild duck potted paste.

Botulism presents no particular distribution as to sex, age, season, or social condition.

Botulism is one of the causes of forage poisoning in horses, and of limber-neck in chickens and turkeys, and may also be responsible for various types of paralysis in domestic animals, including dogs.

⁷⁶ *J. Am. M. Ass.*, 1917, 69: 966.

Period of Incubation.—The symptoms usually appear from eighteen to thirty-six hours after ingestion of the poisonous food. However, cases are on record in which the incubation period has been as short as four hours or as long as six days. There is a direct correlation between the severity of the poisoning and the period of incubation. A short period indicates a severe and often fatal intoxication. In a series of 246 cases collected by Geiger, of which 173 resulted fatally, 147, or 85 per cent, of the fatal cases were persons in whom the period of incubation was less than forty-eight hours.

Symptoms.—The earliest symptom is usually a peculiar indefinite indisposition, associated with a feeling of fatigue, sometimes headache and dizziness, and definite muscular weakness. When the period of incubation is short, the first symptoms may be gastric distress, nausea, vomiting and occasionally diarrhea, which, however, are transient. Botulism differs from the common type of food poisoning, in that there are usually no indications of acute gastrointestinal irritation. Constipation is almost constant.

Disturbances of vision occur early, and are scintillation and dimness of vision, sometimes progressing to blindness, due to impairment of both the extrinsic and intrinsic muscles of the eye. The third cranial nerve is early involved, causing blepharoptosis, dilatation of the pupils, loss of reflex to light, and diplopia. Loss of accommodation soon becomes complete. Nystagmus, strabismus, and vertigo and sometimes photophobia occur. The ophthalmoplegia is merely a phase of the more general paralysis, but is a conspicuous symptom, owing to the striking character of its manifestations.

Coincident with, or closely following the onset of disturbances of vision, the patients complain of difficulty of swallowing and talking, and frequently there is a peculiar sensation of contraction of the throat. The mouth is dry and attacks of strangling occur. Thick, glairy mucus, with dryness of the throat leads to an ineffectual cough. The breath is offensive and fetid. Complete paralysis of peristalsis causes the stubborn constipation.

A striking feature is the progressive muscular weakness which in severe cases closely simulates paralysis. Incoördination of muscular movement is common. The *paralysis* is usually of the ascending type, manifesting itself first in the intestines, perhaps due to involvement of the mesenteric plexus, then gradually passing upward, progressively involving higher centers, until the medulla is reached. The motor areas seem almost never to be involved, although in the Montana outbreak paralysis of the right arm and leg was observed.

The *loss of nervous tone* manifests itself in vague, indefinite indisposition, marked fatigue, dizziness, headache, restlessness, indefinite sensations of chilliness, incoördination and unsteadiness in walking with a tendency to a "step-page" gait, great muscular weakness and sometimes urinary incontinence. The ophthalmoplegia may be partly responsible for some of the above symptoms.

Botulism is characterized by an almost complete absence of sensory disturbances. It is unusual to suffer pain, and the mind remains clear.

Inhibition of many of the secretions, especially saliva, sweat, and tears, is an almost constant manifestation of botulism. Oliguria has been noted.

The pulse is usually rapid, and the temperature subnormal. Fever developing late in the poisoning indicates bronchopneumonia. Respiration at first is not impaired but later in the course of the illness disturbances of respiration become very severe. Difficult articulation and perhaps complete aphonia, accompanied by an inability to swallow, soon appear, due to paralysis of the laryngeal and pharyngeal muscles. Increasing difficulty in breathing, leading eventually to death due to paralysis of the respiratory center, brings the scene to a close.

The general appearance of the patient is distressing; the muscular weakness, the anxiety and utter helplessness, the difficulty in swallowing, the attacks of strangling, the struggle for breath, with its resulting cyanosis, and the unsuccessful attempts to articulate constitute a clinical picture which once seen can never be forgotten. It is essentially that of a bulbar paralysis, with the earliest symptoms indicating injury high up in the brain stem. The disease must be differentiated from other causes of bulbar paralysis and ophthalmoplegias, such as encephalitis lethargica, poliomyelitis, cerebrospinal syphilis; also poisoning from belladonna, gelsemium, hyoscyamus and methyl alcohol. There is no constant pathologic lesion.

The duration varies greatly. Death may occur in forty-eight hours after eating the poisonous food; as a rule, it occurs in from four to eight days, and few die after ten days. Dickson reports one death on the twenty-sixth day. Death usually is due primarily to respiratory failure. Convalescence is extremely slow and tedious. The disturbances of vision and weakness may last for months.

The case fatality rate in botulism has varied greatly in different outbreaks, depending upon the amount and virulence of the toxin. In certain instances it has been extremely high—in some 100 per cent—but in others it has been correspondingly low. In the United States it is high, 61.7 per cent, and surprisingly uniform. Wosnitza⁷⁷ recorded a series of fifty-nine cases of which only four died. In Kerner's series⁷⁸ of 159 cases, there were eighty-four deaths, a mortality of 52.8 per cent, and in Schlossberger's series⁷⁹ of 400 cases there were 150 deaths, a mortality of 37.5 per cent. The most complete collection of cases is that of Mayer,⁸⁰ in 1913, in which he reports 812 cases of which 365 were fatal, a mortality of 44.9 per cent.

The Bacillus.—*Clostridium botulinum*, discovered by van Ermengem in 1895, is a large, slightly motile rod with rounded ends; four to eight

⁷⁷ *Inaugural Dissertation*, Leipzig, 1909.

⁷⁸ *Neue Beobachtungen über die in Württemberg so häufig vorkommenden tödlichen Vergiftungen durch den Genuss geräucherter Würste*, Tübingen, 1820; *Das Fettgift, oder die Fettsäure, und ihre Wirkungen auf den thierischen Organismus. Ein Beitrag zur Untersuchung des in verdorbenen Würsten giftig wirkenden Stoffes*, Stuttgart and Tübingen, 1822.

⁷⁹ *Arch. de Physiol. Heilk.*, 1852, 11: 709.

⁸⁰ *Deutsch. Vrtljschr. off. Gsndtspf.*, 1913, 14: 8.

flagellæ, generally single; rarely occurs in filaments; has a large polar spore; stains readily, and is Gram-positive. It is anaërobic. The spore is a true endospore.

The bacillus grows well at room temperature, between 20° and 30° C., but also thrives at 37° C. Contrary to previous opinion, it may grow in the body. It is a strict anaërobe. It grows under special conditions in symbiosis with certain aërobic bacteria, such as a white sarcina (van Ermengem) or with *B. subtilis* (Romer); and in freshly prepared bouillon under aërobic conditions if a piece of sterile flesh or potato is placed on the bottom of the culture tube. The addition of glucose to the culture medium greatly increases its activity in growth and in toxin formation. In a medium consisting of one part sheep's brain and two parts water it grows well and produces an abundance of spores. The strains studied by van Ermengem produced practically no change in the appearance of milk, but von Hibler and others find that milk casein is precipitated and peptonized. It is strongly proteolytic, and a putrefactive odor is given off. Gas which is usually formed is due to the fermentation of sugars in the medium. The twenty-one cultures studied in my laboratory show minor differences in cultural characteristics; only ten of these strains produce toxin. Atoxic strains must be differentiated from *B. sporogenes*.

Types.—There are at least three and perhaps more types of *Cl. botulinum*. Type A produces a strong toxin and is associated especially with the disease in America. Type B seems to be more widely scattered and, while sometimes responsible for the disease in the United States, is found more particularly in Europe. Of a series of outbreaks studied bacteriologically in the United States, twenty-three were found to be due to type A and only three to type B. Both types may cause botulism in animals as well as in man. Type C, recently isolated by Bengtson, is not known to cause poisoning in man. Some forms of "fish poisoning," especially those reported in Japan, as well as parabolism of cattle in South Africa, are probably types of botulism. There evidently exist a group of related organisms.

Habitat and Distribution.—The spores of *Cl. botulinum* are widely distributed in nature, and probably have their normal habitat in the intestinal tract of herbivora. Hence, they occur almost everywhere in dust, dirt and carrion. They are found in soil, on fruit and vegetables, in the larvæ of worms, in dust and on food. They have been found in many parts of the world, even in the virgin soil of mountains. The habitat and distribution resemble those of other intestinal spore-bearing anaërobes, such as tetanus and the gas bacillus.

The distribution is regional. In the United States type A is the cause of most outbreaks, while in Europe type B is the prevalent variety. This corresponds to the distribution and virulence of these types. In the United States, most of the cases of botulism occur west of the Rocky Mountains or are associated with food which originates there. The frequent occurrence of botulism in southern California seems closely connected with the distribution of *Cl. botulinum* spores in the soil. A thorough study of the habitat and

distribution of the spores in nature has been made by Meyer and his colleagues.⁸¹

Thermal Death-Point of the Spore.—To safeguard against botulism in canned foods, the thermal death-point of the spore was determined in my laboratory by Weiss.⁸² The various factors, such as age of the spore, number of spores, strain differences, hydrogen ion concentration, effect of desiccation, etc., which influenced the thermal death-point were also determined.

Weiss found that, suspended in water, the most resistant types require five hours at boiling temperature, forty minutes at 105° C., fifteen minutes at 110° C., and six minutes at 120° C. These represent bath temperatures

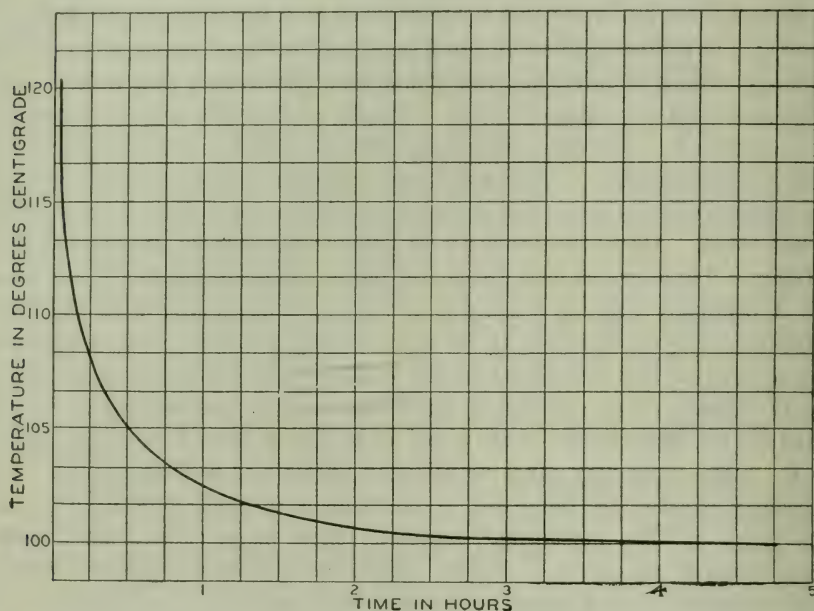


FIG. 55.—CURVE I, SHOWING THERMAL DEATH-POINT OF *CLOSTRIDIUM BOTULINUM*.

and include the time necessary to heat the spore itself to the temperature stated. Weiss further found that young spores are more resistant than old spores; that dry spores are much harder to kill than moist spores; and that acids, alkalies and various chemicals greatly diminish the thermal resistance.

Toxin.—The bacillus produces a soluble, true exotoxin comparable in all respects to the poisons produced in cultures of diphtheria or tetanus. The botulinus toxin is the only one of the true toxins that is poisonous when taken by the mouth. It is thus pathogenic for guinea-pigs, mice, and monkeys, as well as for man. One or two drops of a culture placed upon a piece of bread causes death in a few days. Toxins of diphtheria and tetanus are

⁸¹ *J. Infect. Dis.*, 1922, 31: 501-665. Also a series of subsequent papers.

⁸² *J. Infect. Dis.*, 1921, 28: 70.

not poisonous when taken by the mouth. I have failed to produce tetanus by giving as much as 100,000 M.L.D. to susceptible guinea-pigs or mice.

The toxin is secreted by the bacillus when it grows upon a suitable medium, under anaërobic conditions. Some strains do not produce toxin, and the property may be lost after prolonged artificial cultivation. Toxin production takes place between 20° and 30° C., but best at 37° C., contrary to our previous conception. It has always been believed that *Cl. botulinum* is strictly saprophytic and will not develop and produce its toxin in the body, the toxin being always performed in foodstuffs, but the recent work of Orr⁸³ in my laboratory shows that guinea-pigs and mice may develop botulism when fed enormous quantities of toxin-free spores. The significance of this observation to human botulism must await further study.

Botulinus toxin is exceedingly poisonous. It has been possible to obtain a toxin of which 0.000001 cubic centimeter would kill a 250-gram guinea-pig in from three to four days.⁸⁴ In the Ellezelles outbreak,⁸⁵ about 200 grams of the poisonous ham were sufficient to cause the death of one of the patients; in the Darmstadt outbreak,⁸⁶ a piece of preserved duck the size of a walnut was sufficient to cause an illness which lasted for eight weeks. In Dickson's series of cases, one patient died after "nibbling" a portion of a pod of the spoiled string beans, one died after tasting a small spoonful of the spoiled corn, and a third was ill after tasting a pod of beans which she did not swallow.

Strong toxin was produced by Dickson in pork and beef infusion, and also in media prepared from string beans, green corn; much less virulent toxins were obtained in media prepared from asparagus, artichokes, apricots, and crushed apricot stones. The most powerful poisons are produced in glucose broth. The toxin is not formed in brine containing over 8 per cent of sodium chlorid or in syrup containing over 50 per cent of sugar. This is of practical importance in pickling and preserving foods.

Van Ermengem⁸⁷ showed that the toxin was destroyed by heating at 80° C. for one-half hour, and many later workers have verified this, showing in fact that there is a large margin of safety in such a procedure. Thom, Edmondson and Giltner⁸⁸ showed that the toxin of the Boisé strain is destroyed at some point between 70° C. and 73° C. by heating for ten minutes. Orr⁸⁹ showed that the most resistant of the ten strains of toxin studied by him is destroyed when exposed to 80° C. for two minutes, 72° C. for ten minutes, and 65° C. for 85 minutes. The toxins of most strains are killed at 65° C. in thirty minutes. Cooking therefore is a safeguard. The toxin in solution is very resistant to exposure to light and air. It is not affected by drying or putrefaction.

The toxin has a special affinity for the central nervous system; it is almost

⁸³ *Proc. Soc. Biol. & Med.*, 1919, 17: 47.

⁸⁴ *Deutsche med. Wchnschr.*, 1897, 23: 521.

⁸⁵ *Arch. Pharmacol.*, 1897, 3: 213.

⁸⁶ *Deutsche med. Wchnschr.*, 1897, 23: 521.

⁸⁷ *Arch. Pharmacol.*, 1897, 3: 213.

⁸⁸ *J. Am. M. Ass.*, 1919, 73: 907.

⁸⁹ *J. Med. Research*, 1920, 42: 127.

a pure neurotoxin. It may also act upon the blood-vessels, causing dilatation, thrombi and hemorrhages in various portions of the body.

Van Ermengem⁹⁰ found that white mice, guinea-pigs, rabbits, cats, pigeons, and monkeys are susceptible to subcutaneous, intraperitoneal, and intravenous injection, and that white rats, dogs, chickens, frogs and fish are highly resistant. In his feeding experiments, he found that mice, guinea-pigs, and monkeys are especially susceptible, rabbits are less susceptible, and cats must be given enormous quantities of the toxin before they show symptoms. He found that dogs, rats, and chickens are practically unaffected, the only

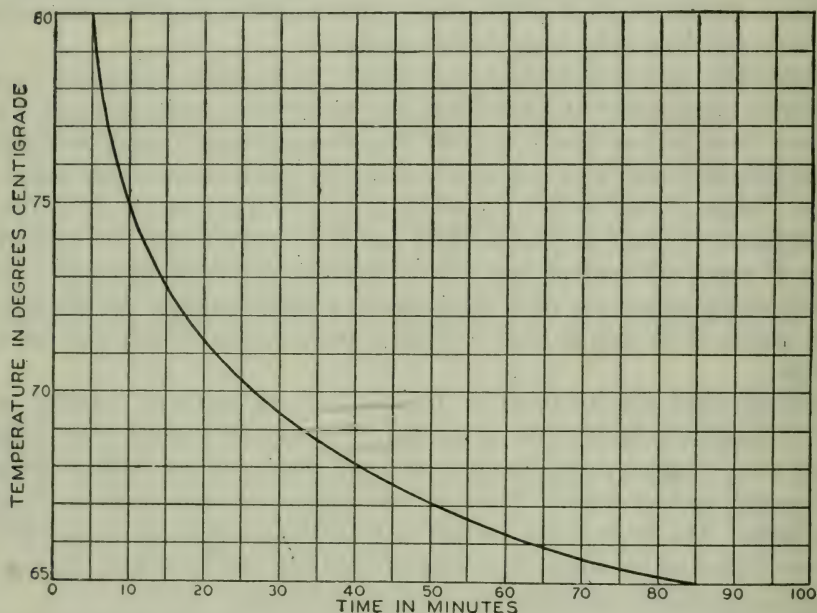


FIG. 56.—CURVE II, SHOWING RATE OF DESTRUCTION OF BOTULINUS TOXIN.

result of feeding very large doses being vomiting, diarrhea and emaciation. It is surprising that van Ermengem failed to obtain positive results in his experiments with dogs and chickens, as Dickson and others have found that botulism may cause limber neck in chickens and turkeys. Horses, goats and other animals are susceptible.

Antitoxin.—A true antitoxin may be obtained by injecting increasing amounts of the toxin into susceptible animals. Kempner first obtained the antitoxin in goats. It is now made by injecting horses. The botulinus antitoxin has both protective and curative virtues in experimental animals, even when given twenty-four hours after the ingestion of the poison, but before

⁹⁰ *Arch. Pharmacol.*, 1897, 3: 213; *Ztschr. Hyg. u. Infektionskrankh.*, 1897, 36; 1; *Handbuch der Pathogenen Mikroorganismen*, Kolle u. Wassermann, Jena, 2nd Ed., 1912, Vol. IV, p. 909.

the onset of symptoms. For practical purposes at least two antitoxins must be on hand, one made by strain A, and the other by strain B.

Like other antitoxins, the botulinus antitoxin must be given very early if it is to be effective, and it is probable that, as in tetanus, it is too late when the symptoms of poisoning are established. The occurrence of limber neck in domestic fowl, if it develops after they have eaten refuse from the kitchen, should be sufficient reason for administering botulinus antitoxin to all persons who have eaten any of the suspected food. The presence and type of toxin in food can be determined in a few hours by injecting mice intraperitoneally. Some of the mice should be immunized with antitoxin type A and some with type B.

In view of the curare-like action of botulinus toxin, Edmunds and Long recommended the careful administration of physostigmin, but later found that it is not an antidote for botulism. By means of artificial respiration, dogs were kept alive four days. Artificial respiration, then, is clearly indicated. Bronfenbrenner and Weiss found that ether anesthesia delays the progress of intoxication and prolongs life. The same is true of other substances, especially morphin. Full physiological doses of morphin are therefore indicated until antitoxin can be obtained. Alcohol precipitates the toxin *in vitro* and in the stomach. Some cases recover without treatment.

Food Involved in Botulism.—It was originally thought that *Cl. botulinum* would grow only in sausage or meat, but it is now known that the presence of animal protein is not necessary for its growth. In Europe, the foods involved in outbreaks have been mainly meats, such as sausage and ham, but in this country other foods have been involved, such as string beans, cottage cheese, corn, asparagus salad, peas, spinach and ripe olives. Botulism has also been attributed to turkey, beef, chicken and fish.

Suspicion often falls upon the wrong food. The period of incubation is usually from eighteen to thirty-six hours, and the first symptom may be nausea and vomiting. Several meals will have been taken between the time of eating the toxic food and the onset of initial symptoms. The food vomited is naturally accused. This mistake was made in the Detroit outbreak, where corn was first accused because it was found in the vomitus, whereas the trouble really came from ripe olives eaten the day before.

Most cases of botulism are caused by food that has received some preliminary treatment, as smoking, canning or pickling, and not by fresh food. Home processed foods have been especially responsible, for the reason that they are often a day or more old when put up, and the temperature of processing is frequently not sufficient to kill the botulinus spores. All the olive cases were due to ripe olives, improperly processed, put up in glass, and insufficiently heated.

Van Ermengem found that the outbreak in Ellezelles was caused by eating ham which had been preserved in brine. Smoked as well as pickled ham has been responsible. In Russia fish has conveyed the poison. Madsen also isolated *Cl. botulinum* from poisonous fish. A most interesting outbreak oc-

curred in Darmstadt in 1904, caused by a salad prepared from home-canned white beans.

Sausages are a frequent source of botulism in Germany. The sausages readily become infected and present ideal anaërobic conditions for the growth of the organism, especially as they are rarely refrigerated and frequently contain old and contaminated scraps. The disease is, therefore, frequently called sausage poisoning. Certain sausages, as, for example, the blood sausage and the liver sausage prepared in Württemberg and Baden, are apt to be toxic. Venison and old roasts have also produced the intoxication.

Prevention.—The prevention of botulism consists in greater care and cleanliness in the preservation of nitrogenous foodstuffs. The bacillus will grow and produce toxin only in foods containing protein. There is no danger of botulism in fresh food. In all instances, the trouble comes from food that has been canned, preserved, pickled or processed in some way, such as pickled ham, home-processed beans, sausages, etc. Home-canned foods are often at fault. It is therefore important to teach safe methods of home canning, especially the importance of putting up only fresh and clean food, heated sufficiently to kill the spores—120° C. for ten minutes. This is the minimum temperature and time to kill the naked spores under laboratory conditions. In actual practice a large factor of safety is necessary in order to provide for penetration of the heat throughout the cans. Canned foods should be processed at a temperature sufficiently high and held there sufficiently long to render the contents of the can sterile, which will include the killing of botulinus spores. Sterile food is safe food. The cans should be tight.

The heat of cooking is sufficient to detoxicate the poison, but does not kill the spore. Food, therefore, that is allowed to stand around after cooking permits bacterial growth, unless kept in the ice box, with a temperature at or near freezing. The importance of proper refrigeration to prevent botulism, as well as other bacterial contamination, is evident.

Cooking is a safeguard, but it must be thorough and recent: thorough in order that the heat may penetrate throughout the mass and kill the toxin; and recent in order to prevent the reformation of the poison. If the food is kept at ordinary temperatures after cooking, botulinus and other organisms may grow and may cause trouble. Toxin may re-form in cooked food allowed to stand twenty-four hours at room temperature. Home-canned goods, or any other processed goods, should be again cooked just before serving. Food preserved in a brine of 8 per cent or greater is safe, for the toxin does not form in salt solution of this concentration. A syrup of at least 50 per cent sucrose has been found necessary to inhibit growth. A hydrogen ion concentration of 4.5 prevents the formation of this poison; therefore, botulism is unknown in acid fruits.

Reliance cannot be placed upon odor, taste or appearance to detect the toxin of botulism in food. It is true that in many instances a history is obtained that the responsible food looked spoiled, tasted wrong, or smelled tainted.

Experts can detect the peculiar butyric acid smell of cultures of botulinus, but we found in my laboratory that some strains produce little or no odor. A safe rule is not to eat food that is soft, mushy, and shows gas with a spoiled or putrefactive odor. Canned food that shows gas formation or other evidence of spoilage should not be eaten, especially if home processed. A very small amount of the toxin-containing food is sufficient to cause symptoms and death. Merely tasting the contents, or just "nibbling" the food has been fatal.

The occurrence of limber neck in domestic fowl, if it has developed after they have eaten refuse from the kitchen, may be an indication for the prophylactic administration of the botulinus antitoxin to all persons who have eaten the suspected food.

When a case of botulism occurs, other persons who may have eaten the suspected food should receive a prophylactic dose of botulinus antitoxin; both strains A and B should be used. The serum is of little avail after symptoms have begun.

DECOMPOSED FOODS

Decomposition is defined as natural decay. In this sense all organic substances, both animal and vegetable, living or dead, are decomposed, for decomposition and recomposition occur as a constant feature of life's processes. At the moment of death recomposition ceases, while decomposition continues. In one sense the hardest rocks decompose or disintegrate; bicarbonate of soda decomposes in the presence of an acid, and many substances decompose in the presence of oxygen, especially when heated. In other words, while decomposition is usually the result of bacterial activity in organic substances, it may also take place as the result of physical, chemical, or electrical agencies. The word "decomposition" is not used in this technical sense in the Pure Food and Drugs Act; there it has the meaning of the word used in ordinary, everyday parlance. Just where technical decomposition ceases and objectionable decomposition begins is often difficult to determine. Decomposition may be objectionable either to the senses or to health. We purposely permit many of our foods to decompose before they are used. Thus, meats hang three days or longer in order to render them more tender and to improve their flavors. During this time decomposition takes place with the production of acids. Some persons prefer meats when highly decomposed or gamy. The gourmand hangs his pheasant by the tail and waits until it drops off. Bread, cheese, butter, buttermilk, sauerkraut, vinegar, cider, and many other foods are products of decomposition. The line must, therefore, be drawn between decomposition that is objectionable and decomposition that is technical. It is difficult to draw the line at decomposition that is objectionable to the senses, for a cheese regarded as a delicacy by one person may be highly objectionable to another. The principal point, then, for consideration is the decomposition that is harmful to health.

Fermentation and Putrefaction.—The question is further complicated when we consider that there are very many kinds of decomposition. Two main groups are recognized: (1) fermentative decomposition, and (2) putrefactive decomposition.

Fermentation refers to the breaking down of carbohydrates with the formation of acids (lactic, acetic, butyric), alcohol, carbon dioxid, etc.

Putrefaction, from *putrere*, to be rotten, is literally a process of offensive decay. It is generally restricted to include only those processes of protein disintegration which give rise to foul-smelling products. For practical purposes, it consists of the decomposition of organic matter, usually protein in character, due to bacterial action.

Pasteur pointed out that putrefaction is essentially an anaërobic process. This has since been abundantly confirmed. Rettger insists that putrefaction is the work of certain obligate anaërobic, which are able to initiate and carry on the decomposition of native protein. *B. putrificus*, *B. edematis maligni*, and *B. anthracis symptomatici* are the best examples. *B. tetani* does not have a place in this group. *B. perfringens*, the gas bacillus of Welch, has only a limited if any proteolytic action. Certain aërobic, such as the proteus family, the colon group, and the subtilis group, also play a part, although they cannot initiate changes in protein.⁹¹

The end products of putrefaction are ammonia, nitrates, carbon dioxid, sulphurated hydrogen, methane, etc., all simple, stable, inorganic compounds which, in ordinary concentration, are not poisonous. It is then the intermediate cleavage products of putrefaction and the end products of fermentation that may be poisonous. The question of decomposition is still further complicated by the fact that there are very many different kinds of fermentation and of putrefaction. Each particular microorganism breaks down organic matter in a specific and limited sense. Ordinarily these processes result from a combination of bacterial action, in which aërobic and anaërobic organisms each play a part in turn. As a rule, putrefaction is restricted or modified in the presence of fermentation. In this sense carbohydrates protect nitrogenous matter.

Putrefactive Changes in Proteins.—To understand clearly the decomposition changes in proteins as a result of putrefaction it is necessary to have a clear conception of their composition and their simpler cleavage products.

The proteins are highly complex compounds of C, H, O, N and S, belonging for the most part to the colloids. The protein molecule is very large, complex and unstable, and probably constantly changing during life's processes. Some fifty or so natural proteins are known, occurring in both animals and plants, and they are classified according to their origin, solubility in solvents such as water, saline solutions and alcohol, reactions, coagulability on heating and other physical-chemical characters.

⁹¹ Bienstock, *Arch. Hyg.*, 1889, 31: 335; 1901, 34: 390; also Rettger, *J. Biol. Chem.*, 1906, 2: 71; 1908, 4: 5.

They are also distinguished from each other by biologic reactions, such as anaphylaxis and precipitation.

The great work of Emil Fischer and his pupils has confirmed and elaborated the theory originally propounded by Hofmeister, that the protein molecule is built up of a series of amino-acids forming a class of products which have been designated the polypeptids by Fischer. Such polypeptids form the essential part of the structure of the protein molecule, but it may contain other groups, such as phosphoric acid and possibly also carbohydrates and lipoids. The chemical structure of proteins is exceedingly perplexing and intricate. It is not a simple molecule, but a molecular complex with a nucleus and many side chains.

The amino-acids are definite chemical compounds in which the amino group, NH_2 , is substituted for a hydrogen atom of the carbon group nearest the acid radical. For example, acetic acid is a simple acid with the formula $\text{CH}_3\text{—COOH}$, while $\text{CH}_2\text{NH}_2\text{—COOH}$ is amino-acetic acid or glycocoll. The aromatic amino-acids are those in which amino-acids are united to the benzene ring. Tyrosin belongs to this group. The general formula of the mono-amino

acids may be stated as $\text{R—CH} \begin{array}{l} \nearrow \text{COOH} \\ \searrow \text{NH}_2 \end{array}$ where R may be of very simple

or very complicated structure; for example, simple chains as in leucin, members of the aromatic series as in tyrosin or tryptophan or sulphur-containing bodies. Lysin and arginin belong to the group of diamino-acids.

Under the influence of chemical agencies, such as acids or alkalies, physical agencies, such as superheated steam, the action of digestive or other ferments or the activities of bacteria, the protein molecule is decomposed and various cleavage products form. These substances may be classed as primary cleavage products, i. e., those which exist as radicals within the molecule; or as secondary products, i. e., those not existing preformed in the molecule but formed by transformation of the primary products.

“When the protein molecule is broken down in the laboratory by processes similar to those brought about by the digestive enzymes which occur in the alimentary canal, the essential change is due to what is called hydrolysis: that is, the molecule unites with the water and then breaks up into smaller molecules. The first cleavage products, which are called proteoses, retain many of the characters of the original protein; and the same is true, though to a less degree, of the peptones, which come next in order of formation. The peptones in their turn are decomposed into short linkages of amino-acids which are called polypeptids, and finally the individual amino-acids are obtained separated from each other” (Halliburton, 1916).

It is important to realize that whatever method is used to decompose the protein molecule the process goes through all these stages and approximately quantitatively as well as qualitatively. Different agencies, however, carry the process to different stages and the characteristic chemical products brought

about by putrefactive bacteria are due to their carrying the processes further and causing extensive secondary cleavage changes.

The conversions into proteoses, peptones and amino-acids are therefore changes which are common to all methods by which the protein molecule is decomposed, and chief interest centers upon the further changes in the amino-acids brought about by the putrefactive bacteria. Bacteria (and fungi) are peculiar in being able to break down the amino-acids into bases and acids which, in general, have not been demonstrated as products of the metabolism of animals and the higher plants.

As long ago as 1902, Czapek, and also Emmerling, pointed out that the amino-acids furnish bacteria with abundant and available nutritive material. The amino-acids are non-toxic compounds. They are listed and discussed on page 607.

The secondary decomposition products which result include definite chemicals, such as indol, skatol, skatolcarboxylic acid, skatolacetic acid, phenylpropionic acid, phenylacetic acid, *p*-resol and phenol. In addition a number of simple substances, such as ammonia, methane, carbon dioxid, sulphurated hydrogen, hydrogen, etc., are formed as end-products. The nitrogen in protein finally appears as nitrates, which is the end of this phase of the nitrogen cycle (page 911).

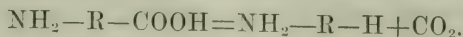
The precise chemical compounds which will be formed depend upon a number of factors, such as the character of the bacteria concerned, the conditions of growth (especially as regards the presence or absence of oxygen), the available sources of nutriment other than the amino-acids, the temperature and the stage of the process.

Hopkins and Cole (1903), for example, studied the changes produced in chemically pure tryptophan by putrefaction. They obtained indol, skatol, and skatolcarbonic acid by the action of aërobic bacteria and skatolacetic acid with anaërobic organisms, in this way showing that the tryptophan radical is the precursor of these substances in putrefaction. In the same way, tyrosin is the precursor of phenol, paracresol, paraoxyphenylacetic acid and other bodies.

Most, if not all, of the sulphur in the protein molecule is contained in the amino-acid cystin and the offensive sulphur-containing bodies, such as hydrogen sulphid, methyl mercaptan (CH_3SH) and ethyl mercaptan, produced during putrefaction are due to the breaking down of this amino-acid.

In addition to these numerous products a very definite group of substances, chemically of the nature of amins, are formed in the later stages of putrefaction, and to these, owing to the toxicity possessed by some of them, an exaggerated importance has been attached as a cause of food poisoning.

A very characteristic action of putrefactive bacteria generally is their power to split off carbon dioxid from the carboxyl (COOH) group of the amino-acids with the production of amins according to the following equation:



In this way a whole series of chemicals is formed which include the ptomains of Selmi and Brieger and other bases, some of which were claimed to exert a poisonous action on man and animals. This decarboxylation of amino-acids seems to be due to a general reaction of a good many putrefactive organisms.

As examples of such changes it may be mentioned that diaminovaleric acid is converted into putrescin, diaminocaproic acid (lysin) into cadaverin, and tyrosin into tyramin. In the same way the poisonous and probably important compound β -imidazoleethylamin (histamin) is the amin of histidin. The decarboxylation of amino-acids is not necessarily accompanied by a putrefactive odor or other obvious signs of bacterial action.

For a further consideration of proteins with reference to nutrition, see page 607.

“Ptomain” Poisoning.—Ptomains are secondary cleavage products of protein putrefaction. Vaughan defines a ptomain as an organic chemical compound, basic in character, and formed by the action of bacteria on nitrogenous matter. On account of their basic properties ptomains give some of the reactions of the vegetable alkaloids and have, therefore, been miscalled putrefactive alkaloids. They are sometimes called “animal” alkaloids, but this too is a misnomer, for they also are formed in the putrefaction of vegetable protein.

The term “leukomain” is used to cover the same or similar basic substances which result from tissue metabolism within the body; that is, leukomains are produced in the living body, ptomains in dead organic matter.

The word “ptomain” was coined by the Italian toxicologist, Selmi, in 1870, from *ptoma*, a corpse. He used the word to describe basic poisonous products analogous to the familiar alkaloids of plant origin. The further exploitation of the expressive word “ptomain” was largely the outcome of studies by Gautier in 1872, who also introduced the term “leukomains.”

Chemically, ptomains are ammonia substitution compounds; two-thirds of them contain only carbon, hydrogen, and nitrogen. Those having oxygen in their composition are the more poisonous. Most ptomains are inert or are no more poisonous than the corresponding ammonia salts. In composition they show a predominance of the amin radical (NH_2). Of the bases containing oxygen, most of them are trimethylamins [$(\text{CH}_3)_3\text{N}$]. It was Brieger who pointed out that a certain quantity of oxygen is necessary for the formation of poisonous bases. These poisonous bases appear about the seventh day of putrefaction and then disappear.

It is important to remember that ptomains, in sharp contradistinction to toxins, are non-specific, that is, they are not the products of intracellular metabolism characteristic of the microorganisms which produce them. They are merely decomposition products of the protein molecule and are elaborated by all bacteria that are capable of producing this degree of protein cleavage when grown on suitable nutrient media and under favorable conditions of growth. They may be produced by bacteria which possess no pathogenic

power, while on the other hand highly pathogenic bacteria which are not active in attacking protein may produce little or no ptomains.

The term "ptomain poisoning" is a misnomer. A study of this subject for over five years has convinced me that there is no such thing. Savage states that the term "ptomain poisoning" is clearly incorrect, and Jordan states that "ptomain poisoning" is a refuge from etiologic uncertainty. Vaughan agrees with Jordan and Savage that the term "ptomain poisoning" is incorrect and should not be applied to food poisoning. Novy states that "the rather popular expression 'ptomain poisoning' is a survival of the period when it was believed that bacteria produced their injurious effects by means of basic or alkaloid-like products. Long ago the importance of ptomains disappeared, due in the first place to the discovery of toxins, and in the second place to the fact that these substances are not secondary products of protein cleavage." Chapin states that "ptomain poisoning" is a good term to forget. Vaughan, in 1895, detected in poisonous cheese an active agent to which he gave the name tyrotoxinon. However, he afterwards admitted that this is not the substance most commonly found in poisonous cheese, although the names tyrotoxinon and ptomain poisoning remain in popular parlance.

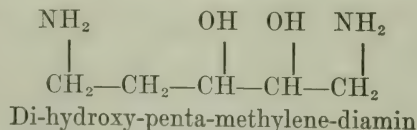
Ptomains include substances which are chemically very different. The classification is unscientific and is gradually being abandoned by chemists. A few of them are physiologically active, even poisonous when injected parenterally. None of them are actively poisonous when taken by the mouth, and even the active ones do not produce nausea, vomiting, colic and diarrhea.

It is not so much *decomposed* as *infected* food that may be dangerous.

Owing to the importance which is still attached by some to these substances and their historical interest, particulars of a few are given.⁹²

Methylamin, CH_3NH_2 .—This is the simplest amin and has been obtained from decomposing herring, haddock and other fish. It does not possess any toxic action and very similar remarks apply to di- and tri-methylamin and to ethylamin.

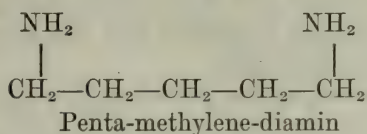
Sepsin.—The best known poison which has been isolated in an approximately pure state from decomposing nitrogenous material is sepsin. Much work was done upon this substance by Schmiedeberg and later by Faust, who obtained sepsin in a purified state in sufficient quantities carefully to study its action and composition. Faust obtained the crystals from putrefied yeast and blood; 25 milligrams of the sulphate introduced intravenously will kill a large dog in two hours. The formula is:



Sepsin is very unstable. It is rendered inactive at 60° C. for a short time,

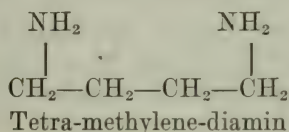
⁹² V. C. Vaughan and F. G. Novy, *Cellular Toxins*, 1903.

and is readily converted into cadaverin or pentamethylenediamin. The chemical structure of cadaverin is:



Cadaverin is one of the best known of the ptomain group. Its presence indicates that the putrefactive process at one time contained sepsin which, by reduction, has been changed into cadaverin.

Putrescin is another diamin, which almost invariably occurs together with cadaverin, to which it is closely related. It was first described by Brieger in 1885, and has been obtained from putrefying internal human organs, herring, mussels, etc. It is recognizable on the fourth day of putrefaction, and appreciable quantities appear by the eleventh day. It is still present after two or three weeks. Baumann in 1888 showed the rational formula to be:



Putrescin is a homolog of cadaverin and appears in putrefaction before that substance. Van Slyke and Hart (1903) found a little putrescin in ordinary Cheddar cheese. Once formed putrescin and cadaverin appear to be very resistant to bacterial action.

Putrescin and cadaverin are of interest because they have been found in the intestine, derived from the putrefactive decomposition of proteins, and sometimes in the urine in cystinuria. They are said to have some physiological properties, setting up, according to Behring, poisonous symptoms in mice, rabbits and guinea-pigs. Udránszky and Beauman, however, failed to obtain any evidence of intestinal irritation when dogs were fed with enormous doses of cadaverin.

The cholin group of ptomains includes cholin, neurin, muscarin and betain and is of more interest.

Cholin is a normal constituent of every cell, forming the nitrogenous portion of the lecithin molecule. It is only very moderately toxic, but the closely related neurin into which it may be transformed is highly poisonous. It has been suggested that one form of food intoxication is due to the cholin, obtained from the lecithin in the food, being converted in the gastro-intestinal tract into neurin. While cholin in itself is not very toxic, Hunt has shown that acetylcholin is one hundred thousand times more poisonous. Cholin is a base widely distributed in nature; it is found in the yolk of eggs, in bile, brain substance, fat, seeds, and other substances. It can also be prepared from pure

lecithin, which is a lipoid normally present in brain substance, yolk of eggs, and perhaps all cells. The lecithin may be readily decomposed by bacterial action perhaps to cholin and cholin salts. While acetylcholin has never been demonstrated in food, it is possible that this or similar poisons may be produced in decomposing foodstuffs.

Brieger obtained *neurin* in the putrefaction products of horse, beef and human blood after five to six days' action in summer.

Muscarin was obtained, accompanied by cholin, by Schmiedeberg and Koppe from poisonous mushrooms.

Both *neurin* and *muscarin* are extremely poisonous and very similar in their action. Subcutaneous injection of but 1 to 3 milligrams of muscarin in man produces salivation, rapid pulse, reddening of the face, weakness, depression, profuse sweating, vomiting and diarrhea. Neurin acts very similarly. "The toxicity of these substances is so great that not a large amount would need to be formed by oxidation of cholin to produce severe symptoms, although it is not known that this occurs actually in the body. When introduced by the mouth, the lethal dose of neurin is ten times as great as when injected subcutaneously, indicating that chemical changes in the gastro-intestinal tract offer some protection against intoxication by these substances when taken in tainted food. Cholin, although by no means so poisonous as neurin, has a similar action when administered in sufficiently large doses. According to Brieger it is about one-tenth to one-twentieth as toxic as neurin" (Wells, *Chemical Pathology*).

Mytilotoxin is chiefly of interest in that it is said to be the specific poison in connection with mussel poisoning and was obtained by Brieger in 1885 from toxic mussels. He was, however, unable to obtain it from ordinary mussels which were allowed to putrefy for sixteen days. According to Brieger it produces all the characteristic effects seen in mussel poisoning. Its connection with mussel poisoning is considered on page 768.

The more the question of ptomaines is studied the less do they appear concerned in cases of food poisons. It is now clear that most, if not all, cases of so-called ptomain poisoning are nothing more nor less than acute infections with *B. enteritidis*, *B. ærtrycke*, *B. cholerae suis*, and other micro-organisms belonging to this group.

ADULTERATION OF FOOD

Adulteration of food consists of a large number of practices, some of which are fraudulent, others technical in nature. Some forms of adulteration are injurious to health, but for the most part they have an economic rather than a sanitary significance. Foods may be adulterated in a variety of ways: by the removal of nutritive substances; by the addition of injurious substances; by the fraudulent substitution of cheaper articles; by misbranding; or by the sale of food that is filthy, decomposed or putrid.

Prior to the passage of the Pure Food and Drugs Act in 1906 a very large

percentage of the food sold in the United States was found to be "adulterated" in one way or another. In Massachusetts, the State Board of Health began to examine foods for adulteration in 1883. It was then found that between 60 and 70 per cent of all foods examined were adulterated. As a result of official surveillance the percentages fell in a few years to approximately 15 per cent and have remained between 10 and 20 per cent since. This does not mean that from 10 to 20 per cent of all foods found on the market are adulterated, for, to a great extent, samples are collected from suspicious sources, so that the ratio of adulteration of food analyzed in the laboratory is higher than that of the same foods sold on the market. At the agricultural experiment station in Kentucky it was found that 40 per cent of 727 samples were adulterated; at the Connecticut Agricultural Experiment Station 41.5 per cent of 574 samples of spices and over 25 per cent of coffee samples were found to be adulterated (1899). The situation has greatly improved as a result of the Pure Food Law.

Examples.—Among the common adulterations may be mentioned the following: cottonseed oil is sold as olive oil; honey may contain glucose; cocoa and chocolate are frequently mixed with both starch and sugar; coffee was formerly extensively adulterated with caramel, pea-meal, chickory, and saccharose extracts; lard is mixed with cheaper fats or cottonseed oil; saccharin is substituted for cane sugar; cereals give bulk and weight to sausages; flour or turmeric or corn meal to mustard. Water is added to milk, or the cream is subtracted. Oleomargarin is sold as butter; distilled and colored vinegar is sold as cider vinegar; ground spices are adulterated with cocoanut shells, rice, flour, and ashes; water, sugar, and tartaric acid is sold as lemonade; wines and liquors are sometimes adulterated with alum, baryta, caustic lime, salicylic acid, wood alcohol, and hematoxylin. Terra alba, kaolin, and various pigments are sometimes added to candies; gum drops are largely made with petroleum paraffin products; much of the maple sugar formerly sold was made from brown sugar.

Definitions.—A food is considered adulterated in accordance with the Food and Drugs Act of June 30, 1906:

1. *Mixing.*—"If any substance has been mixed and packed with it so as to reduce or lower or injuriously affect its quality or strength." This is the simplest form of adulteration, and a good example is the addition of water to milk. Cocoa shells are sometimes mixed with cocoa or chocolate. Glucose and caramel are added to maple syrup; talc to flour.

2. *Substitution.*—"If any substance has been substituted wholly or in part for the article." As illustrations we have the substitution of cottonseed oil or corn oil for olive oil; glucose or saccharin for sugar; cereals, which cost much less than meat, in sausage. Apple cores and parings are frequently used as a substitute for currants and other fruits in jellies.

Saccharin or ortho-benzo-sulphamid, $\text{C}_6\text{H}_4 < \begin{matrix} \text{CO} \\ \text{SO}_2 \end{matrix} > \text{NH}_2$, is made from

toluene. It is several hundred times sweeter than sugar and comparatively cheap. It has, therefore, been used as a substitute for sugar as a sweetening agent in the inferior qualities of ginger ale and other soft drinks, and to some extent in canned corn, peas, etc., as well as in candies and other articles. Saccharin is a chemical obtained from coal tar and is without food value; it is not entirely harmless. The Referee Board reports that "the continued use of saccharin for a long time in quantities over 0.3 of a gram per day is liable to impair digestion; and the addition of saccharin as a substitute for cane sugar reduces the food value of the sweetened product and hence lowers its quality." Saccharin-containing foods are therefore regarded as adulterated within the meaning of the Food and Drugs Act. The substitution of cheap chemicals for high-priced natural flavoring extracts, the substitution of acetic acid or even mineral acids for genuine vinegar, the paraffin polishing of rice, and similar devices are nothing but common frauds, which may in some cases also be injurious to health.

3. *Abstraction of Valuable Constituents*.—"If any valuable constituent of the article has been wholly or in part abstracted." Skimming milk is a good illustration of this part of the law, or the abstraction of cocoa butter from chocolate. There is, however, no objection to abstracting valuable or nutritive substances provided the label properly announces the facts; thus, skimmed milk or cocoa are legitimate foods. So also the caffein may be taken out of coffee and the product sold as caffein-free coffee. The essential oils are sometimes extracted from cloves or other spices, which are subsequently ground and used as an adulterant with unextracted spice. Abstraction without honest labeling is a fraudulent practice.

4. *Concealing Inferiority*.—"If it is mixed, colored, powdered, coated, or stained in any manner whereby damage or inferiority is concealed." This is a very frequent form of adulteration, and, as a rule, is undesirable and sometimes injurious. Substances used to color foods are usually considered in three classes: (1) mineral dyes, (2) vegetable dyes, (3) anilin or coal-tar dyes. The principal *mineral dyes* are: copper sulphate, oxid of iron, and potassium nitrate. Copper sulphate is used to give a green color to peas, pickles, and similar foods. The copper probably unites with the albuminous matter to form new compounds which have a bright green sickly color. The oxid of iron and also sulphites are used upon meat to give it a red color; potassium nitrate will also give a bright red color to pickled or corned meat. Many *vegetable dyes* are used, such as annatto (the juice of the *Bixa orellana*, a South American tree), which is used to color butter. Carrot juice is also used; turmeric in mustard; and logwood in wines. The *coal-tar dyes* have largely replaced the vegetable and mineral pigments in foods, on account of their brilliant color and cheapness. They are used in sausages, confectionary, jellies and jams, meats, flavoring extracts, etc. The permitted dyes are harmless to health.

The artificial coloring of food is a false standard and serves no useful purpose. When the coloring matter is used to conceal damage or inferiority

the practice is indefensible, as when spoiled meats are made to look bright red and fresh, or when oleomargarin is colored in order to imitate butter and sold as such. Flour may be bleached with nitrogen peroxid. The flour absorbs this poisonous gas as a sponge absorbs water and instantly becomes white. Processes of this kind should be regarded as a common fraud, for the flour is not improved in any way except in appearance, which is, after all, a deception. The City of New York requires flour that has been bleached with oxid of nitrogen, or nitrous oxid, or nitrates, or chlorin to be legibly and conspicuously labeled "Bleached with ——" Fruits are bleached by exposure to sulphur fumes, which leaves objectionable sulphur compounds. Candies and chocolate are coated with gum benzoin or shellac. Concealing inferiority may be both an economic fraud and a health menace.

5. *Addition of Poisons.*—"If it contains any poisonous or other added deleterious ingredient which may render such article injurious to health." This section of the law is intended to include adulterants, such as formaldehyd, sulphites, arsenic, hydrofluoric acid, lead, salicylic acid, borax and boracic acid, as well as any other injurious substance. Most of the storm center of the opposition to the Pure Food Law centered around this paragraph, owing to the difficulty of deciding in certain instances whether small amounts of benzoic acid or benzoates, boric acid or borates, are injurious to health or not. This question is discussed more in detail under chemical preservatives.

6. *Composition.*—"If it consists in whole or in part of a filthy, decomposed, or putrid animal or vegetable substance or any portion of an animal unfit for food, whether manufactured or not, or if it is the product of a diseased animal or one that has died otherwise than by slaughter." Examples: oysters contaminated with sewage; eggs known as "rots and spots"; animals which have died from disease or by accident; figs containing an excessive quantity of worms and worm excrement. This paragraph of the law has caused much discussion, especially the meaning of the word "decomposed." This question is considered more in detail under the paragraph Decomposed Foods.

7. *Misbranding.*—The term "misbranding" is specifically defined in the Pure Food and Drugs Act and provides for all possible conditions of fraud, mislabeling, imitation, substitution, and other forms of deception. Misbranding is regarded as a form of adulteration under the Pure Food and Drugs Act. The practices of misbranding under any circumstances are so evidently fraudulent or dishonest that they cannot be justified on any score and are wholly condemned. It is true that many instances of misbranding do not directly affect health, except in so far as they deceive the consumer; that is, he is purchasing at a high price an article which contains less nutritive value than claimed for it. An honest label which correctly states the character, origin, amount, and the constituent parts of an article is as much a desideratum in food products as it is in commercial articles of all kinds. Honest labeling is the heart and soul of the pure food movement.

PRESERVATION OF FOODS

The preservation of meat, milk, vegetables, and other perishable foods is one of the important questions we have to deal with in hygiene. The proper preservation of foodstuffs involves not only the art of keeping them "fresh" and wholesome, but also keeping them so that they will not lose their nutritive value. Finally, foodstuffs must be preserved so that they will not acquire injurious properties. The preservatives ordinarily in use are: cold, drying, salting, smoking, canning, preserving, and chemical treatment. The preservation of food by cold and sterilization by heat have the advantage over other processes in that they cause comparatively little alteration in the food.

Practically all these methods have long been in use. The only modern innovation in the preservation of foods is in the perfection of the old processes, based upon our knowledge of antiseptics and germicides. Heat and cold represent old family methods which have been extended and improved in the modern canning and cold storage industries. The drying of fruits, fish, and meats is a practice of very ancient origin. The use of salt doubtless antedates all historical records. Sugar either alone or with acetic acid in the form of vinegar and with various spices is an old contrivance and well known everywhere. The application of creosote obtained crudely from the smoke of incompletely burned wood is the ancient forerunner of some of the modern packing processes.

Concerning the value and legitimacy of these old family methods there is comparatively little difference of opinion; salt meat is not as good as the fresh article; dried apples do not make the best apple pie; chipped beef is not an adequate substitute for a fresh steak. However, it is absolutely necessary to preserve food in some way in order to tide over the winter or the dry seasons, to furnish food to people living and working in desert and arid regions, and to feed the hordes of people massed together in great cities. It would be impossible to maintain the large population of a modern metropolis if it were dependent upon a daily supply of fresh food materials.

The art of preserving foods successfully depends upon the science of bacteriology. It is now possible to preserve most foods for long periods without materially injuring their nutritive value or seriously interfering with their appearance and taste.

The chief mischief was the threat of a blind use of chemical germicides without regard for their effect upon health. The simplest and cheapest way to preserve food is by adding one of these chemicals, and the method was, therefore, seized upon by alert men whose chief interest was of the pecuniary kind. The question was to find the smallest percentage of a chemical which would prevent the decay of some particular food product, trusting to luck that the preservative used might prove harmless to the consumer. Often these chemicals were added with a liberal hand; further, it was soon found that chemical preservatives could be used to preserve food products for the market

from materials already so decayed as to be unsalable in their original condition.

The National Pure Food and Drugs Act of 1906 was passed largely to meet this situation. This law considers any food which contains "any added poisonous or other deleterious ingredient which may render such article injurious to health" as adulterated. To Harvey W. Wiley belongs the credit of inducing Congress to pass this legislation against opposition and for an aggressive administration that proved useful in bringing the whole question prominently before the public.

Cold.—Cold prevents or hinders bacterial activity. Low temperatures in themselves do not kill bacteria, but prevent the growth and multiplication of most of them. In other words, cold is an antiseptic rather than a germicide. The effect varies with the temperature. Many plants and even frogs may survive freezing; in fact, some molds will grow at the freezing point. Furthermore, cold delays or checks the action of enzymes, which cause ripening. The rate of chemical and biological activity is a function of temperature.

Burdon-Sanderson, in 1871, was the first to show that freezing does not kill bacteria. Von Frisch demonstrated that subjecting a putrefying solution to a temperature of -87° C. for some hours did not effect sterilization. Leidy, in 1848, showed that water derived from melted ice contained not only living infusoria, but also rotifers and worms. Pictet and Young found that anthrax and symptomatic anthrax cultures were not killed after an exposure of 108 hours to -70° C. Later Macfadyen proved that the temperature of liquid air does not kill bacteria; he subjected cultures to temperatures of -315° F. Ehrlich has shown that cancer cells kept cold will live and remain virulent for at least two years.

While no microörganism pathogenic for man will grow and multiply at the low temperatures of the refrigerator, there are a number of saprophytic bacteria and molds that develop abundantly at temperatures as low as 0° C. Milk, meat, eggs, and other products kept in cold storage, but below the freezing point, may show a notable increase in the number of bacteria. A number of tests made in my laboratory showed that in the case of milk these low temperature microörganisms belong mainly to the putrefying and proteolytic group. They produce an alkaline reaction in the milk and a bitter taste. There is no evidence that poisonous products are formed under these conditions.

For the most part pathogenic bacteria withstand freezing temperatures. They, however, suffer a quantitative reduction when frozen (see Ice and the effects of freezing upon bacteria, page 1073). The element of time here plays an important rôle, as most of the frailer pathogenic bacteria eventually die. From a sanitary standpoint the protection afforded by refrigeration is partial; it cannot be depended upon to disinfect. In fact, it acts as a preservative for some viruses.

Some *animal parasites* die in cold storage; some, however, survive. Thus, *Trichinæ* die at or below 5° F. in less than 20 days. If the temperature is maintained at 15° F. or below, the larvæ of *Tania saginata* (the beef tape-

worm) are killed within six days, but this cannot be depended upon for *T. solium* (the pork tapeworm).

Temperature for Refrigeration.—The best temperature at which foodstuffs may be kept must be determined for each case. Some substances, such as meat and poultry, are better preserved when actually frozen; others, such as shell-eggs, milk, potatoes, oranges, etc., are materially injured by freezing. For temporary preservation fish are kept at refrigerator temperatures. If preserved in cold storage, they may be frozen, then dipped in water and refrozen in order to completely encase them in ice. They are then stored at a temperature of -16° C. The coating of ice, which is renewed as occasion requires, prevents loss of water due to surface evaporation. Under these circumstances fish may be preserved two years without appreciable change in appearance, flavor or nutritive value.⁹³

Household Ice Box.—In any event, the temperature of the ice box should not rise above 7° C. At this temperature bacterial growth does not entirely cease, although very markedly hindered. Few household refrigerators reach this temperature or maintain it for any length of time—either through faulty construction or on account of insufficient ice. Often the ice box is placed in a sunny corner, or, for convenience, near the kitchen stove. The doors of the ice chest frequently do not fit well, which results in needless waste and imperfect refrigeration. A study of household refrigerators discloses the fact that the temperature is often 15° C. and higher. Such conditions make good incubators, favoring bacterial growth. *B. enteriditis* will grow at 10° C. The necessity for scrupulous cleanliness, aëration, and dryness in all refrigerating devices needs only be mentioned.

Cold Storage.—In ordinary refrigerating plants moisture condenses on the surface of the objects exposed. In the case of meat this moisture dissolves some of the proteins, extractives, and salts, and makes a perfect culture medium for bacteria and molds. It is, therefore, better to hang meats in a current of dry, clean air, in order to desiccate the surface, before they are placed in the refrigerator. The dried surface delays the inward growth of the inevitable bacterial contamination upon the surface.

Articles of food may be kept in a satisfactory condition in cold storage for a very long time. The time varies with the article and its condition when placed in storage, also with the temperature and other factors. A striking illustration of the great preserving power of low temperatures occurred several years ago in northern Siberia. In consequence of a great landslide on the banks of the Kolyma, the head of a mammoth became exposed and was so well preserved that even the fleshy trunk remained. It is said that famished wolves and half-starved natives began to eat of the flesh. The Russian government sent Hertz to rescue what remained. The mammoth had remained in cold storage perhaps thousands of years. Some of the soft parts were sent to the Museum at Leningrad. This must not be taken as justification of pro-

⁹³ C. S. Smith, *Biochem. Bull.*, 1913, 3: 54.

longed storage or the "cornering" of foods for economic gain in mammoth cold storage warehouses. While meat, poultry, eggs, and vegetables may be kept in a satisfactory condition for months and transported over seas, cold storage should not be unduly prolonged. In any case, the consumer is entitled to know whether the article is fresh or stored, and the time it has been in cold storage. These facts should be stated upon the label or stamp.

The question of cold storage poultry was investigated by the Massachusetts State Board of Health, and the conclusion was reached that it made practically no difference whether the fowl were drawn or not, but that they must be perfectly fresh when placed in cold storage. Poultry kept below 0° C. shows little or very slow change. It was found that cold storage fowl are even less contaminated with bacteria than freshly killed birds that have hung for a few days. However, the cold storage foods, when removed from the refrigerator, decompose more quickly than the fresh.

Contrary to what might be expected, drawn poultry decomposes more rapidly after removal from cold storage than undrawn. This is because in the process of drawing the intestines are broken and the carcass is exposed to intestinal and other bacteria. If the entire alimentary canal, esophagus, crop, gizzard, and intestines are removed intact, and with bacteriologic care to prevent contamination, the bird is practically safe from putrefaction.

Summary.—From a sanitary standpoint, then, refrigeration is one of the best methods of preserving foodstuffs. The advantages of cold as a preservative are that it neither adds any constituent to the food nor takes away any constituent from the food. Cold imparts no new taste, nor does it seriously alter the natural flavor. It does not diminish its digestibility nor cause a loss of nutritive value. It has slight if any effect on most of the vitamins. The material is left in approximately its original condition. Cold may, therefore, be regarded as one of the simplest and best antiseptics we have for the preservation of foods. It is now almost universally applied to prevent decomposition and decay. The housewife uses it to keep food in cold cellars, deep wells, and the like. During the last fifty years the use of ice for the purpose of refrigeration has become commonplace. Fresh and wholesome food may now be transported to and from the tropics, and the sustenance of large communities in insular and arid regions is made possible and pleasurable through the preserving use of cold.

Drying.—Drying, desiccation, or evaporation is a favorite and primitive method of preserving meats, and in recent years it has been extended to include vegetables, milk, eggs, and almost every kind of watery food. Dryness furnishes ideal antiseptic conditions. Microorganisms must have moisture to grow and multiply. Most pathogenic bacteria soon die when dried, hence the process has a decided sanitary advantage. Further, dried fruits, vegetables or meats are rarely eaten raw, and the cooking is a further sanitary safeguard.

Drying has two principal advantages which simplify the problem of handling and distribution: (1) It improves the keeping qualities of the food, and (2) it reduces the weight.

The effectiveness of drying as a food preservative depends upon the thoroughness with which the process is carried out. It is not so well adapted to meats as to vegetables and fruits. Dried meats lose their natural flavor, which may be replaced with others less real. The keeping qualities of dried food depend upon the degree of dehydration.

Effect on Vitamins.—Drying has little or no influence upon some of the vitamins. It certainly does not diminish the activity of vitamin B which protects against beriberi. Dried leaves, such as celery tops and alfalfa, are still good sources of vitamin A, even when dried in the sun, or by artificial heat in a current of air after preliminary treatment with steam. Dried milk is also an efficient source of vitamin A. Drying affects chiefly vitamin C. A diet of dried foods has long been associated with scurvy. Even Captain Cook knew that this deficiency disease could be prevented by the use of fresh food. Studies have shown that drying alone does not injure this vitamin so much as oxidation, but as food is usually exposed to air in the process of drying, vitamin C is injured. Milk may be dried so that it contains about one-half its original antiscorbutic property.

Changes in Dried Foods.—The changes which take place in dried foods are more than simple loss of water. There may be physical changes which are not reversible. Some of the constituents become insoluble. Enzymes and oxidation continue their activity, although slowly. Milk and eggs under ordinary conditions of desiccation turn rancid. The effect on the vitamins has already been discussed.

Methods.—The methods of drying have been greatly improved and widely extended, owing to the demand for dried foods during the World War. The old-fashioned sun drying has been replaced by vacuum drying and scientific methods. In the tunnel method, the food is placed on racks in tunnels or chambers through which the current of heated air is passed. In the kiln method, the food is placed on the perforated floor and heated from below by means of a furnace. Each food must be processed by a method best suited to its peculiarities.

Dried Meat.—The successful drying of meat requires a suitable temperature and the absence of air. The temperature must be kept below the point where the proteins coagulate and the fats melt. If the drying is too slow, the meat will spoil in the process; if too rapid, a hard protecting layer forms on the surface. Meat is therefore best dried in a vacuum in cut pieces at a suitable temperature. Dehydration by the vacuum method is, in fact, generally applicable to many different foods.

In the dry climates of South America and on our western plains, meat is cut into thin strips, suspended in the air, and exposed to direct sunlight. In a short time the moisture disappears and the hard, dry pieces keep indefinitely, or as long as they are kept dry. The meat retains a fair degree of palatability and practically all of its nutrient properties. This is known as jerked beef.

Dried beef is also prepared by first treating the meat with condiments and

then drying it artificially. Chipped beef or dried beef is prepared in this manner, except that the meats are often smoked as well as salted and desiccated, so that in their preparation more than one method of preservation is employed.

Powdered meats are prepared by complete desiccation, and such products are found upon the market as a finely ground powder. Meat powders are made not alone from fresh meats in their natural state, but are also prepared after more or less artificial digestion.

Dried Fruits.—Dried apples are taken as a type of dried fruits and vegetables. The apples may be dried naturally by cutting them into convenient sizes and exposing them to the action of the sun. This is more a domestic than a commercial industry. When apples are dried by this simple process they darken and become unattractive in appearance, which is due to the oxidizing action of the enzymes when exposed to the air. When properly prepared the dried apple has its moisture content reduced to 24 per cent. Dried apples containing 26 to 27 per cent are apt to ferment.

In order to prevent the darkening of the surface during the long exposure necessary to secure the proper degree of evaporation, apples are usually subjected to the fumes of burning sulphur. The sulphur dioxide acts as a bleaching agent and the sulphurous and sulphuric acids retained in the apple act as preservatives. Apples treated with sulphur fumes are less likely to decay or become infected with molds than a similar product not exposed to sulphur fumes. The process is objected to from the standpoint of health, for the reason that the sulphurous acids and sulphites are admittedly injurious. The United States Department of Agriculture found that approximately half of the evaporated fruits purchased on the open market had been treated with sulphur fumes. In order to obtain a satisfactory dried product it is of some importance that the fruits be selected, so as to reject all imperfect, rotten, or infected specimens.

Evaporated apples is a term applied to apples dried artificially instead of being exposed to the sun's heat. The process is rapid and satisfactory, and has no sanitary objections.

Dried Milk.—Milk must be dried quickly in order that it will not spoil during the process, and the temperature must not be high enough to coagulate the lactalbumin, otherwise the addition of water would not restore the milk to its former homogeneous state. Milk may be dried in a very thin film on heated and revolving metal drums or on belts in a current of dry, warm air; or *in vacuo*. In this way the milk can be reduced to a dry state in a very short time and without reaching a temperature sufficiently high to materially alter its quality. A favorite method of drying milk consists in atomizing it under pressure and projecting it into a warm chamber, the temperature of which is so regulated that the fine particles are completely deprived of their water before they reach the bottom of the cabinet. The milk is thus reduced almost at once to a fine powder. Dried milk when mixed with water is practically restored to its original condition, except that the solubility of some of the constituents is interfered with. It is very difficult to preserve milk powder

against rancidity. It keeps well if it contains less than 2.5 per cent water, but even then the results may be disappointing in handling whole milk.

Dried milk should, of course, be made from milk derived from healthy cows handled under sanitary conditions and free from infection. The milk should be pasteurized before it is reduced to a powder. Powdered milk is finding an increasing and legitimate field of usefulness for cooking, household purposes, as a beverage for adults and even for infant feeding.

Dried milk seems to retain all the essential qualities of the fresh product, except that it has lost some of its antiscorbutic property, and is less attractive. Since practically 88 per cent of milk is water, there is a decided gain so far as handling, transportation and keeping are concerned. For partially dried milk, such as condensed and evaporated products, see section on Milk, p. 727.

Dried Eggs.—Eggs are broken out, mixed and dried by spreading the mass in a thin film on a revolving drum, or by the spray method, similar to that of drying milk. Egg powder keeps well and retains practically all the nutritive value of the original egg.

Salting and Pickling.—The preservation of meat with brine or common salt is one of the oldest processes known. The brine should contain from 18 to 25 per cent of salt. For red meats a little potassium nitrate is often added; this salt has slight antiseptic properties, but brings out the red color. Haldane has shown that nitrite is formed from the nitrate, and that some nitrous-oxid hemoglobin is formed, which gives a bright red color to the meat. In the processes of salting some of the meat protein, bases, and extractives are dissolved out and the fibers become hardened; the nutritive value and digestibility, therefore, are somewhat diminished. The brine should never be less than 8 per. cent, for *Cl. botulinum* will not grow and produce its toxin in this concentration.

The preserving action of salt depends largely upon dehydration, but also upon the direct effect of the chlorin ion, removal of oxygen, and on interference with rapid action of proteolytic enzymes.

Pickling includes preservation of food in brine, vinegar, weak acids, and the like. These substances have antiseptic and also feeble germicidal properties, depending upon their concentration.

Pickled meats are prepared by soaking meat, especially pork, in a brine made of common salt, though other substances, such as sugar, vinegar, and spices, are often added. Sulphite of soda, boric acid, borax and benzoic acid, and other chemical preservatives formerly used are no longer permitted by the United States Bureau of Animal Industry^{93a} (p. 750). With proper methods chemical antiseptics are not necessary. The vinegar which is employed, or acetic acid, may be injected into the carcass before it is cut up. When the arteries are filled with vinegar in this way it rapidly permeates to all parts of the meat and acts as an excellent and unobjectionable preservative in cases where an acid taste is desired. It is stated that carcasses which have

^{93a} Order No. 211, Bur. An. Ind., U. S. Dept. Agric.

been injected with vinegar are easily preserved and require far less salt and other condimental substances than when not so treated. The process has no sanitary objections.

Trichinae die after a prolonged period of pickling. *Cysticerci* die a natural death in twenty-one days following the death of the host. They are killed promptly in brine, provided the salt comes in direct contact. Owing to the difficulties of penetration, this cannot be depended upon, and therefore pickled beef from infected cattle is considered free from viable *cysticerci* only after the expiration of three weeks following slaughter. The life of many pathogenic bacteria is shortened in brine, depending upon the concentration and temperature, but the salt must be looked upon as an antiseptic rather than a germicide; that is, it prevents growth rather than kills the bacteria that are present. From a sanitary standpoint there is some, though slight, danger of conveying infection in foods that have been improperly salted or pickled. Attention is called to the fact that the first cases of botulism studied by van Ermengem were caused by ham kept in brine under conditions favoring anaërobic growth. Ripe olives which were responsible for botulism were also treated in brine. In the first instance, the ham stuck partly out of the water, and in the second instance the brine was very weak and dirty; and the heat of preserving insufficient.

Preserving.—*Jellies and Preserves.*—By preserving is commonly understood the addition of a large amount of sugar. The principal preserves are jellies, marmalades, jams, and fruit butters. These substances are entirely free from the danger of conveying infection, partly on account of the antiseptic action of the sugar, but mainly because they are always cooked in preparation. A strong solution of sugar will prevent growth, but cannot be depended upon to kill parasites. However, most pathogens die under such conditions in the course of time. A very strong syrup (50 per cent sugar) is necessary to prevent the growth and development of *botulinus* toxin. Jellies and preserves have a comparatively clean bill of health.

Jellies are frequently adulterated by the substitution of apple stock. Apples contain a large number of pectose bodies which favor jellification. Pectose is a carbohydrate ($C_6H_{10}O_5$)* similar in composition to cellulose. When boiled with an acid, they are hydrolyzed to pectin which take the jelly form on cooling. Pectose bodies which will insure jellification may be purchased on the market.

A common method of manufacturing jelly for the trade has been to use a stock of apple juice or cider, or a preparation made from the cores, skins, and rejected portions of the apple at evaporating factories, or from whole rejected apples. This stock is used as a common base for the manufacture of jellies of different kinds. Apple juice used as a substitute for other fruit juices in the making of preserves is a common fraud and an adulteration, according to the Food and Drugs Act, unless plainly stated upon the label. Jellies are also colored artificially, particularly with the coal-tar dyes. Artificial flavors are sometimes employed. The chemical preservatives most frequently added to

jellies and preserved fruits are benzoic acid and benzoate of soda. Salicylic acid and others are prohibited.

Smoking.—The smoking of fish, beef, hams, and other food products consists mainly in rapid drying plus the germicidal action of certain substances in the smoke.⁹⁴ The meat or fish is exposed to the smoke of a smoldering wood fire of oak, maple, or hickory, usually after a preliminary salting. The articles so exposed become dry and impregnated with pyroligneous products—acetic acid and creosote, formaldehyd, and other germicidal substances. An artificial or quick method of smoking meat formerly used, but now contrary to law, consisted in brushing the pieces or dipping them in pyrolignic acid at definite intervals, and finally drying them in the air.

The effects of smoking do not go deep—the penetration is only partial; therefore, in sausages of generous diameter putrefaction often occurs in the interior. Smoked sausage may, therefore, be dangerous, as far as various parasites and the products of decomposition are concerned, and the same is true of smoked ham and other meats exposed in large pieces. As smoked meats are often eaten raw, the occasional survival of parasites in such products has some sanitary significance.

Canning.—The process of canning is practically synonymous with sterilization and is, therefore, one of the best sanitary safeguards we have against parasites and the injurious products of putrefaction in foodstuffs. The process of canning was discovered by the confectioner, M. Appert, of Paris, in 1804 to 1809, long before the days of bacteriology.⁹⁵ Appert found that meats and other foods in sealed vessels would usually keep indefinitely if, after being sealed, they were kept for an hour in boiling water. He improved the process, in 1810, by introducing a method of sealing the cans after the heating process had driven out the air and replaced it with steam, so that when cool a vacuum is formed. For all practical purposes this is the universal method of canning to-day.

In recent years, the method of fractional sterilization has been used, especially in home canning processes. In this method the cans are given a second, and even a third heating spaced at intervals of twenty-four hours. This permits the resisting spores to germinate during the interval, and the newborn bacteria are readily killed in the next heating.

The time and temperature vary for each food, for the size of the can and the bulk of the pack. Bacteria are killed at definite temperatures in a given time, but the heat must penetrate. It takes longer to heat the contents of a

⁹⁴ The process was probably accidentally discovered in connection with crude attempts to use artificial heat for drying purposes.

⁹⁵ Nicholas Appert, in France, first preserved food in glass jars by sealing them hermetically and heating, in 1804. He published *The Art of Preserving Animal and Vegetable Substances* in 1810. In 1810 Peter Durand obtained a patent in England for preserving fruits, vegetables and fish by hermetically sealing them in tin and glass cans. In 1820 William Underwood and Charles Mitchell, emigrant employees from a canning factory in England, opened a factory in Boston where they canned plums, quinces, cranberries and currants. Glass was used exclusively until 1825, when Thomas Kensett secured a patent for use of tin cans and commenced to use them in his factory.

large can than a small can, and penetration is much slower through spinach or sweet potato than it is through soup. The time and temperature for processing each article, therefore, depends on various factors which can be determined, depending on the size of the pressure boiler and the load. Unless attention is given to these matters, spoilage is inevitable. These same principles apply to home canning. Only fresh and sound fruits and vegetables should be put up.

There is also a prevalent notion that once opened, the contents of the can should be emptied into some other vessel. It is generally believed that food kept in an open tin can acquires injurious properties. This is a fable, like the souring of milk due to a thunderstorm. On the other hand, canned food may become contaminated or infected after opening, and the same care as to cleanliness and refrigeration is necessary with fresh food as with cooked food.

The process of canning fortunately does not interfere seriously with most vitamins. Its effect varies with the food and the vitamins. It is now well known that most of these "unknown dietary factors" are not destroyed by heat in an acid medium and in the absence of oxygen; practically all our food-stuffs have an acid reaction. The antiscorbutic vitamin (vitamin C) is the most sensitive to heat. It has been shown that it is oxidation rather than heat which affects vitamins, and that when the "accessory factors" are injured it is usually when the food is heated in contact with the air, as in cooking. The effect of heat on the vitamins is complicated, and the literature is large. It has been collected and tabulated by Kohman.⁹⁶ Hess has shown that canned tomatoes retain their antiscorbutic properties.

Before meats are canned they are first parboiled for eight to twenty minutes, in order to secure the shrinkage before the meat is placed in the can. In the parboiling there is a certain loss of fat, soluble mineral matter, meat bases, and water. However, the shrinkage of the meat concentrates it, as far as nutritive value is concerned, and, therefore, compensates for the loss. The parboiled meat is then placed in the tin and a small quantity of the soup liquor added. The cans are closed and then placed in autoclaves and subjected to steam under pressure. Usually a small hole is left in the can in order to permit the exit of air and gases. This is sealed off at once after heating. The cans are then subjected to a second heating at 225° to 250° F. from one to two hours. A modified process consists in placing the cans upon an endless conveyor which exposes the can to a high temperature in an oil bath a sufficient length of time to sterilize the contents at one exposure.

Canned foods are sterile foods; they are the safest foods that come to our table. Canning is synonymous with cooking, and when properly canned, the contents are thoroughly cooked. The process of canning has proved of inestimable benefit to mankind. It enables nourishing food of a perishable character to be kept and transported to great distances and to be used in localities

⁹⁶ Bull. No. 19L, National Canners Assn., May, 1922.

where fresh foods are unobtainable. Without this method of preserving foods the pioneer and the explorer would be seriously handicapped. Large army and navy maneuvers would be materially impeded, and great metropolitan cities would be impossible. Wiley states that "the winning of the West has been marked by the débris of the rusty can."

Canned foods are not only safe, but wholesome and nutritious. The process permits us to have a well-balanced ration throughout the year—irrespective of season. The canning industry is growing to enormous proportions, and, on account of the great importance of the process, the character and quality of foods thus preserved should be wholly above suspicion, and no adulteration or sophistication of any kind permitted. In the interest of honest and informing labeling, every can should be plainly stamped with the quantity and true nature of its contents and also the date when it was first sterilized.

Spoilage may result from insufficient processing, defective cans or the use of unfit material. These losses are generally classed under the heads of *swells*, *flat sours*, and *leaks*. The ends of a swelled can bulge, and when opened there is evidence of gaseous fermentation and spoilage. "Flat sour" is a term applied to acid decomposition without the formation of gas. "Leaks" are usually due to imperfect covers or to pin holes.

If we analyze the different factors responsible for the spoilage of canned fruits and vegetables, we will find that the most important are: (1) use of unfit raw material; (2) use of unfit cans and glass jars; (3) carelessness in the matter of cleanliness; (4) overfilling of the cans; (5) carelessness in sealing the cans; (6) imperfect methods of processing. No can that shows evidence of spoilage should be used as food.

Emphasis has always been placed upon the necessity of a vacuum for the proper preservation of canned foods. Bacteriologists, however, have shown that sterile foods may be kept indefinitely in the presence of air. *Sterility*, then, is the great desideratum, both from the sanitary and economic standpoints. The time and temperature of processing varies with the food and its condition as to freshness and cleanliness. Allowing foods to stand before processing permits resisting molds and spores to form and renders sterilization unnecessarily difficult.

Practically all foods canned in the ordinary way contain some tin. The amount varies with the acidity, and also the age and temperature of the package. Meat extracts, on account of their acidity, take up more tin than do most other meat products. For the same reason, certain fruits and vegetables, such as canned peaches, cherries, pears, apricots, pineapples, tomatoes, asparagus and tomato soup take up tin from the can. Canned lobster and shrimp are relatively active as solvents of tin on account of the amino-acids they contain.

Fortunately, tin is not very toxic. Cushny states that chronic poisoning from tin is unknown, and that animals present no symptoms when subjected to prolonged treatment with larger quantities of tin than are contained in any preserved foods. Schryver found no indications of cumulative action

when as much as 2 grains per day is taken. So far as we know, tin plays no part of importance in toxicology.

The tin now used to coat sheet iron is practically free of lead. The tin coating is not always perfect, and the exposed iron is readily attacked and may spoil the contents by discoloration or metallic taste. For certain foods the tin is coated with gum or varnish, which is not acted upon by the contents of the package. Glass containers have advantages, which are counterbalanced by expense, weight, bulk and breakage, both in handling and in processing.

CHEMICAL PRESERVATIVES

Chemical preservatives are nothing more nor less than antiseptic substances; that is, substances which restrain the growth and development of bacteria and molds. Chemical preservatives in the proportions commonly used may have little or no germicidal action. Such substances as sugar, salt, vinegar, vinegar extract of spices, and the pyroligneous products in wood-smoke are not regarded as "chemical" preservatives, but as "natural" preservatives or condimental substances, although their mode of action is precisely the same as the chemical preservatives. There is a great prejudice against the use of any preservative for our foods if this preservative is a "chemical" or "drug," whereas no objection is raised to the same substance if derived from "natural" sources. Thus, foods exposed to a smoldering wood-fire become impregnated with pyroligneous acid, which includes creosote, acetic acid, and probably formaldehyd and other substances having antiseptic properties. This method of food preservation is not only countenanced by the law, but is favored on account of the savory result and the antiquity of this "natural" process.

There was a great increase in the use of chemical preservatives in foods during the several decades prior to 1906, when the Pure Food and Drugs Act went into effect. Chemical preservatives were grasped upon, owing to the fact that this was the cheapest and surest method of preservation. Fortunately we possess two efficient and wholly unobjectionable processes for the preservation of food, viz., refrigeration and sterilization by heat, which for the most part make it unnecessary to resort to the use of chemical preservatives. One of the most objectionable uses that can be made of chemical preservatives or any other method of food preservation is to conserve foods which are so decayed as to be unfit or possibly injurious to health if used fresh. The law cannot be too strictly enforced in order to prohibit the use of chemical preservatives and condiments used to disguise such foods, which may then be sold at high prices as first quality.

Upon general principles it is undesirable to add a chemical substance of whatever nature to food for the purpose of preserving, coloring, or improving its appearance, and in most countries this practice is prohibited by law. There are, however, a few instances in which the addition of some chemical preservatives in minimal amount seems harmless, and occasionally even desirable, as,

for example, small quantities of benzoate of soda in catsup; a thin film of gum benzoin as a protective coating for chocolate, etc.

Countries differ widely concerning the use of chemical preservatives in food. Much more latitude is permitted in England than in other countries. No sweeping generalization can be made concerning all chemical preservatives. Each substance must be considered for itself, and each substance must further be considered in relation to the particular foodstuffs for which it is proposed. It may, however, be stated as a general rule that any chemical which is poisonous in large amounts should be considered as poisonous in small amounts until the contrary is proved. In other words, the consumer is entitled to the benefit of the doubt. The toxicology of various food preservatives is in its infancy and frequently presents a very difficult and complex problem. Thus, lead in one large dose is not particularly harmful. The older practitioners frequently gave twenty, thirty, and more grains of sugar of lead (acetate of lead) for diarrheal affections. Only a minute portion of the lead taken in one large dose is absorbed; the rest is quickly eliminated. However, if the same amount of lead should be taken in small subdivided daily doses, enough would be absorbed and retained by the tissues so that serious chronic lead intoxication would develop, resulting from the cumulative action. On the other hand, hydrocyanic acid, one of the most poisonous chemicals known, is harmless in small amounts, for the reason that when introduced into the body it meets the available sulphur (H_2S), with which it unites to form a sulphocyanid, as $KSCN$. The potassium sulphocyanid is not poisonous, and it has been shown experimentally that animals are able to withstand larger quantities of hydrocyanic acid by first giving them substances which increase the available amount of sulphur to form this chemical combination. Benzoic acid in large amounts is irritating and produces well-defined symptoms of poisoning; small amounts of benzoic acid are paired in the liver and eliminated by the kidneys as hippuric acid, a harmless constituent of the urine. Hydrochloric acid, and possibly acetic acid and alcohol are all poisonous in large amounts, but they may be regarded as harmless if the amounts taken are sufficiently small. There can be no defense for the use of formaldehyd, salicylic acid, sulphites, and a host of other chemicals. So far as we know, the human organism possesses no natural mechanism for rendering them harmless. There can be no defense for the use of chemical preservatives to hide inferiority.

Benzoic Acid and Benzoate of Soda.—These are weak germicides at best, and benzoate of soda in dilutions of 1:1,000 commonly used in food has feeble antiseptic power. Benzoic acid is an organic acid contained largely (12 to 20 per cent) in gum benzoin, and also in balsam of Peru and balsam of Tolu. It is obtained from gum benzoin, from the urine of herbivorous animals, and artificially from toluene, by treating it with chlorin and heating with water to $150^{\circ} C$.

The reason why benzoic acid in moderate amounts is believed to be harmless is that the body possesses a special mechanism for taking care of this substance. Many of our ordinary foods contain substances which are trans-

formed in the body into benzoic acid. Some foods, such as cranberries, contain this acid in notable amounts. Benzoic acid meets glycocoll (one of the decomposition products of protein) in the liver. Benzoic acid and glycocoll form hippuric acid, a harmless constituent of the urine. We know, therefore, that the human organism is prepared to take care of and render harmless a certain amount of benzoic acid; we know that this mechanism is a very efficient one and is capable of taking care of relatively large amounts of benzoic acid.

The United States permits the use of benzoate of soda in quantities not exceeding 0.1 per cent in certain foods, but the addition must be plainly stated on the label. There can be no serious objection from the standpoint of health to the addition of this amount of sodium benzoate to catsup, on account of the small quantity of this article consumed at any one time. Further, on account of the long time a bottle of catsup is usually kept after it is opened in the household, there is, thus, the added economic gain of preserving the catsup until it is all consumed. The same object may be obtained by the use of a sufficiently strong vinegar extract of spices, but the question may be asked whether the aromatic and preserving substances in the vinegar extract of spices may not be more irritating than the sodium benzoate.

Spices.—Hoffman and Evans⁹⁷ have shown that ginger, black pepper, and cayenne pepper fail to prevent the growth of microorganisms. Nutmeg and allspice have slight antiseptic properties, but only for a very few days. Cinnamon, cloves, and mustard, on the other hand, have very marked antiseptic powers and are valuable preservatives. The active antiseptic constituents of mustard, cinnamon, and cloves are the aromatic or essential oils which they contain.

Borax and Boric Acid.—Both boric acid and borax are only mild antiseptics. They are not very potent germicides. They are generally used together, for the reason that the combination of the two is more efficient than either one alone. Locally boric acid is not very irritating, and for this reason it has been extensively used in surgical practice. To some skins, however, it is very irritating, and cases are reported of its absorption from wounds and cavities when used too freely, causing depression and eruptions, such as erythema and urticaria. Fatal results have been reported in a few cases from injecting the solution into abscess sacs, or from washing out the stomach with it; or from taking a very large amount by mouth.⁹⁸

Borax and boric acid are not allowed as preservatives in food in the United States. Formerly, they were extensively used for preserving meats, milk, butter, oysters, clams, fish, sausage and other foods. For meat, they were often mixed with salicylic acid and applied externally. For milk it was a common practice to add to one quart of milk 10 grains of a mixture of equal parts of borax and boric acid; for butter the amount used was about one-tenth of an ounce to the pound.

The effect of small amounts of boric acid and borax upon healthy human

⁹⁷ *J. Indust. & Eng. Chem.*, 1911, 3: 835.

⁹⁸ *J. Am. M. Ass.*, 1921, 76: 378.

beings has been extensively studied and has resulted in conflicting testimony. These substances are not normal constituents of the body, nor are they normal constituents of foods. Furthermore, there are better methods of conserving food. Therefore the use of borax and boric acid should be prohibited until satisfactory evidence has been adduced that they are free from harm in the amounts commonly used for preserving food.

Formaldehyd.—Formaldehyd was used extensively as a preservative for milk and occasionally for other articles of food. Concentrated solutions of formaldehyd in large quantities are irritating, and death in isolated instances has been reported from the swallowing of amounts of from 1 to 3 ounces. There has been much discussion as to the effect of the small quantities ordinarily used as a food preservative. Bliss and Novy⁹⁹ and Halliburton¹⁰⁰ have shown conclusively that small quantities of formaldehyd greatly delay the digestion of proteins by the gastric and pancreatic juices, the digestion of starch by the pancreatic juice, and the curdling of milk by rennet. It is also known that some individuals are especially susceptible to the effect of formaldehyd, small quantities in the food causing dyspepsia and other disturbances of digestion. Formaldehyd unites directly with protein matter to form new compounds of an undetermined nature. Thus, formaldehyd added to egg albumin prevents its coagulation by heat, and added to gelatin prevents liquefaction. It hardens tissues, so that it will render fish and meat tough and brittle, even in proportions as dilute as 1 to 5,000, hence it is not generally applicable as a food preservative. In small amounts it delays decomposition; in large amounts it is an active germicide. Its use in milk was recently advocated by no less an authority than von Behring, but this view met with almost unanimous protest.

There can be only one opinion concerning the use of formaldehyd in foods, and that is absolute condemnation of the practice. It is prohibited by the statutes of practically all nations having pure food laws.

Salicylic Acid.—Individuals differ greatly in their susceptibility to salicylic acid. In mild cases of poisoning with this substance there is a feeling of fullness in the head with roaring sounds in the ears, dimness of vision, profuse perspiration, confusion, and dullness. Large doses of the acid cause intense irritation of the throat and stomach, leading to vomiting and difficulty in swallowing. Later there may be diarrhea. Eczema and other skin eruptions may appear, and dimness of vision and deafness may continue for some time. The long-continued use of salicylic acid and its salts has led to a form of chronic poisoning in which the chief symptoms have been loss of appetite, diarrhea alternating with constipation, irritation of the kidneys, skin eruptions, and mental depression. Such results are said to have followed the use of articles of diet preserved with salicylic acid. The use of such foods may be objectionable in the case of aged, feeble, and susceptible persons. Salicylic acid and the salicylates are more efficient antiseptics than boric acid or borax,

⁹⁹ *J. Exper. M.*, 1899, 4: 47.

¹⁰⁰ *Brit. M. J.*, 1900, 2: 1.

but they are not used extensively on account of the taste, or rather the tendency to cause unpleasant flavors. They were for the most part used in jams, fruit juices, soda-water syrups, cider, wines, and other sweet preparations. The objection to the use of salicylic acid in food is practically unanimous and well founded. It is prohibited by the regulations under the pure food act of the United States.

Sodium Nitrate.—Sodium nitrate or potassium nitrate (saltpeter) is not used as a preservative, but as an indirect coloring matter. It retains and accentuates the red color of meat. It is not known to be harmful in the small quantities in which it is commonly employed to give a good color to corn and pickled meat products. Under certain circumstances, nitrates are permitted by the United States pure food regulations.

Potassium permanganate is also used on the surface of meat to destroy the surface evidence of decomposition. This may be detected by heating a knife in hot water, plunging it into the meat, and withdrawing it quickly, which brings out the hidden odors of putrefactive changes.

Sodium Fluorid.—Sodium fluorid has been extensively used as a preservative, antiseptic, and insecticide. It has considerable antiseptic power, putrefaction being delayed by the addition of 1 part to 500; and 1 in 200 arrests completely the growth of bacteria. It is highly poisonous to nearly all the lower forms of life, especially to microörganisms, including algæ. It does not coagulate protoplasm but acts as a general protoplasmic poison. For mammals, sodium fluorid is not a very toxic substance, the fatal dose by the mouth being 0.5 gram per kilogram of body weight, and subcutaneously 0.15 gram per kilogram of body weight. The fluorids on administration are deposited in the bones, which usually become white and brittle, and contain crystals of calcium fluorid. It is well to call attention to the fact that fluorin, in very small traces, is a normal constituent of bone, teeth, milk, eggs, etc. In large amounts and concentrated, it is directly irritating to the mucous membrane and produces vomiting, diarrhea, and abdominal pains. Death of a ten-year-old girl has been caused by the ingestion of one teaspoonful in a little water, given in mistake for Rochelle salts.¹⁰¹ Baldwin¹⁰² reports a number of cases of sickness and death resulting from the accidental ingestion of sodium fluorid, usually taken in mistake for baking powder. Recovery from non-fatal doses is usually rapid and complete. There is no evidence that small quantities ingested daily for a long period of time are harmful.

Sodium fluorid forms the basis of most roach powders which contain from 16 to 47 per cent of the fluorid finely ground up and intimately mixed with the bait. It is fatal to roaches when so ingested. (See page 379.)

Hydrofluoric Acid.—Schultz¹⁰³ exposed cats for four days to concentrated fumes of hydrofluoric acid without serious effects. The air was so impregnated that glass held at some distance from the source was etched. Hydrofluoric

¹⁰¹ *Bull. Mass. State Bd. of Health*, 1911, 6: 341.

¹⁰² *J. Am. Chem. Soc.*, 1899, 21: 517.

¹⁰³ *Arch. f. Exper. Path.*, 1889, 2.

acid is much used for disinfecting vats and tanks in making fermented drinks. It is a powerful germicide.

Sulphites.—Sulphites act as antiseptics and also preserve the red color of meats. Sodium sulphite and bisulphite and sulphurous acid are used principally upon fresh meats, where they act as a preservative and as a retainer of color. Sulphites, even in minute amounts, interfere with the action of ferments, and thus influence digestion. Free sulphurous acid is very irritating. Sodium sulphite is very poisonous when injected subcutaneously or intravenously. Death occurs by paralysis of respiration. Much larger quantities are tolerated by the mouth, the sulphite being slowly absorbed. The greater part is converted to the harmless sulphate during and after absorption. The quantities ordinarily used in preserved food cause no immediate symptoms, even when continued for several months. In 1908, the Imperial Board of Health in Germany forbade the use of sodium sulphite in food on account of its dangerous properties, and it is also forbidden by our Federal Pure Food Act of 1906. Sulphur dioxid is much employed for the bleaching of fruits. No objection is made to foods which contain the ordinary quantities of sulphur dioxid, if the fact that such foods have been so prepared is plainly stated upon the label of each package. An abnormal quantity of sulphur dioxid placed in food for the purpose of marketing an excessive moisture content is regarded as fraudulent adulteration, under the Food and Drugs Act of June 30, 1906.

Sodium Bicarbonate.—Sodium bicarbonate is sometimes added to milk in order to neutralize the excess of acid, and this delays souring. It is too ineffective as a germicide for general use as a food preservative.

Hydrogen Peroxid.—Hydrogen peroxid is perhaps one of the least dangerous of the chemical preservatives, and is considered by some to exert no deleterious effect whatever in the quantities commonly used. It was at one time advocated and used for the preservation of milk and also of wine, beer and fruit juices. In milk it oxidizes and destroys vitamin C.

Arsenic.—Arsenic in food comes from a variety of sources. Glucose was apt to contain it, especially if impure acid had been used to hydrolyze starch in its production. This was the source of the arsenic in the beer which caused the epidemic of peripheral neuritis in 1900 in England. Samples of the glucose contained from 0.01 to 0.1 per cent of arsenic. The finished beer contained from 1 to 3 grains of arsenic per gallon. Arsenic may also contaminate certain anilin dyes as well as shellac,¹⁰⁴ which is now so much used as a coating for some kinds of cheap confectionery and baker's goods, and also as a varnish on receptacles and containers of various kinds. Another source of arsenic in food is from insecticide sprays (pages 275-277).

In England liquid food is considered adulterated if it contains as much as 0.01 grain of arsenic per gallon, and solid foods are considered deleterious if they contain as much as 0.01 grain per pound.

The use of preservatives containing lead, arsenic, or other substances known to be poisonous finds no advocate.

¹⁰⁴ Cir. No. 91, Bur. Chem., U. S. Dept. Agric., 1912.

THE PREPARATION OF FOOD

Cooking.—Cooking may be regarded as the greatest sanitary innovation ever introduced by man to protect himself against infection. Fortunately, most microorganisms pathogenic for man are destroyed comparatively easily by heat. Therefore, thorough cooking renders food reasonably safe so far as these dangers are concerned. Fire is the great purifier. Cooking has other advantages. It softens the connective tissue and renders meat more tender. The bundles of fibrillæ are loosened from each other, the albumin is coagulated, the flavors are improved, and new flavors are developed, which enhance its digestibility.

Metchnikoff in his "new" hygiene dwelt upon the great sanitary value of cooking. Perhaps no other single factor in preventive medicine protects us to an equal degree against infection. Metchnikoff believes that we should eat nothing in its raw state. This seems almost as extreme as the cult which proclaims the contrary. The newer knowledge of nutrition confirms the experience that it is advantageous to eat a certain amount of raw food, particularly fruits and vegetables. If for any reason cooking were to cease, there would be such a great increase of infections as to amount to a calamity.

One of the important functions in the preparation of food is to render it savory; tender, and appetizing. Foods that appear inviting aid digestion by stimulating the secretion and flow of the digestive juices. Foods that are rendered soft and tender are more readily digested, but it should not be forgotten that the teeth need exercise to keep them in good condition. Tough meats may be pounded to separate the connective tissue bundles, or may be chopped or minced as an artificial aid to mastication, or may be steeped for several hours in fresh milk or sour milk, in which case the fibers are softened through the action of the bacteria and their enzymes. In the case of vegetables, cooking breaks open and softens the cellulose envelopes and fibers; the starch grains swell and burst, and the insoluble starch is converted into soluble starch or dextrin.

Cooking has a few minor disadvantages—there is a loss of mineral salts, and some of the nutritive constituents, also a diminution in the antiscorbutic property of food generally.

Temperatures.—Heat also kills the true toxins,^{104a} which are destroyed in a few minutes between 70° to 80° C. Foods may sometimes contain heat-resisting poisons. Thus, boiling has no effect upon muscarin, a poison in certain toadstools. Heat also does not destroy a principle sometimes found in poisonous mussels. The colon bacillus and other microorganisms produce thermostable substances that are poisonous when injected into the lower animals, but the relation of these heat-resisting toxic substances to food poisoning in man is conjectural.

^{104a} The toxin of scarlet fever is heat resistant but not a danger in food.

Trichinae, according to some authorities, die at 65° C., but the United States Bureau of Animal Industry after repeated experiments places the thermal death-point at a temperature of 55° C. Some writers state that cysticerci, or the larval stage of tapeworms, die at 52° C. The nonsporulating bacteria are for the most part destroyed at 60° C. Food thoroughly cooked throughout will always reach these temperatures, but much meat and many vegetable food substances are preferred rare or underdone, and, while the outside of a large piece of meat may be thoroughly cooked or even charred, the interior may be practically raw or at least not have reached the temperature necessary to destroy parasites. A dish of spaghetti, charred on the outside, may not kill typhoid bacilli in the center of the mass (page 107).

Meat that is well cooked throughout always reaches from 60° to 70° C. on the inside. It should be remembered that heat penetrates a large piece of meat slowly. Küchenmeister found that joints require boiling for several hours for the interior to reach a temperature of 77° to 80° C. Perroncito placed a ham of about 6 kilos weight in cold water which was then raised to the boiling point. The water boiled when the interior of the ham was only 25° C. After 35 minutes, it was 35° to 40° C., and after two hours, the temperatures in different parts of the interior were 46°, 55°, 58°, 62°, 64°, and 67° C. Rupprecht found that boiling for 45 minutes as practiced in Saxony did not produce a higher temperature than 75° C., and this only in thin pieces of meat. He found that the interior temperature of a rapidly roasted sausage was only 28.7° C. Meat placed in a quick oven or boiled soon forms a hard, coagulated and insulated coating that retains the juices, but retards the penetration of the heat.

Delepine and Howarth carried out experiments upon the temperatures reached in baking meat pies. They noted that the temperature of the center of the pie, said to be underbaked, but having all the external appearances of being well baked, did not exceed 47.2° C. The center of a pie, obviously overbaked and acknowledged to be so, had not reached beyond 86.6° C. Delepine points out that pies might be so cooked that bacteria might continue to grow in their center during the greater part of their stay in the oven, and the bacteria would certainly not be killed.

Fireless Cookers.—The observations of Becker, Grove, and others concerning the heat of cooking are practical and important in the preparation of food. Exposure to moist heat at 60° to 70° C. for a long time has the advantage of cooking foods thoroughly throughout. This treatment prevents burning or the results of overheating; the juices are retained. The process requires little or no attention. Meat is thereby rendered tender and juicy, vegetables thoroughly soft, and the starch grains are all opened. A modification of this method is found in the fireless cookers now offered for sale in various forms. These devices consist simply of a well-insulated box. The food is heated, placed in suitable compartments, and a temperature above 70° C. maintained for many hours. Most of the parasites that concern us are thus killed throughout the mass—another advantage of fireless cooking.

Cooking Utensils.—Certain precautions are advisable in the choice of pots and pans used in cooking. Brass and copper are not advisable, and if used must be kept scrupulously clean. Copper acetate (verdigris) which sometimes forms in copper food containers, is greatly feared but is not very toxic. Acid foods should not be cooked in copper vessels, and milk and saccharin substances should not be kept in copper containers on account of the possibility of the organic acids dissolving the copper. Foods should not be fried in copper pans owing to the dissolving action of oleic, palmitic and stearic acids in lard and other fats. Mallory ¹⁰⁵ believes that chronic poisoning with copper is the cause of hemochromatosis. While the fully developed disease is relatively rare, he states that the early stages and lighter forms are fairly common but unrecognized. Iron, tin, nickel and aluminum ware are least objectionable. Enameled ware is satisfactory provided the glaze does not contain lead. If the glaze contains lead it must be insoluble in acid. Glassware, being insoluble, is least objectionable from this standpoint. Foods cooked in metal pots and pans always contain some of the metal, which is usually in solution in the form of salts, or may be in metallic form, or combined with chemical constituents of the food. The amount varies, especially with the reaction, temperature, time of cooking and other factors.

Methods of Cooking.—Much depends upon the method of cooking. The principal methods in ordinary use are: roasting, broiling, boiling, frying, and stewing.

Roasting or broiling causes considerable shrinking, due mainly to loss of water. The heat coagulates the exterior of the meat and thus prevents the further loss of juices and drying up. In order to obtain adequate heating of the meat throughout a large joint without burning and drying the exterior, it is necessary to baste it from time to time with hot melted fat. This also helps to form a protective coating.

In *boiling* the meat is placed either in hot or cold water, depending upon the object desired. If it is desired to maintain the flavors within the mass, the meat should be plunged into boiling water. This quickly coagulates the albumins at the surface. If a rich broth is desired, the meat should be placed in cold water and gradually heated. In this way the soluble proteins and extractives pass out into the surrounding water. The albumin of meat begins to coagulate at 134° F. (56° C.); the connective tissue is changed to gelatin and dissolved above 160° F. (72° C.). Long boiling makes meat fibers tough.

Frying consists in placing meat or other substances into very hot fat, lard, or vegetable oil. This causes a speedy coagulation of the surface similar in all respects to that brought about in the first mentioned process of boiling. The flavors and juices are thereby retained. If the fat is not very hot it will penetrate the tissues and cause the meat or other substance to become greasy and unpalatable. Fried substances are apt to be indigestible on account of the

¹⁰⁵ *Am. J. Path.*, 1925, 1: 117.

large amount of grease that adheres to and penetrates into them. It is, therefore, better to plunge food into deep fat, piping hot.

In *stewing* the meat is cut into small pieces and placed in cold water, which then is heated slowly to about 180° F. (84° C.), at which the whole is kept for several hours. If heated above 180° F. the meat becomes tough, stringy, unpalatable, and of diminished digestibility.

Fermentation is of great use in the preparation of foods. The best example is the leavening of bread. The yeast ferments the carbohydrates in the flour with the production of carbon dioxid and alcohol. The carbon dioxid renders the bread porous; the gas is held within the loaf on account of the glutinous property of the protein (gluten) in the flour. Fermentation is an adjunct in the preparation of many other foods and beverages, such as cheese, sauerkraut, vinegar, beer, wine, cider, etc.

CHAPTER II

ANIMAL FOODS

The animal foods used by man are not of great variety and source. They include the flesh and various organs of the herbivorous animals, swine, domestic and wild fowl, eggs, fish, shellfish, insects and their products (honey), milk, and milk products. The flesh of carnivorous animals, except that of fish, is unpalatable.

The most important animal foods from the standpoint of the sanitarian are milk and meat.

MILK ¹

Milk is our most important food. It is the best single food. The exceptional value of milk is due to the fact that it contains all the essentials of a balanced diet; it is rich in some vitamins, the quality of its protein is especially good, the fat favors growth, and it has a high calcium content in readily usable form. Milk, furthermore, is palatable, readily digestible, and is subject to a great variety of modifications. Even at present prices, it is one of the cheapest of the standard articles of diet, and the most economical source of protein. Milk is a protective food, in that it guards against deficiency diseases when used in combination with other foodstuffs of either animal or vegetable origin.

Those peoples who have employed the leaf of the plant as their sole protective food are characterized by small stature, relatively short span of life, high infant mortality, and by contented adherence to the employment of the simple mechanical inventions of their forefathers. The peoples who have made liberal use of milk as a food have, in contrast, attained greater size, greater longevity, and have been much more successful in the rearing of their young. They have been more aggressive than the non-milk-using peoples, and have achieved much greater advancement in literature, science and art. They have developed in a higher degree educational and political systems which offer the greatest opportunity for the individual to develop his powers. Such development has a physiological basis, and there seems every reason to believe that it is fundamentally related to nutrition.²

The total milk production in the United States in 1924 was twelve billion gallons. About two-fifths of this is consumed as milk, two-fifths as butter, and one-fifth as cheese, ice cream, condensed, evaporated and dried milks. The average per capita consumption of fluid milk in the United States has risen to about one pint daily. It was 0.5 pint in 1900; 0.93 in 1917; and 0.94 in

¹ When not otherwise specified throughout this chapter milk refers to cow's milk.

² E. V. McCollum and N. Simmonds, *The Newer Knowledge of Nutrition*, 3rd Ed., Macmillan, New York, 1925.

1923. More milk is used in the north than in the south; very little in the tropics, and practically none at all in China, Japan, and some other countries. About 16 per cent of the average dietary in the United States consists of milk and milk products.

While good milk has done more than any other single food to obtain and maintain health, bad milk was formerly responsible for more sickness and deaths than perhaps all other foods combined. There are several reasons for this: (1) Milk conveys a greater variety of infections than any other food. Bacteria grow well in milk; therefore, a very slight infection may produce widespread and serious results; (2) of all foodstuffs milk is the most difficult to harvest, handle, transport, and deliver in a clean, fresh, and satisfactory condition; (3) it is the most readily decomposable of all our foods; (4) finally, milk is the only standard article of diet obtained from animal sources consumed in its raw state.

Fresh milk products may be quite as dangerous as the milk from which they are made. Milk laws which ignore milk products are incomplete from the sanitary side, and will fail to accomplish their purpose from the economic side.

Milk is the only single substance whose sole function in nature is to serve as a complete food. It is a perfect food for the suckling of the same species. The milk of one mammal does not fit all the needs of the nursling of another kind: cow's milk is best for the calf, bear's milk for the cub, lion's milk for the whelp, and mother's milk for the baby. After weaning, milk is the best single food to promote growth and nutrition in children. All students of dietetics favor the free use of milk for growing children, who may well take as much as a quart a day, depending upon the amount and character of the remainder of the diet, and also upon individual idiosyncrasies. Milk is quite as nutritious and beneficial whether taken as such or as part of various food preparations.

For the adult milk has certain deficiencies, although patients often do very well for weeks on an exclusive milk diet. As a sole article of diet for adults milk is too low in iron,³ and lacks the reproductive vitamin; it has too much water and too little roughage. Adults should get about a pint of milk a day in the diet.

In view of the many advantages and few drawbacks, sanitarians unanimously encourage the production and use of pure milk, and discourage the distribution and use of poor milk. It is the only food for which there is no effective substitute.

Composition.—Milk is the secretion of the mammary gland. In composition it is exceedingly complex, consisting chiefly of water; several proteins in colloidal suspension; fats in emulsion; sugar, and a number of inorganic

³ Milk is deficient in iron. It has long been known that there is deposited in the liver of the newborn animal a reserve supply of iron, which ordinarily suffices to tide it over the suckling period. Ordinary drinking water almost always contains small amounts of iron, and this doubtless aids in some degree in preventing iron starvation in the infant.

salts in solution; also vitamins, phosphatids, enzymes, as well as antibodies, cells, gases, and other substances. Milk from all species of animals shows a general agreement in physical properties and composition, containing essentially the same ingredients, but exhibiting differences in the relative amounts of the several constituents.

In the fresh state milk is a yellowish white, opaque fluid. Cow's milk has a specific gravity of 1.027 to 1.035; it freezes at a temperature somewhat lower than the freezing point of water (-0.554°C); the electrical conductivity is 43.8×10^{-4} for cow's milk, and 22.6×10^{-4} for human milk. In other words, 58 per cent of the molecules in cow's milk and 26 per cent in human milk are dissociated. The specific heat of milk containing 3.17 per cent of fat is 0.9457. The coefficient of expansion is greater than that of water. Milk shows no maximum of density above 1°C .

Freshly drawn milk of carnivorous animals is, as a rule, acid in reaction. This is probably due to carbon dioxide and acid phosphates. Human milk and that of most of the herbivora are slightly alkaline; cow's milk has been described as amphoteric. Fresh cow's milk is slightly acid to phenolphthalein; but strongly alkaline to methyl orange, indicating that the acidity is due in part to the acid phosphates. The P^{H} values of fresh cow's milk range between 6.5 and 6.8; fresh mother's milk varies between 7.1 and 7.6.

Under the microscope milk is found to contain fat globules and cells, as well as bacteria, debris, and other objects.

The gases dissolved in milk are oxygen, nitrogen, and carbon dioxide (3 to 4 per cent by volume). Oxygen and nitrogen are carried into milk mechanically from the air in the process of milking. Other substances found in milk, but in small quantities, are lecithin, cholesterol, citric acid, lactosin, orotic acid, and ammonia.

The composition of cow's milk may be understood from the schemes prepared by Lucius L. Van Slyke and S. M. Babcock, given on page 698.

Van Slyke and Bosworth ⁴ suggest the following as representing the principal constituents of milk more closely than previous statements. The amounts are based on milk of average composition:

<i>Constituents</i>	<i>Per Cent</i>
Fat	3.90
Milk sugar	4.90
Proteins combined with calcium	3.20
Dicalcium phosphate (CaHPO_4)	0.175
Calcium chlorid (CaCl_2)	0.119
Monomagnesium phosphate ($\text{MgH}_4\text{P}_2\text{O}_8$)	0.103
Sodium citrate ($\text{Na}_3\text{C}_6\text{H}_5\text{O}_7$)	0.222
Potassium citrate ($\text{K}_3\text{C}_6\text{H}_5\text{O}_7$)	0.052
Dipotassium phosphate (K_2HPO_4)	0.230
Total solids	12.901

⁴*J. Biol. Chem.*, 1915, 20: 2.

(Van Slyke)

Milk = 100.0	{ Water = 87.1		{ Nitrogen compounds = 3.2 { Casein = 2.5 Albumin, etc. = 0.7	{ Fat = 3.9
	{ Solids = 12.9			
	{ Solids not fat = 9.0			
	{ Milk sugar = 5.1 Ash (salts) = 0.7			
	{ Carbon dioxide			
	{ Nitrogen			
	{ Oxygen			
	{ Gases { Carbon dioxide Nitrogen Oxygen			
	{ Ash (salts) = 0.7 9.0			
	{ Milk sugar = 5.1 Ash (salts) = 0.7			

(Babeock)

Milk = 100.0	{ Butter fat = 3.6		{ Fat 3.6	{ Total solids 12.7
	{ Olein			
	{ Palmitin			
	{ Stearin			
	{ Myristin			
	{ Butin (trace)			
	{ Butyrin			
	{ Caproin			
	{ Caprylin (trace)			
	{ Caprinin (trace)			
Milk = 100.0	{ Casein 3.00		{ Glycerids of insoluble and nonvolatile acids 3.3	{ Solids not fat.. 9.1
	{ Albumin 0.60			
	{ Lactoglobulin			
	{ Galactin			
	{ Fibrin (trace)			
	{ 0.20			
	{ 3.80			
	{ Milk sugar			
	{ Citric acid			
	{ Potassium oxid 0.175			
Milk serum = 96.4	{ Sodium oxid 0.070		{ Glycerids of soluble and volatile acids 0.3	{ Solids not fat.. 9.1
	{ Calcium oxid 0.140			
	{ Magnesium oxid 0.017			
	{ Iron oxid 0.001			
	{ Sulphur trioxid 0.027			
	{ Phosphoric pentoxid .. 0.170			
	{ Chlorin 0.100			
	{ 0.7			
	{ Water			
	{ 87.3			

100.0

Milk Proteins.—The proteins constantly found in milk are casein, an albumin, a globulin and an alcohol soluble protein. The four proteins are immunologically distinct. The casein and the lactalbumin are basic nitrogenous constituents of milk and occur also in colostrum. Together, the proteins are complete, containing all the amino-acids in proper proportions to build tissue. A trace of fibrin, mucin and other proteins sometimes occurs.

The proteins in milk of a given species are quite constant both in composition and amount; it is, therefore, not necessary, as a rule, to make a special analysis for them. They may be estimated by subtracting the fat, sugar, and ash from the total solids.

Casein is a highly specialized protein found in the secretion of the milk glands of all mammals, but nowhere else in nature; it is a nucleo-albumin, and as such contains phosphorus. Casein is a complete protein—that is, it contains all of the seventeen or eighteen amino-acids necessary to rebuild human protein. It is insoluble in water, but by virtue of its property as an acid it forms soluble salts with alkalis. There are two series of casein salts, basic and neutral; solutions of the latter have a milky appearance. In milk, casein is found dissolved in the form of a neutral calcium salt, which accounts in part for the white opalescent appearance of milk whey. Casein really exists in milk in the form of *caseinogen*, that is, casein in combination with calcium phosphate. The caseinogen is held in solution by the calcium phosphate. It is not coagulated by heat, but is precipitated by acids, for the reason that acids take the calcium from the calcium phosphate, and thus throw the casein out of solution as a curd. This flaky or lumpy precipitate is again soluble in limewater and dilute alkalis. Casein is also precipitated by rennin. Cow's milk contains 2.66 per cent of casein; woman's milk between 0.5 and 0.75 per cent.

Lactalbumin is very similar to the serum albumin of the blood, but it appears to differ from this in some particulars. It coagulates by heating to 70° C., but not with dilute acids, and is precipitated by a saturated solution of ammonium sulphate, but, like all other albumins, is not precipitated in a neutral solution of sodium chlorid and magnesium sulphate. Lactalbumin contains sulphur but no phosphorus. It is present in amounts varying from 0.2 to 0.8 per cent, but is much more abundant in colostrum.

Lactoglobulin occurs in milk merely in traces, while colostrum is comparatively rich in this protein. It coagulates at 75° C., it is precipitated in the same way as serum globulin, and, like serum globulin, is insoluble in water, but is soluble to some extent in weak salt solution. Lactoglobulin and serum globulin are chemically indistinguishable.

Fat.—The fat is suspended in the milk serum in the form of an emulsion. The droplets or globules vary in size. On the average they are smaller in milk from Holstein than from Jersey, Guernsey, or shorthorned breeds. Under the microscope some of the fat globules seem to have an albuminous membrane, but this interpretation is questioned. The fat droplets are lighter

than the milk serum, therefore they rise on standing (gravity cream), or else they may readily be separated by centrifugal force (centrifugal cream). Cream, or top milk, does not consist of fat alone, but contains all the constituents of the milk; it is simply milk rich in fat. Upon shaking the fat globules gradually coalesce into larger drops and lumps to form butter.

The first milk drawn from the udder is commonly poor in fat. This is known as "fore" milk. The middle portion contains about the average percentage, and the last, known as "strippings," is always the richest in fat. The strippings may contain as much as 9 or 10 per cent.

Heat increases the viscosity of milk, and hence hinders the rising of the fat drops; if heated above 63° C. for any length of time the formation of the cream line is retarded or prevented. For this and other reasons, therefore, the richness of milk cannot always be judged by the depth of the cream layer.

Milk fat consists of a mixture of different neutral fats, the principal of which are olein, palmitin, and stearin. These are neutral triglycerids of the corresponding fatty acids. Besides these are found the triglycerids of myristic, butyric, and caprylic acids. The last two are volatile and give to butter its characteristic odor and flavor. Crowther and Hynd⁵ state that the only acids present in more than minimal proportions are the unsaturated acid, oleic acid, and the eight saturated acids of the acetic series (C_4 to C_{18}); namely, butyric, caproic, caprylic, capric, lauric, myristic, palmitic and stearic acids. The composition of the fat is subject to variation, depending upon racial or individual peculiarities, also upon the character of the food and other conditions.

Milk fat is rich in vitamin A. This vitamin promotes growth; its absence induces xerophthalmia and serious disturbances of nutrition. Osborne and Mendel have shown that butter fat may have a blast of steam passed through it for two hours and still retain its peculiar growth-promoting properties. This observation is in harmony with those of McCollum and Davis, that heating butter fat to the temperature of boiling water does not affect its peculiar dietary value. It is apparent, therefore, that any conditions to which milk fats are liable to be subjected during the cooking of foods will not greatly alter its value as a source of vitamin A. Evaporated and dried milks also retain the virtues of this vitamin.

It is now clear that milk fat has no superior among hydrocarbons in dietetic or nutritional value. It is the chief source of vitamin A in our diet.

The percentage of butter fat in milk has long been one of the standards by which milk is tested. The richness of milk gauged by the amount of fat it contains is more of an economic than a sanitary question. Milk with a lower percentage of fat from Holstein cows is relatively just as nutritious a food as richer milk from Jersey and Guernsey cows. Even skimmed milk containing little or no fat is a valuable food. The problem is one of honest

⁵ *Biochem. J.*, 1917, 2: 139.

labeling and the marketing of various grades at prices corresponding to their nutritive contents. When the standard for butter fat in milk is relatively low, say 3.25 per cent, it is a temptation for dairy men to standardize or adjust. A high fat standard encourages the breeding of better cows, requires caution in their feeding and care, and puts a premium upon good dairy methods.

In normal milk the larger proportion of fat droplets agglutinate into tiny clusters or masses. At a temperature of 65° C. or above, these clusters are broken up and the globules are more homogeneously distributed throughout the liquid. When milk is sprayed or atomized at a pressure of about 3,000 pounds at a temperature of about 75° C. the individual fat globules are broken up into fine particles, which remain as a uniform and permanent emulsion known as "homogenized milk." This process applied to cream increases its volume and viscosity, so that cream containing 20 per cent butter fat appears to have the body and richness of a 30 per cent cream.

Researches of Huebner, Keller, and Czerny show that the fats and not the proteins are the cause of much of the digestive disturbances in infants. When the fat is excessive in amount the infant at first seems to thrive, but sooner or later loses weight and appetite, and shows other symptoms, associated with stools composed largely of fat soaps and of a pale gray, hard, and dry constituency. The alkaline bases are so largely drawn upon from the body to saponify the excessive amount of fat in the intestines, that a condition resembling acidosis may appear; furthermore, fermentative changes take place in the intestines and the "catastrophe" ensues.

Fat is the most variable constituent in milk. The amount varies with different animals, and even in the same animal from time to time.

Milk Sugar, or Lactose.—Milk sugar, or lactose ($C_{12}H_{22}O_{11}$), is peculiar to milk; it is found nowhere else in nature. Commercially, milk sugar is obtained from whey as hard rhombic crystals, which have a slightly sweet taste and are soluble in six parts of cold water. Lactose is readily acted upon by microorganisms and reduced to glucose and galactose; the glucose is further changed to lactic acid. This is the common cause of sour milk (see The Fermentation of Milk, page 702).

Lactose, like glucose, reduces Fehling's solution when heated; it is dextro-rotatory. When heated above the boiling point of water it changes to a brownish color as a result of the formation of lactocaramel.

The amount of lactose in milk of any given species is remarkably constant. Breast milk contains (7 per cent) almost twice as much as cow's milk (4.75 per cent). Lactose is not digested and absorbed as quickly as other sugars and therefore favors the fermentative flora in the intestines (see page 711).

Vitamins in Milk.—The early impression that milk must be rich in all the vitamins has met with some disappointments. Considerable variation is known to exist in the contents of vitamins A and C and uncertainty as to vitamin B.

Milk, butter and cream are rich in *vitamin A*; in fact, they are the chief sources of this vitamin in the diet. Vitamin A favors growth and nutrition

and its absence induces xerophthalmia. It is particularly resistant to heat, drying and age. The leafy vegetables also contain this dietary food factor.

Milk contains *vitamin B*, the antineuritic vitamin which protects against beriberi. This vitamin also resists heat, drying and age. It is widely distributed in food of both animal and plant origin.

Milk has but a moderate and variable amount of *vitamin C*. The antiscorbutic property of milk is directly proportional to the amount of this vitamin in the feed of the cow or the diet of the mother. It is a much more fragile vitamin than A, B or D, and readily affected by age, oxidation, heat and other influences. Orange and tomato juices are valuable and available sources of this dietary factor.

Milk contains antirachitic properties, *vitamin D*, but neither cow's milk nor mother's milk can be depended upon to protect growing infants against rickets. It is heat resistant and is not affected by drying or age. It is particularly abundant in cod-liver oil.

Milk contains little of the reproductive vitamin of Evans and Bishop, *vitamin E*.

Milk probably does not contain P. P., the pellagra preventive vitamin. It is absent from butter.

Ferments in Milk.—Milk contains a large number of very active ferments or enzymes which have taken a back seat since the discovery of vitamins. Milk also possesses certain other properties common to blood and living tissues, but, while milk may properly be regarded as a vital fluid, it is not a living fluid as was formerly claimed. In fact, milk begins to decay the moment it is drawn; oftentimes decomposition begins while the milk is still within the udder.

Some of the ferments in milk are normal constituents of that secretion, while others are produced by bacterial activity. Many tests have been devised to determine the kinds and activity of the ferments in milk. The tests most frequently and successfully used are those for catalases and reductases. The absence of certain thermolabile ferments in milk indicates that it has been heated. The presence of bacterial ferments gives an indication of the age of the milk, and the number of bacteria it contains, and also helps to distinguish between fresh normal milk and pathologically changed milk. Some of these tests are in practical everyday use.

The enzymes in milk are the following:

Galactase.—Galactase is a proteolytic ferment, similar to trypsin. It was found by Babcock and Russell to be abundant in separator slime. Ordinarily galactase by itself acts too slowly to cause any material change in the proteins in the short intervals which elapse between the withdrawal of the milk from the animal and its consumption as food. Snyder claims that this enzyme probably assists digestion, in that when milk is used in a mixed diet the proteins have been found to be from 4 to 5 per cent more digestible than when milk is omitted from the diet.

Lactokinase.—Hougardy has shown that milk contains a ferment or a

kinase similar to enterokinase. Lactokinase has been found to accelerate the digestion of proteins by pancreatic juice. This property is destroyed by heating the milk at 73° to 75° C.

Lipase.—This fat-splitting ferment was found in milk by Marfan and Gillet. Human milk exhibits this property to a higher degree than cow's milk. The former has a lipolytic activity of from 20 to 30 on Harriot's scale, while cow's milk shows an activity of only 6 to 8. Lipase withstands cold, but is destroyed by heating to 65° C.; it is non-dialyzable and is held back by a porcelain filter. It probably hydrolyzes the higher fats of milk, at least to some extent, and may possibly account for a small part of the acidity of some milk.

Catalase.—Milk contains no true oxidases or oxidizing ferments proper (Kastle). It decomposes hydrogen peroxid and has the power of effecting the oxidation of a considerable number of easily oxidizable substances in the presence of hydrogen peroxid or ozonized oil of turpentine. In other words, milk contains catalase and peroxidase. Catalase is widely distributed among animals and plants; in milk it is probably of bacterial origin. Jolles has pointed out that human milk decomposes five or six times as much hydrogen peroxid as cow's milk. Considerable importance has been attached to this difference, which has also been used to distinguish human milk from cow's milk. Little is known of the function of catalase. Hydrogen peroxid is probably formed in both animal and vegetable tissues during vital activities. The catalase would destroy it and thus prevent its accumulation in the cell, which otherwise would destroy its life.

Peroxidase.—Milk contains substances capable of inducing the oxidation of guaiacum and other readily oxidizable substances by means of hydrogen peroxid or ozonized oil of turpentine. These substances are known as peroxidases. The peroxidases are destroyed when milk is heated to 80° C. The color reactions for these ferments are a convenient test to determine whether milk has been heated beyond a certain temperature or not. The interpretation of this reaction must, however, be guarded, as Gillet and Kastle found that even normal fresh milks vary in the amount of peroxidases which they contain.

Reductase.—Raw milk possesses reducing properties; for example, it reduces Schardinger's reagent, which consists of a solution of methylene-blue containing small amounts of formaldehyd. The reductases in milk are probably of bacterial origin. On account of the bacterial origin of both the catalases and reductases in milk, the detection of these enzymes has a sanitary significance.

Diatase (Amylase).—Béchamp in 1882 isolated from milk a ferment which liquefies starch and converts it into sugar as readily as diatase. These observations have not been confirmed by other investigators (Mora, Van De Velde, and Landtsheer, or Kastle).

"Leukocytes" in Milk.—A large number of cells are normally present in milk. These are not to be regarded as the result of inflammation, unless

they have the characteristics of "pus" cells. Those found in normal milk are leukocytes and degenerated epithelial cells. The number of cells in milk is greatly increased in the presence of garget; toward the end of lactation; on approaching calving time; during periods of excitement, and by various other factors. Varrier-Jones⁶ insists that the only method of distinguishing a pus cell from a normal leukocyte is by means of a differential stain and a differential count. Frost⁷ has described a method of distinguishing live from heated and dead cells.

A cell content of 500,000 or over to the cubic centimeter, especially in a mixed milk, is regarded by the Boston Board of Health as suggestive of some inflammatory condition of the udder, more particularly if associated with streptococci. Such milk is excluded until after satisfactory veterinary inspection of the herd.

Mineral constituents of milk are of the highest importance to the growing animal. The inorganic salts of milk are stated on page 697.

Milk is the best source of calcium in the dietary, both on account of the quantity and the usable form in which it exists. Sherman and Handey⁸ have shown that growing children need one liter of milk a day for optimum calcium intake. Children up to the age of puberty therefore should get about a quart of milk a day. The vegetables are the only other class of foods where calcium content is high enough to be a chief source of the mineral in children's dietaries, but children do not utilize the calcium of vegetables as efficiently as they do that of milk.

Colostrum is the fluid secreted by the mammary gland during the first few days after birth and before lactation becomes established. Colostrum and milk differ markedly in appearance, quality, composition and function. Colostrum contains on an average four times as much protein as the milk of the same cow. This increase is mainly due to the presence of a great quantity of globulin.

In women, colostrum does not appear for at least twelve hours after delivery. Not more than five cubic centimeters is secreted in the first twenty-four hours, and not more than ninety cubic centimeters after forty-eight hours. The cow's udder, on the other hand, is filled with about twenty pounds (9 kgms.) at the time of parturition. The calf takes about two pounds at the first feeding shortly after birth.

Theobald Smith and his coworkers⁹ discovered that colostrum is the chief agent for transferring protective antibodies to the newborn calf. It is very difficult to raise a calf if colostrum is withheld or even postponed twenty-four to thirty-six hours. Without colostrum, calves usually succumb during the first week to intestinal infections and interstitial focal nephritis caused by a particular type of colon bacillus, which is non-motile, indol-producing, and

⁶ *Lancet*, 1924, 2: 537.

⁷ *J. Am. M. Ass.*, 1916, 66: 889.

⁸ *J. Biol. Chem.*, 1922, 53: 53.

⁹ *J. Exper. M.*, 1922, 36: 181, 453; 1923, 37: 671; 1925, 41: 3.

fails to act on saccharose. Colostrum, then, is essentially protective against miscellaneous and ordinary bacteria which are harmless later on when the immunity functions of the calf have begun to operate.

The calves are protected whether the colostrum is fed to them or injected intravenously or subcutaneously. The specific agglutinin against this type of colon bacillus is present in the cow's blood and in colostrum, but is absent from the blood of the newborn calf; it appears within fifteen minutes of a feeding of colostrum. The protecting influence of colostrum is doubtless due to this and other antibodies.

Human colostrum seems to be less important. No harm has been discovered in infants deprived of it altogether. The human placenta is much more permeable than that of ruminants, because there is only a single layer of cells separating the maternal blood from the fetal blood, whereas in ruminants the placenta has a barrier of a three-cell layer. Antibodies pass the human placenta readily; thus, diphtheria and tetanus antitoxins have been demonstrated in the cord blood. Colostrum contains some of these antibodies, but less than the blood. The difference in the amount and composition of colostrum in man and cow corresponds to the difference in the requirements and functions of the baby and the calf.

The Mammary Gland as an Excretory Organ.—The mammary gland acts to some extent as an excretory organ, in the sense that waste nitrogenous substances filter through from the blood into the milk. Among these are the so-called non-protein nitrogenous extractives, amino-acids, urea, creatin, creatinin and uric acid. Milk may therefore occasionally become a vehicle of substances that are undesirable as a dietary ingredient. Every efficient physiologic mechanism fails at times. Foreign proteins may occur in the milk, but in minute quantities, demonstrable only by anaphylactic reactions. More important is the excretion of drugs and foreign substances in milk.

The Excretion of Drugs in Milk.—The following drugs taken by the mouth have been found in the milk of nursing women: aspirin, iodine, mercury (calomel), arsenious acid, potassium bromide, and probably also hexamethylenamine, salicylic acid, and salicylates, ether, antipyrin, bromides, and many others; the list is very long. It is probable that opium, all volatile oils, purgative salts, and rhubarb are excreted to a certain extent in the milk. It is well known how readily the flavor of cow's milk is affected by turnips, garlic, wild onions, moldy hay and grain, or damaged ensilage. Fermented distillery waste gives a bad flavor and may also cause the secretion of small quantities of alcohol in the milk. The importance of these facts is self-evident. Cows in pastures sometimes feed on poisonous weeds, and these poisons may pass into the milk. In the production of certified milk, cows are never allowed to graze, but are given carefully selected feed. Certain substances, as ensilage, when fed to cows, cause a laxative property to appear in the milk, and thus it is possible to affect the baby through the feed of the cow. The color of milk is also influenced by the feed.

The Differences between Cow's Milk and Woman's Milk.—The following table from Rotch summarizes the principal points of differences between cow's milk and human milk.

Woman's Milk Directly from the Breast	Cow's Milk, Freshly Milked
Reaction, amphoteric (more alkaline than acid) ..	Amphoteric (more acid than alkaline)
PH values, 7.1 to 7.6.....	PH values, 6.5 to 6.8
Water, 87 to 88 per cent	86 to 87 per cent
Mineral matter, 0.20 per cent	0.70 per cent
Total solids, 13 to 12 per cent	14 to 13 per cent
Fats, 4.00 per cent (relatively poor in volatile glycerids)	4.00 per cent (relatively rich in volatile glycerids)
Milk sugar, 7.00 per cent	4.75 per cent
Proteins, 1.50 per cent	3.50 per cent
Caseinogen, $\frac{1}{3}$ to $\frac{1}{2}$ of the total proteins.....	2.66 per cent
Whey-products, $\frac{2}{3}$ to $\frac{1}{2}$ of the total proteins.....	0.84 per cent
Coagulable proteins, small proportionately	Large proportionately
Coagulation of proteins by acids and salts, with greater difficulty. Curds small and flocculent ...	With less difficulty, curds large and tenacious
Coagulation of proteins by rennet, does not coagulate readily	Coagulates readily
Action of gastric juice, proteins precipitated but easily dissolved in excess of the gastric juice ...	Proteins precipitated but dissolved less readily

The differences between these two milks are greater than the table indicates. While cow's milk may be modified to approximate woman's milk in composition, it can never be just the same or just as good for infants.

Cow's milk is more opaque than woman's milk, although the latter may contain a greater percentage of fat. This is due to the opacity of the calcium-casein, which is present in greater proportion in cow's milk. Cow's milk is faintly acid or amphoteric when freshly drawn, but ordinarily is distinctly acid in reaction when consumed. Woman's milk is amphoteric or alkaline.

There is three times as much protein in cow's milk as in woman's milk. The reason for this is obvious, when we recall that the ratio of the growth of the calf to that of the infant is about as two to one. Furthermore, the protein in cow's milk consists chiefly of casein (3.02 per cent) and little lactalbumin (0.53 per cent), while woman's milk contains 0.59 per cent of casein and 1.23 per cent lactalbumin. The sugar in the two milks varies greatly in amount, but not in kind. Cow's milk contains almost four times the amount of inorganic salts compared to woman's milk. Of more importance, the salts in cow's milk consist mainly of calcium and magnesium, while those in woman's milk consist mainly of potassium and sodium bases. These differences have an important bearing upon infant metabolism. There is no great difference in the average amount of fat in the two milks; however, both in woman's milk and in cow's milk the fat is the most variable constituent.

The importance of mother's milk for the baby cannot be emphasized too often. There is no adequate substitute. Breast nursing is best for the baby

and best for the mother. Mother's milk contains specific and useful antibodies, vitamins and doubtless other unknown qualities, inadequately represented in cow's milk. Cow's milk can be modified to resemble human milk, but there are essential differences that may have remote effects upon the soundness of the teeth, the resistance of the tissues, etc. Babies should be nursed at least six months unless there are contra-indications, and gradually weaned during the second six months.

The diet of the mother affects the quantity and quality of the milk. The lactating woman needs a generous diet, varied with vitamins, well balanced and with about one-third more calories. The nature of the diet even influences the quality of the proteins in the milk.¹⁰ The mother should lead a calm life, for emotional stress, fatigue and worry may affect the milk. She needs exercise, sunshine, plenty of rest and food for two.

Milk Standards.¹¹—The word "standard" used in this connection is not intended to imply excellence, but simply to express the lowest possible limit that the law permits for a pure or normal milk. There are at least four standards by which milk should be judged: (1) *physical standards*; specific gravity, temperature, taste, odor, etc.; (2) *chemical standards*; especially the percentage of fat and total solids; (3) *bacteriological standards*; the number of bacteria per cubic centimeter and absence of pathogens; (4) *sanitary standards* determined by veterinary and medical inspection. Standards have also been established for pasteurization, production, transportation and handling. All are necessary for the satisfactory control of a milk supply.

Cow's milk should not contain less than 8.5 per cent of solids not fat, and not less than 3.25 per cent of milk fat.¹²

It has been found an advantage to keep the butter-fat standard relatively high and the total solids at a minimum of 12 per cent. This allows 8.5 per cent for solids not fat, such as the proteins, milk, sugar, and inorganic salts. A 3.25 per cent butter-fat and a 12 per cent total solids is the minimum that should be allowed.

If the law recognizes a low standard for total solids, it permits manipulation of the milk, such, for example, as adding water. It also encourages the production of milk from inferior cows. High standards encourage good dairy methods, require good feed, and place a premium upon the better breeding of milch cows.

The determination of fats and total solids is used to detect skimming or watering; however, it is possible to skim milk or water it, within limits, without the possibility of detecting it through the fats and total solids.

If dependence is placed upon the total solids, mistakes may also occur.

¹⁰ *J. Agric. Research*, 1924, 29: 12.

¹¹ The subject is fully discussed in the Reports of the Commission on Milk Standards of the N. Y. Milk Committee, U. S. Public Health Reports, May 10, 1912, and Feb. 16, 1917.

¹² Recommendation of the Official Agricultural Chemists, and the Commission on Milk Standards, and adopted by the U. S. Department of Agriculture and a number of states and cities.

The total solids represent the proteins, fats, sugar, and inorganic salts. They may readily be tampered with.

Bacteriological standards usually adopted are: certified milk, not more than 10,000 bacteria per cubic centimeter; grade A milk (raw) should not have more than 10,000 bacteria per cubic centimeter; grade A (pasteurized) should not have more than 200,000 before pasteurization and less than 10,000 after pasteurization; market milk (grade B) should have not more than 1,000,000 per cubic centimeter before pasteurization, and less than 50,000 per cubic centimeter after pasteurization, and should not contain *B. coli* in 1 cubic centimeter.

Grades of Milk.—Milk varies greatly in sanitary quality and in nutritive value. These differences are not obvious to our unaided senses. Milk should therefore be graded just as other commodities, such as wheat, beef, fruit, coal, etc., are graded.

The grading of milk in accordance with a simple classification has great economic and sanitary importance. Such a system furnishes the purchaser with a ready method of knowing just what he is buying, and furthermore helps the farmer get a better price for a superior product.

The grades of milk recommended by the Commission on Milk Standards of the New York Milk Committee are:

GRADE A

Raw Milk.—Milk of this class shall come from cows free from disease as determined by tuberculin tests and physical examinations by a qualified veterinarian, and shall be produced and handled by employees free from disease as determined by medical inspection of a qualified physician, under sanitary conditions, such that the bacterial count shall not exceed 10,000 per cubic centimeter at the time of delivery to the consumer. It is recommended that dairies from which this supply is obtained shall score at least eighty on the United States Bureau of Animal Industry score card.

Pasteurized Milk.—Milk of this class shall come from cows free from disease as determined by physical examinations by a qualified veterinarian, and shall be produced and handled under sanitary conditions, such that the bacterial count at no time exceeds 200,000 per cubic centimeter. All milk of this class shall be pasteurized under official supervision, and the bacteria count shall not exceed 10,000 per cubic centimeter at the time of delivery to the consumer. It is recommended that dairies from which this supply is obtained shall score at least sixty-five on the United States Bureau of Animal Industry score card.

GRADE B

Milk of this class shall come from cows free from disease as determined by physical examinations, of which one each year shall be by a qualified veterinarian, and shall be produced and handled under sanitary conditions, such that the bacterial count at no time exceeds 1,000,000 per cubic centimeter. All

milk of this class shall be pasteurized under official supervision, and the bacterial count shall not exceed 50,000 per cubic centimeter when delivered to the consumer.

It is recommended that dairies producing grade B milk should be scored, and that the health departments or the controlling departments, whatever they may be, strive to bring these sources up as rapidly as possible.

GRADE C

Milk of this class shall come from cows free from disease, as determined by physical examinations, and shall include all milk that is produced under conditions such that the bacterial count is in excess of 1,000,000 per cubic centimeter.

All milk of this class shall be pasteurized, or heated to a higher temperature, and shall contain less than 50,000 bacteria per cubic centimeter when delivered to the consumer.

Whenever any large city or community finds it necessary, on account of the length of haul or other peculiar conditions, to allow the sale of grade C milk, its sale shall be surrounded by safeguards such as to insure the restriction of its use to cooking and manufacturing purposes.

These grades are gradually being adopted. Other grades are used, such as certified milk, inspected milk, market milk, etc.

There is a growing tendency to classify all milk into raw and pasteurized. This is the most significant classification from a sanitary standpoint.

Certified Milk.—Certified milk is milk produced under the supervision and direction of a medical milk commission. Certified milk is fresh, clean and unaltered. It is raw milk of good quality and uniform composition, obtained by cleanly methods from healthy cows. The term "certified milk" was coined by Henry L. Coit of Newark, New Jersey, who in 1892, needing good milk for his own baby, formulated a plan for the production of clean, fresh, good milk under the auspices of a medical milk commission.

The medical milk commission is appointed by the county medical society; in some places the commission is either organized by or associated with the health department. The commission should include at least five members to look after (a) the sanitation of the dairy, (b) the veterinary supervision of the herd, (c) the medical supervision of the employees, and (d) the chemical and bacteriological supervision of the milk.

The use of the term "certified milk" should be limited to milk produced in accordance with the requirements of the American Association of Medical Milk Commissions.¹³ The first requisite in the production of certified milk is to enlist the coöperation of a trustworthy dairyman who is willing to enter into a contract with the medical milk commission. In accordance with the

¹³ See annual reports of this Association; also *U. S. Pub. Health Rep.*, 1912, 27: 645; and Methods and Standards for the Production of Certified Milk, adopted by the American Association of Medical Milk Commissions, June 20-26, 1923.

terms of this contract, the dairyman binds himself to comply with the specifications set forth and in return his milk is certified. The certificates are the caps for the bottles which are furnished by the commission.

The dairies are subjected to periodic inspections, and the milk to frequent analyses. The cows producing certified milk must be free from tuberculosis, as shown by the tuberculin test and physical examination by a qualified veterinarian, and from all other communicable disease, and from all diseases and conditions whatsoever likely to deteriorate the milk. They must be housed in clean, properly ventilated stables of sanitary construction, and must be kept clean and properly fed and cared for. All persons who come in contact with the milk must exercise scrupulous cleanliness, and must not harbor the germs of typhoid fever, tuberculosis, diphtheria, or other infections liable to be conveyed to the milk. Milk must be drawn under all precautions necessary to avoid contamination, and must be immediately cooled to 45° F., placed in sterilized bottles, and kept at a temperature between 35° and 45° F. until delivered to the consumer. Pure water, as determined by chemical and bacteriological examination, is to be provided for use throughout the dairy-farm and dairy. Certified milk should not contain more than 10,000 bacteria per cubic centimeter, and should not be more than thirty-six hours old when delivered.

Certified milk is raw milk and, therefore, may convey the infectious agents of disease; in fact, this has happened. Such occasional danger may be guarded against by pasteurization.

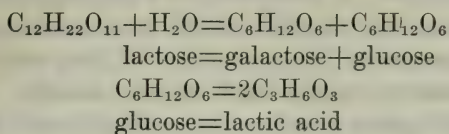
The Decomposition of Milk.—Milk spoils in various ways as the result of bacterial growth; the kind of decomposition depending upon the kind of bacteria which predominate. Milk, as a rule, ferments, but sometimes it putrefies. In the former case the main change takes place in the carbohydrates; in the latter the proteins are broken down. The fermentation, known as the souring of milk, is accompanied by an acid reaction and a precipitation of the casein. Putrid milk turns alkaline and bitter, owing to the formation of peptones. Sour milk is regarded as the "normal" form of decomposition, because it is the usual change and is not harmful. Putrid milk is believed at times to contain toxic substances; it is at least suspicious.

Sour Milk; Lactic Acid Fermentation.—Milk curdles or sours when the soluble caseinogen is precipitated as casein. The caseinogen exists in milk as a complex molecule containing calcium phosphate loosely bound to it; it also contains calcium as part of the molecular complex. The formula may be expressed thus:



The casein is held in solution (colloidal suspension) by the calcium phosphate and other soluble salts of calcium. Any chemical reaction that removes the calcium phosphate from this combination causes a precipitation of the caseinogen as casein. The casein may be precipitated by various substances, such as rennin or acids. In the normal curdling or souring of milk the casein is

precipitated by lactic acid produced through the action of bacteria upon lactose. The lactic acid results from hydrolysis of the lactose as follows:



The bacteria usually concerned in the souring of milk are: *B. acidi lactici* of Hueppe, *B. lactis acidi* of Leichmann, *Streptococcus lactis* of Kruse, *B. bulgaricus* of Metchnikoff, *B. coli*, and a great number of other microorganisms capable of fermenting sugar with the production of acid. Sour milk, obtained from clean milk, is a beneficial food.

Sour Milk and Intestinal Flora.—Sour milk contains myriads of lactic acid bacteria. Metchnikoff called attention to the importance of a normal lactic acid flora in the large intestines, which inhibits putrefactive processes and thereby stands guard against "auto-intoxication." He recommended the use of certain bacteria in sour milk, especially *B. bulgaricus*. It is a fallacy, however, to suppose that the flora of the intestines may be influenced through ingestion of these bacteria by the mouth, even when taken in enormous numbers, as in sour milk. A sour milk diet is uncertain in its effects and often disappointing in its results.

Contrary to widespread belief, the bacterial flora of the alimentary tract is not a replica of that of the food we eat. Of the many varieties of microorganisms gaining entrance into the alimentary tract, few succeed in establishing themselves. The diet of early infancy is rich in carbohydrates in the form of lactose. The *B. bifidus* then establishes itself in the intestines. As the diet changes with age, the colon group of bacteria begins to assert itself, not because these organisms dominate in the diet, but because they, above all others, thrive as well in a medium from which carbohydrates are absent, as in one containing them. *B. coli* and related bacilli constitute nearly 60 per cent of the viable fecal flora.

The number and kinds of bacteria vary in different parts of the digestive tube. The number becomes enormous in the large intestine. It is estimated that about 33×10^{12} are excreted daily by a normal adult. These bacteria supply 46 per cent of the total fecal nitrogen. It has even been questioned whether we could live without intestinal bacteria. Levin,¹⁴ however, found the intestines of a number of Arctic animals sterile. Loeb and Northrup¹⁵ have been able to raise large numbers of banana flies under sterile conditions. These experiments show that the duration of life of these flies varies inversely as the temperature of their environment.

When it was shown that the Bulgarian bacillus cannot be seeded in the

¹⁴ *Ann. l'Inst. Pasteur*, 1899, 13: 558.

¹⁵ *J. Biol. Chem.*, 1916, 27: 309; 1917, 32: 103; also *Proc. Nat. Acad. Sc.*, 1916, 2: 456.

intestines because it is not a normal inhabitant of man, in fact does not occur in the feces of Bulgarian peasants, attention was turned toward the closely allied *Bacillus acidophilus*. *B. acidophilus* is a normal inhabitant of the human alimentary tract, especially of children and some adults, where ordinarily it is found in small numbers. It is doubtful if they can be "implanted" or increased in numbers by taking them by the mouth. Living cultures in milk, reinforced with lactose, as advocated by Cheplin and Rettger, are claimed to relieve constipation and otherwise be beneficial especially by restraining putrefactive processes. However, indican and phenol excretion in the urine continue and may even increase with milk lactose cultures of *B. acidophilus*. Lactose alone is laxative. It is so poorly absorbed from the bowel that it gets into the lower ileum and colon and there modifies the culture medium so that the fermentative bacteria outgrow the putrefactive ones. *The intestinal flora are controlled by diet rather than by bacteria ingested*. Furthermore, the bacteria passed in the feces are not so much those taken in by the mouth as those that grow in the bowel. The true secret of acidophilic and other lactic acid therapy is not to emphasize the administration of the microbe, but to regard the proliferation of the microbe in the intestinal tract merely as an indication of a correct and successful regimen.

Putrid Milk.—*Alkaline Putrefaction.*—When boiled milk is allowed to stand at room temperature, it gradually acquires an alkaline reaction,¹⁶ a bitter taste, and finally curdles, yielding a soft, slimy curd. On further standing this curd is peptonized to form a somewhat clear fluid, and if these putrefactive changes are allowed to proceed for a sufficient length of time a semi-transparent liquid is obtained, having no resemblance to milk. In this form of decomposition the main change occurs in the protein constituent of the milk. These putrefactive changes seldom occur; they are undesirable and are believed sometimes to be dangerous, in that toxic substances may be produced. The principal cause of putrefaction in milk is the spore-bearing group of bacilli, belonging to and resembling the hay bacillus and also the anaërobes.

Slimy or Ropy Milk.—Under some circumstances certain mucilaginous substances develop in milk through abnormal fermentation. Slimy milk has been obtained of such viscosity that it could be drawn out into threads ten feet in length, and of such thinness as to be scarcely visible. In Norway such milk is esteemed a delicacy; in this country, however, it is objectionable. From a health standpoint ropy milk is not injurious unless it is slimy as a result of mucopurulent materials caused by diseased conditions in the mammary glands. The bacteria which produce ropy milk are widely distributed in nature. Of these *Bacillus lactis viscosi* (Adametz) is the commonest organism found in Europe, and a similar organism occurs in this country. *B. lactis viscosi* is very hardy; it may find its way into the milk through the water supply of the dairy, and then becomes widely diffused. It is sometimes very troublesome, but may be eradicated through cleanliness. Sometimes it is

¹⁶ Schorer found that such milk becomes less acid but seldom becomes actually alkaline in reaction.

necessary to resort to disinfection. Other organisms producing sliminess in milk are the *Micrococcus freudenreichii*, two forms of streptococci, and certain of the lactic acid bacteria.

Alcoholic Fermentation of Milk.—This is an unusual fermentation which sometimes occurs as a result of yeasts, aided in their action by certain species of bacteria. Alcoholic fermentation of milk seldom occurs spontaneously, but may be induced by direct inoculations with certain ferments, such as those employed in the production of kumyss and kefir.

Kumyss was originally made from mare's milk; it is now also made from cow's milk by the addition of cane sugar and yeast. *Kefir* is a similar beverage, originating in the Caucasus, where the fermentation is carried out in leather bottles and is started by means of "kefir grains" which contain yeast and various microorganisms.

Bitter Milk.—Freshly drawn milk sometimes has a bitter taste; in other instances milk acquires such a taste on standing a few hours. The former is due to improperly feeding the cow with such herbs as lupines, wormwood, raw Swedish turnips, cabbages, etc. The latter case is due to the growth of certain bacteria in the milk after it is drawn. Conn ascribes the power of ruining the taste of freshly drawn milk in a few hours to a micrococcus, while Weigmann thinks a bacillus is responsible. The condition is undesirable, and sometimes causes much trouble for the dairyman, but it has no particular sanitary significance.

Colored Milk.—Blue milk is usually due to the *Bacillus cyanogenus*. Such milk is apparently harmless. Red milk may be due to the presence of blood coming from an injury, or acute infection of the udder. Sometimes it results from the feeding of the cow on plants containing red pigment, such as the madder root. A red color may also be produced by the *B. erythrogenes*, *B. prodigiosus* and sarcinæ. Red milk caused through the agency of bacteria is not known to be harmful.

Adulterations of Milk.—*Skimming.*—The removal of part or all of the cream and selling the remaining fluid as whole milk is an economic fraud, and has no reference to health, except that the milk is correspondingly lowered in nutritive value. Adding skim milk is also a form of adulteration difficult to detect.

Watering.—The practice of watering is not nearly so frequent as formerly. If the water be pure it must be regarded more as a fraud than a health problem. The addition of water to milk may be detected because it lowers its specific gravity, raises its freezing point, and lowers its index of refraction and also its viscosity.

Thickening agents, such as chalk, calves' brains, and glycerin, have never been common practices. Gelatin or lime is sometimes used to thicken cream. Cream may also be thickened by homogenizing it. *Coloring matter* is sometimes added with the object of concealing skimming or watering or to make the milk look richer. Annatto, a vegetable dye, is most commonly used; orange and yellow azo coal tar are also used. *Alkalies*, such as sodium carbonate or

bicarbonate, are occasionally added to milk to reduce its acidity or to improve its taste or to delay curdling. *Sweet substances*, such as saccharin or sugar, are occasionally added to milk, either to raise the specific gravity and thus disguise watering, or to disguise the sour taste of milk just on the turn.

Chemical Preservatives.—Chemical preservatives, such as borax and boric acid, salicylic acid, benzoic acid and benzoates, potassium bichromate, peroxid of hydrogen, fluorids, formaldehyd, and others, have from time to time been used in milk. The oxidizing preservatives injure the vitamins, and the alkalies camouflage acid fermentation. The practice of adding any chemical preservative to milk meets with the unqualified disapproval of the sanitarian. Almost all countries prohibit the use of such foreign substances. The only proper preservatives for milk are *cleanliness* and *cold*.

Dirty Milk; The Dirt Test.—Practically all milk contains more or less dirt. For the most part, this dirt consists of cow feces. The presence of dirt may best be determined by filtering a pint of milk through a little disk of absorbent cotton. This produces a stain varying in intensity from a yellowish to a brownish or black spot. A Gooch crucible, a Lorenz apparatus, or simply an ordinary funnel may be used to filter the milk. Warm milk filters much more readily than cold milk. This simple test is one of the practical routine tests used for the administrative control of milk supplies. The intensity of the stain and the amount of deposit upon the cotton is a tell-tale which appeals strongly to farmers and dairymen, as well as to consumers. It is a good practice to send these disks of cotton, with a letter, to the farmer, showing him the amount of dirt contained in his milk. The disks may be dried and kept with the records of the health office.

It should be remembered that milk that has been "clarified" or strained will not show the dirt test.

Clarification.—Clarification consists in mechanical straining by the use of centrifugal force. Clarifiers of the de Sival type whirl the milk with sufficient force to throw the heavier substances to the circumference of the revolving bowl where they become attached while the milk serum with its fat and normal solids escape through the central outlet. Milk so treated therefore will not deposit a sediment on standing, and the value of the dirt test is destroyed. The material adhering to the circumference of the bowl is called "*separator slime*" and consists of dirt, foreign particles of all sorts, bacteria, pus, blood, mucus, leukocytes, epithelial cells and cell detritus, and a small amount of the substances normally present in milk.

The advantages of clarification are that it is more efficient in removing visible dirt than filtration through a strainer. But, it is not a substitute for sanitation, since it does not remove soluble dirt, nor is it a substitute for pasteurization since it does not effectively remove pathogenic bacteria. It breaks up clumps of bacteria and by distributing them tends to increase the bacterial count of raw milk. The merits of the process have often been abused by dealers who have made exaggerated claims and mislead the public with a false sense of security.

Number of Bacteria in Milk.—As a rule, milk contains relatively and actually more bacteria than any other article of diet. Milk may contain more bacteria than any other known substance; in fact, many more than are found in sewage. Mere numbers, however, need not alarm us, for it is the kind that most concerns us. By universal consent, however, milk containing an excessive number of miscellaneous bacteria is not suitable for infant feeding. If milk were a transparent fluid, the enormous growth of microorganisms present in average market milk would be plainly visible to the naked eye.

The bacteria get into the milk from a number of different sources. Some of them are in the milk before it leaves the udder. They grow up the milk ducts into the milk cistern; hence, the fore-milk contains more than the mid-milk or strippings. It is practically impossible to obtain sterile milk directly from the teat in any large quantity. As soon as the milk leaves the teat it receives additional contamination from all objects with which it comes in contact, as the hands, the pail, the dust in the air, etc.

The most important factors to obtain milk with a low bacterial count are: clean and sterilized pails, cans and other utensils; clean, healthy cows with clean udders and teats; and the use of the small top milk pail. Experiments have shown that the greatest numbers of bacteria come from the dirt and dung that falls into the pail during milking and from the pail itself, if not clean and scalded. To keep the counts low, it is necessary to chill the milk at once and to hold it at about 40° F.

It has been repeatedly demonstrated that the rank and file of dairy farmers in their ordinary cow stables can, by the practice of elementary sanitary methods, produce milk which regularly contains less than 10,000 bacteria per cubic centimeter. This requires not only intelligence, but also conscientiousness.

Judged by the number of colonies on Petri plates, the number of bacteria in milk increases every time it is handled or exposed in any way. Separator milk seems to contain more bacteria than the original milk. The same is true of filtered milk. This is due to the fact that while some of the dirt is taken out, the particles are broken up and the bacteria dispersed throughout the fluid, thereby giving more colonies on plate cultures.

Garget, or inflammation of the udder, is a very common affection of cows, and is associated with streptococci, staphylococci, lactic acid and colon bacilli, etc. Milk from a gargety udder will contain enormous numbers of microorganisms.

When milk contains bacteria harmful to man, they almost always get into the milk from human sources, either directly or indirectly. The chief exception to this rule is the bovine tubercle bacillus and the micrococcus of Malta fever.

The bacteria in milk are not equally distributed throughout the fluid. There are more bacteria in cream than in the underlying skim milk—particularly in gravity cream. As the cream rises it mechanically carries the bacteria along with it, very much as a snowstorm sweeps the atmosphere. Milk

formulae for infant feeding are often made of top milk, which, however, may contain from five to one hundred times the number of bacteria per cubic centimeter found in the whole milk. In twenty-six samples of milk Anderson found the gravity cream contained about four times as many bacteria as the sediment layer, and about one-third as many as the whole milk. Schorer found that the cream from milk of high bacterial count contained several thousand times as many bacteria as the underlying skim milk.

Certified milk should not contain over 10,000 bacteria per cubic centimeter; grade A not over 200,000 before, and not over 10,000 after pasteurization; grade B milk not over 1,000,000 before and not over 50,000 after pasteur-



FIG. 57.—A DARK, POORLY VENTILATED COW SHED, DIFFICULT TO KEEP CLEAN.

ization. New York has placed the limit at 1,000,000 per cubic centimeter. Even this standard, however, has not been rigidly enforced. Boston has a standard of 500,000; Rochester 100,000.

In Washington, in 1908, the average bacterial count of the market milk was 22,000,000 per cubic centimeter, as found in many hundreds of samples of the city supply. In 1909, the average was reduced to 11,000,000.

Excessive numbers of bacteria in milk indicate that it is dirty, old, or warm. One or any combination of these factors favors a rapid growth and multiplication of the bacteria in milk.

The number of bacteria in milk is the best single index we have of its general sanitary character.

Pathogenic Bacteria in Milk.—For the most part, bacteria do not pass a healthy udder. However, we can place no trust in the filtering ability of the mammary gland. It is known that the virus of foot-and-mouth disease, which is ultramicroscopic, and the virus of Malta fever (*Brucella melitensis*), and also the virus of milk-sickness are almost constantly found in the milk of

affected animals. On the other hand, tubercle, anthrax and other large bacilli do not pass the mammary gland unless there is a lesion of the udder.

For the most part, when milk contains bacteria pathogenic for man, the milk is contaminated from human sources. This is almost invariably the case with diphtheria, scarlet fever, septic sore throat and typhoid fever. Occasionally, however, the udder may become infected with bacteria pathogenic for man without affecting the cow. For example, *Streptococcus epidemicus*, the cause of septic sore throat, may get into the udder where it grows and multiplies and may thus infect the milk for a month or more. In this case, however, the original infection is introduced from human sources (see page 722). The hemolytic streptococci, β type, ordinarily found in milk are not known to be pathogenic for man.

Bacteria in Human Milk.—Investigation of the breast milk of one hundred women¹⁷ disclosed streptococci in 49 per cent of the samples. Only 2 per cent of these were regarded as an indication of a pathologic process in the breast, although the number of streptococci in milk with a high polymorphonuclear count was greater than in normal milk. *Bacillus coli* and staphylococci, especially *Staphylococcus albus*, were present in normal milk from healthy glands. The bacteriology of mother's milk has received scant attention, but it is now plain that it may be significant in the problem of infant feeding.

The Germicidal Property of Milk.—The germicidal property of milk has been much misunderstood. It is specific and varies in different animals and even in the same animals at different seasons.¹⁸ It is destroyed at 75° C. for fifteen minutes, or between 80 and 90° C. for two minutes. At most, the germicidal action is feeble and transitory and cannot take the place of cleanliness and ice, but may be taken advantage of by prompt use of fresh milk. It is true that bacteria develop more quickly in heated milk than raw milk, provided the raw milk is fresh; it should be remembered, however, that stale milk, even milk that is a day old no longer possesses this restraining action. The germicidal property is, therefore, ordinarily absent in market milk.

Rosenau and McCoy¹⁹ have shown that judged by the number of colonies that develop upon agar plates, the bacteria in milk first diminish, then increase in number. This occurs only in raw milk during the first eight or twelve hours after it is drawn. Although the bacteria seemingly decrease in numbers, they never entirely disappear. After the initial decrease there is a continuous and rapid increase, until the milk contains almost infinite numbers in each cubic centimeter. The power of milk to restrain the development of bacteria lasts from six to twenty-four hours, depending upon the temperature at which the milk is kept. When the milk is kept warm, 37° C., the decrease is pronounced within the first eight or ten hours; after this the milk has entirely lost its restraining action. When the milk is kept cool, 15° C., the de-

¹⁷ *J. Hyg.*, 1924, 23: 64.

¹⁸ *Brit. J. Exper. Path.*, 1924, 5: 271.

¹⁹ *U. S. Hyg. Lab. Bull.*, No. 56, 1909.

crease is less marked but more prolonged. They further showed that at least part of this decrease is due to agglutination.

DISEASES SPREAD BY MILK

The diseases conveyed through milk are: tuberculosis, typhoid and paratyphoid fevers, diphtheria, scarlet fever, septic sore throat, Malta fever, foot-and-mouth disease, and milk-sickness; also some of the summer complaints of children, and the diarrheal and dysenteric diseases of adults, which are often referable to infected milk. Epidemic arthritic erythema and infantile paralysis have recently been added to this list.

As a rule, milk becomes infected from human sources, sometimes on the farm, sometimes at the dairy, sometimes in transportation, and occasionally in the household. Sometimes the milk becomes infected as a result of disease of the animal, as in the case of bovine tuberculosis, Malta fever, foot-and-mouth disease, streptococcal garget, etc.

When all the facts are brought together they make a strong indictment against raw milk. Thus, the following official record ²⁰ of milk-borne infections in Massachusetts tells the story.

	Total Cases Reported	Number of Cases Traced to Milk	Cases Traced to Milk, per Cent	Number of Outbreaks Traced to Milk	Outbreaks Traced to Milk, per Cent
<i>Typhoid Fever</i>					
1907-1915	23,482	2,215	9.4	50	69.4
1915-1918	6,331	496	7.8	16	57.1
1919-1923	4,105	297	7.2	12	70.6
TOTAL	33,918	3,008	8.86	78	66.8
<i>Septic Sore Throat</i>					
1907-1915*	2,512	7	9.7
1915-1918	1,401	867	61.9	7	25.0
1919-1923	829	118	14.2	3	17.6
TOTAL	2,230	3,497	?	17	14.6
<i>Scarlet Fever</i>					
1907-1915	70,569	2,747	3.9	10	13.9
1915-1918	25,328	140	.6	4	14.3
1919-1923	46,777	53	.1	1	5.9
TOTAL	142,674	2,940	2.06	15	12.8
<i>Diphtheria</i>					
1907-1915	69,646	131	0.2	5	6.9
1915-1918	33,807	30	0.1	1	3.6
1919-1923	42,386	8	0.02	1	5.9
TOTAL	145,839	169	0.116	7	6.0

* Not reported until 1915

Since the general adoption of pasteurization for the milk supplies of the

²⁰ *Am. J. Pub. Health*, 1924, 14: 963.

large cities in Massachusetts, milk-borne outbreaks occur characteristically in the small unpasteurized supplies.

In addition to the specific diseases, milk may be injurious as a result of other causes. Thus, Le Blanc has pointed out that the milk of cows in heat may cause gastro-intestinal disturbances. The toxic effects of milk and milk products of nymphomalous cows are even more marked. Milk should not be used within fifteen days of parturition. The requirement for certified milk is placed at thirty days before and fifteen days after. Such milk is apt to produce diarrhea, colic, and other digestive disturbances. Milk may further be harmful as a result of such diseases as mastitis or garget, gastro-enteritis, septic and febrile conditions of the cow.

Tuberculosis.—Milk is the chief vector for the conveyance of bovine tubercle bacilli from cow to man. They get into milk either directly as a result of tuberculosis of the udder, which occurs in from 1 to 2 per cent of all tubercular cows, or indirectly through cow manure. In the latter case the tubercle bacilli are coughed up, swallowed, and passed in the feces. Practically all market milk contains cow feces. Tuberculosis in cattle is very prevalent. In Holland nearly one-tenth of all cattle killed for food are tuberculous; in Berlin, 16 per cent; in Saxony, 30 per cent; in Pennsylvania, from 2 to 3 per cent. The "milk" from a tuberculous udder, when examined under the microscope, may contain as many tubercle bacilli as are ordinarily found in tuberculous sputum. The milk from a tuberculous udder of one cow may contain sufficient bacilli seriously to infect the mixed milk of twenty-five or thirty cows. In one case Ostertag found that 0.001 cubic centimeters of the secretion from a tuberculous udder was sufficient to cause tuberculosis in a guinea-pig. In such a case a child would receive myriads in a gill.

Tonney examined the market milk of Chicago in 1910 for the presence of tubercle bacilli. In 10.5 per cent of 144 samples of raw milk he found tubercle bacilli in sufficient numbers to infect guinea-pigs. Of nineteen samples of pasteurized milk examined, none contained tubercle bacilli.

Hess in 1909 examined 107 samples of market milk in New York City, with the result that seventeen of them, or 16 per cent, were found to contain tubercle bacilli.

Anderson examined 223 samples taken in the city of Washington, and reported sixteen, or 6.72 per cent, as positive. The tests made by the Bureau of Animal Industry of the milk in Washington disclosed 7.7 per cent infected. Goler reports about 5 per cent of the milk supply of Rochester, New York, infected.

To sum up, we have evidence from four typical American cities. A total of 551 samples of milk have been examined, in which tubercle bacilli were found in forty-six, making a percentage of 8.3. This may be taken as representative for the entire country.

Sheridan Delepine²¹ reports that the mixed milk of Manchester, England,

²¹ *J. State Med. Rep.*, November, December, 1914.

collected at railway stations or other places than the farm, contained tubercle bacilli in samples examined in the following inclusive periods:

1897-1899	17.2 per cent
1900-1904	10.3 per cent
1905-1909	6.8 per cent
1910-1913	9.0 per cent

Wherever these investigations have been carried out similar and sometimes higher results have been obtained, both in Europe and in this country. It is believed that the figures are an underestimate, for the methods used in the laboratory are not sufficiently delicate to detect a few tubercle bacilli in milk. Unless these microorganisms are present in considerable numbers, they are apt to escape detection. In any event, it is clear that the common market milk furnished all large cities and probably most small towns very often contains tubercle bacilli.

Mohler, Washburn, and Doane found tubercle bacilli to live a year and more in cheese 220 days old. In these experiments the cheese was purposely infected and fed or inoculated into guinea-pigs at various times. Tubercle bacilli are frequently found in butter and other milk products, especially if the milk or cream of which they are made has not been pasteurized.

The relation of bovine tuberculosis to man is considered on page 160.

Method of Detecting Tubercle Bacilli in Milk.—It is not a simple matter to discover tubercle bacilli in milk, butter, and similar products. Direct microscopic examination for acid-fast bacilli is not satisfactory, because ordinarily there are comparatively few tubercle bacilli in mixed market milk, and furthermore, many acid-fast microorganisms other than tubercle bacilli may occur in milk.

It is not a simple task to isolate tubercle bacilli in pure culture, because the enormous number of other saprophytic microorganisms overgrow the cultures.

It therefore becomes necessary to resort to animal experimentation in order to detect and isolate tubercle bacilli. The guinea-pig is the most susceptible and suitable animal for this purpose. The material under examination may be injected subcutaneously or intraperitoneally. A number of animals should be inoculated with the sample in question for the reason that a certain proportion of them will succumb to acute infections, especially streptococci or members of the hemorrhagic septicemic group, which are often found in milk. For this reason, it is advisable to inject variable amounts for the purposes of the test.

The tubercle bacilli in milk may be first concentrated by centrifugation. Some of them will fall to the bottom with the sediment, others will rise with the cream, being enmeshed and carried by the fat globules. The cream and sediment may then be injected into guinea-pigs, either separately or mixed. It should be remembered that this process not only concentrates the tubercle bacilli, but other microorganisms that may be in the milk.

The sediment may first be treated with antiformin, which destroys many of the frailer cocci and bacilli, but spares the tubercle bacilli. Antiformin is a strong alkaline solution of chlorinated soda (see page 1363).

The guinea-pigs that survive the early infections that contaminate the milk should be watched for symptoms of tuberculosis—enlarged glands, loss of weight and fever. Those that do not die in two months should be tested with tuberculin. Inject 2 cubic centimeters of Koch's old tuberculin subcutaneously. This must be diluted back to the original strength of the bouillon culture. If the guinea-pig is tuberculous it will die within twenty-four hours—usually in from eight to sixteen—with characteristic lesions and reaction about the tuberculous foci.

Typhoid Fever.—Of milk-borne epidemics, typhoid fever takes the lead. Typhoid bacilli may swarm in milk without altering its taste, odor, or appearance. In Washington 10 per cent of all the cases of typhoid fever during the four years 1907 to 1910 were traced to milk. The recent figures for Massachusetts are given in the table on page 718. Note that despite the improvement in the number of cases, the percentage of cases due to infected milk remains about the same. All outbreaks occur on raw milk routes. The milk may become infected by a convalescent, a carrier, or a missed case.

Bolduan estimates that from three hundred to four hundred cases of typhoid fever each year come in contact with the milk supplied New York City. He further states that "the startling total of 90 to 120 typhoid carriers now probably menace the milk supply of this city." This estimate is based upon the fact that about 200,000 persons come into more or less contact with the milk from over 40,000 dairy farms (see Typhoid Fever, page 111).

Typhoid fever has also been traced to cream, ice cream, and other milk products.

Milk-borne outbreaks of *paratyphoid fever* have been described by Levine and Eberson,²² Williams²³ and others.

Scarlet Fever.—Milk-borne outbreaks of scarlet fever are sometimes extensive and serious. The milk is practically always infected from human sources. So far as known, cows do not have scarlet fever. There is a suspicion, however, that some streptococcal infections of the cow may reproduce a disease resembling scarlet fever in man.

Two outbreaks of scarlet fever due to ice cream have been reported. One took place in South Kensington, England, in 1875, following a large dinner where the dessert was frozen pudding.²⁴ A clear-cut outbreak due to ice cream occurred in Flint, Michigan, in July and August, 1924, involving forty-one cases extending over seven days. The ice cream was infected by the maker, who had a mild case of scarlet fever.²⁵ (See Scarlet Fever, pages 218 and 225.)

Diphtheria.—Diphtheria bacilli in milk practically always come from human sources, either cases or carriers. In a few rare instances ulcers upon

²² *J. Infect. Dis.*, 1916, 18: 143.

²⁴ *Rep. Loc. Govt. Bd., Suppl.*, 1875, 72.

²³ *J. Am. M. Ass.*, 1925, 84: 251.

²⁵ *Am. J. Hyg.*, 1925, 5: 669.

the teat of the cow have become infected with diphtheria, and the bacilli are thus transferred to the milk. Such an occurrence, however, is unusual. As a rule, diphtheria outbreaks caused by infected milk are more limited both as to numbers and area than milk-borne outbreaks of typhoid or scarlet fever (see Diphtheria, page 190).

Septic Sore Throat.—The first milk-borne outbreak of “septic sore throat” recognized in this country occurred in and about Boston in May, 1911. Since then similar outbreaks have occurred in Baltimore; Concord, New Hampshire; Chicago, and elsewhere. The infection has spread over the country.

Septic sore throat due to infected milk has been well known in Great Britain for many years. Swithinbank and Newman state that a year never goes by in which there are not outbreaks of sore throat or tonsillitis due to milk or cream. The infection usually gets into the milk from human sources, although it is suspected that some streptococci associated with diseases of the udder may be pathogenic for man.

Theobald Smith and J. H. Brown²⁶ have shown that the responsible organism is *Streptococcus epidemicus*²⁷ and furthermore disclose a difference between human (*epidemicus*) and bovine (β type) streptococci.

The bovine streptococci produce garget in cows but do not cause tonsillitis in men; on the other hand, the human streptococci produce sore throat in man but have slight pathogenicity for cows.

Smith and Brown studied the streptococci from five milk-borne epidemics at first hand, together with cultures from the big epidemics of Chicago, Baltimore and Boston. From this work it is now clear that septic sore throat in man is of human origin, even when the disease is contracted through milk infected in the udder, for it was found that while the human streptococcus is but slightly pathogenic for cows, this organism may become implanted in the udder. This may take place through milking, wiping with infected cloths, through passing quills up the milk ducts and in similar ways. When this takes place, garget does not ensue but the streptococcus becomes seeded in the udder and may remain for six weeks or longer. In other words the cow may become a “carrier” of the human streptococcus, thus explaining why milk-borne outbreaks of septic sore throat are sometimes long drawn out.

The hemolytic streptococci found in milk show distinct cultural and biological differences from *Streptococcus epidemicus*. No single test, however, can be relied upon to differentiate them. Brown, Frost and Shaw²⁸ found hemolytic streptococci in considerable numbers in the certified milk of five dairy herds. These streptococci belonged to several distinct cultural and serological groups. There is no evidence that any of them, whether from gargety cows or from mixed milk are pathogenic for man. Therefore, to justify condemnation of the milk supply, the streptococcus found should correspond in all respects with the characteristics of *Streptococcus epidemicus*.

²⁶ *J. Med. Research*, 1915, 31: 455; also W. G. Smillie, *J. Infect. Dis.*, 1917, 21: 45.

²⁷ *J. Infect. Dis.*, 1926, 36: 381.

²⁸ *Ibid.*

The *disease* often presents a severe clinical type and may result in death. Apparently it is not readily communicable from person to person. The inflammation and swelling of the tonsils and of lymph glands of the neck and of the mucous membranes are more severe than ordinarily; edema is a feature, and many cases present pseudomembranous formation and other indications of a virulent infection. There is a sharp febrile reaction, prostration, and sometimes delirium. The duration of the disease may be prolonged, and complications occur in about one-quarter of the cases. These consist mostly of enlarged regional lymph-nodes, which may suppurate; abscesses, arthritis, endocarditis, peritonitis, erysipelas, pneumonia, pyemia, acute nephritis, otitis, and other sequelæ indicating the invasion of the blood with a virulent streptococcus.

The *Boston outbreak*, in 1911, was characterized by its extraordinary virulence and comparative immunity of children, and high mortality among the aged and infirm. In this outbreak there were over 2,000 cases with about forty-eight deaths. One of the features of special interest was that the milk incriminated had always been a particularly clean, fresh, and satisfactory supply. It was obtained from tuberculin-tested cows under veterinary supervision; and the milk itself subjected to frequent chemical and bacteriological tests. The milk was bottled at the dairy, the bottles were sterilized, and many extra precautions were taken to ensure its cleanliness. For twenty-eight years not a breath of suspicion was attached to this milk until this catastrophe occurred. It emphasizes the lesson that raw milk is apt to be dangerous milk, and our only protection against these particular dangers is through pasteurization.²⁹

Another instructive outbreak, reported by Benson and Sears,³⁰ occurred in Portland, Oregon, March 24 to 31, 1922. In this epidemic there were 487 cases and twenty-two deaths. "Almost all of the cases and all but one of the deaths occurred among people who had drunk raw milk from one dairy which had been rated as one of the best in the city." This is the common story.

Milk-Sickness.—Milk-sickness is an acute non-febrile disease due to the ingestion of milk, milk products, or the flesh of animals suffering from a disease known as trembles. The disease is characterized by great depression, persistent vomiting, obstinate constipation, and high mortality. The picture is that of a poisoning rather than an infection.

Milk-sickness is primarily a disease of cattle, secondarily of man. In cattle it is called slows or trembles, and formerly was prevalent in the central part of the United States. The pioneers suffered severely in winning the West. Nancy Hanks, the mother of Lincoln, died from the disease in 1818 after an illness of a week. As forests are cleared and pastures fenced the disease becomes less frequent. It is still met with in the valley of the Pecos River, New Mexico, in parts of Tennessee and North Carolina.

²⁹ For a more detailed study of this and other milk-borne outbreaks, see *The Milk Question*, by M. J. Rosenau.

³⁰ *J. Am. M. Ass.*, 1923, 80: 1608.

The *cause* of the disease has been obscure. Alkali drinking water has been accused; pathogenic microorganisms have been described; and poisonous plants have been suspected.

Marsh³¹ studied the rayless goldenrod, *Aplopappus heterophyllus*, and concludes that milk-sickness is a poisoning due to this plant. He produced trembles or alkali disease in horses, cattle and sheep by feeding sufficient quantities of the rayless goldenrod. The toxic principle is excreted in the milk, and calves and lambs may be poisoned in this manner. Stock eat this weed only where there is little good forage. The obvious remedy is to see that the animals are well fed. It is entirely practical to dig out the weed in fenced pastures.

Malta Fever.—Malta fever is a disease primarily of goats; secondarily of man. The infection is transmitted from goats to man through raw milk containing *Brucella melitensis* (see page 407).

Recently it has been shown that *contagious abortion* of cows is due to the *Bacillus abortus*, which may contaminate milk; it is pathogenic for many animals, but there is no evidence that it is harmful to man. Schroeder and Cotton found this bacillus in eight out of twenty-seven samples of market milk tested. The extraordinary relation between these two diseases is discussed under Malta fever, page 408.

Foot-and-Mouth Disease.—Foot-and-mouth disease is an infection primarily of cattle and secondarily of man. It is caused by a filtrable virus, and is noteworthy for being the first ultramicroscopic virus to be discovered (Löffler and Frosch, 1898). The infection is transmitted to man through the ingestion of raw milk, buttermilk, cheese, or whey from diseased cows. Children are not infrequently infected by drinking unboiled milk when the disease is prevalent in the neighborhood. In man the disease is mild. The symptoms resemble those observed in animals: there is fever, sometimes vomiting, painful swallowing, heat and dryness of the mouth, followed by an eruption of vesicles in the buccal and mucous membranes, and very rarely by similar ones on the fingers. The vesicles are about the size of a pea; they soon break, leaving small erosions, which rapidly heal. The disease is seldom fatal except occasionally in very weak children (see page 404).

The Diarrheal Infections.—Milk is responsible for gastro-intestinal troubles, some of which are specific.

Bacillus enteritidis and its congeners grow well in milk and are the cause of milk-borne outbreaks of *food infection*. Fresh milk products are also responsible (see page 645).

Dysentery has been traced to milk in a number of instances. *B. dysenteriae* grows well in milk, which is a common cause of infantile diarrheas.

Milk is usually too acid for the vibrio of *cholera*.

One of the chief causes of the high infant mortality is *summer diarrheas*, but even these are not all due to stale, dirty, and bacteria-laden milk. Many

³¹ *Bull. Bur. An. Ond.*, U. S. Dept. Agric., No. 1391, May, 1926.

of the diarrheal diseases of infancy are true cases of bacillary dysentery, which is transmitted in a great variety of ways. However, the improvement in the milk supply for babies has directly, and in large part indirectly, resulted in a decrease in infant mortality in recent years (see page 505).

All the above infections may also be conveyed in fresh milk products.

Epidemic Arthritic Erythema.—An outbreak of this disease occurred at Haverhill, Massachusetts, in January, 1926, involving eighty cases with no deaths. It was studied by Place, Sutton and Willner.³² The disease resembles dengue clinically, has a sharp onset with a chill, fever, headache and toxic manifestations. The eruption resembles that of dengue, is measly or rubella-form, and affects especially the extremities. Pain and swelling in the larger joints are a common feature. The period of incubation was two to three days. All except one of the cases drank raw milk from the same dairyman. The source of the infection was not traced. Place and his associates have isolated a Gram-negative bacillus, non-spore-bearing, which seems to be the cause of the trouble.

A similar outbreak occurred during May-June, 1925, at Chester, Pennsylvania, involving four hundred cases with no deaths. This epidemic was first diagnosed as dengue.³³

Infantile Paralysis.—To this long list of milk-borne diseases must now be added infantile paralysis. Two small outbreaks, one at Cortland, New York, and the other at Spring Valley, New York, have been described, which seem attributable to milk infected by a case of the disease during the acute stage on the dairy farm (see page 389).

The Character of Milk-borne Epidemics.—Milk-borne epidemics usually have an explosive onset, rise to a peak, and decline gradually. The character of the curve depends upon the amount of infection in the milk, and the manner of its distribution, the number of persons who drink it, and other factors. If the infection in the milk is dilute or attenuated, the disease crops out among a few susceptible persons who drink it. If the infection is concentrated and the milk is widely used, the curve of the outbreak will have the steeple-like character of a water-borne epidemic. The length of the epidemic varies with the period of incubation of the disease and with the length of time the milk is infected. The number of people involved may vary from a few to a hundred or several thousand. Only a single bottle of milk may be infected, and thus convey the disease to only one person; on the other hand, many gallons of mixed dairy milk may become infected and produce disease in many hundred persons. As a rule, milk outbreaks last a comparatively short time, and extend over a circumscribed area, as the disease follows the milk wagon. At first the disease occurs almost exclusively among users of the infected milk. Afterward secondary cases may occur.

The disease shows a special incidence among milk drinkers. It is interest-

³² *Boston M. & S. J.*, 1926, 194: 285.

³³ Report (unpublished) by Charles Armstrong, U. S. Public Health Service, July 18, 1925.

ing to note that sometimes only one person of a number living in the same house is attacked, and such a one is a person who drinks the milk raw.

Milk-borne diseases often attack those living under the best sanitary conditions. The reason for this is that such people drink milk more freely than the poor. Milk outbreaks among the well-to-do are unnecessary tragedies to the sanitarian. Many milk outbreaks show a greater incidence of the disease among women and children, who are usually credited with drinking more milk than men. Sometimes adults are attacked, and sometimes the young. The character of milk-borne outbreaks varies with the disease and other circumstances.

There is apt to be a short period of incubation, probably on account of the concentration and large amount of the infection; however, the disease may be mild or virulent. Multiple cases occur simultaneously in the same house. Such an occurrence is very suggestive to the epidemiologist, and frequently gives him the first hint of an impending milk epidemic.

Milk-borne outbreaks of disease are always due to raw milk: often milk of good quality, even certified milk. *There is no record of a milk-borne outbreak attributable to properly pasteurized milk.*

MILK PRODUCTS

Adjusted or Standardized Milk.—The addition or subtraction of cream with the object of obtaining a uniform and definite percentage of fat is known as adjusting or standardizing milk. Holstein and Jersey milks are mixed so as to adjust the fat content. Not infrequently the cream is removed by manipulation and mixing so as to reduce the fat to the minimum legal limit. A more commendable practice is to fortify the fat content by separating the cream from a portion of the milk and adding this to the general supply.

Adjusting and standardizing is a common practice among large milk dealers. Sanitarians have always looked askance upon the practice, for while the addition of cream can hardly be objected to, its subtraction cannot be approved. The process should be controlled by the health authorities and such milk distinctly labeled as to its modification.

Reconstructed Milk.—Reconstructed milk is also called remade milk; sometimes "synthetic" milk. A better name is recombined milk, or reconstituted milk, for it consists of combining powdered whole milk or skim milk powder, condensed or evaporated whole milk or skim milk, with butter or milk fat and water. Machines may now be had for mixing, recombining or emulsifying these materials for the production of reconstructed milk or cream. If such products are made entirely from milk constituents, they may be labeled "recombined," or "reconstructed" milk, but if any other fat is substituted in whole or in part for milk fat, then the product should be labeled "artificial milk," "milk substitute," or "filled milk."

There is a legitimate field for remade milk. Thus, during the World War it became necessary to find a milk supply for the new city of Nitro, West

Virginia, with 25,000 and more inhabitants.³⁴ The city was established in a section unsuited for dairying and no available supply of fresh milk could be found. The Government solved the problem by reconstructing milk by homogenizing butter fat and mixing this into skim milk powder and water.

Filled milk is a compound made up of skimmed milk to which 3 or 4 per cent of coconut oil is added. It is then condensed to approximately one-half its volume.

The importance of sanitary control and proper labeling of these products is obvious.

Condensed and evaporated milks are concentrated by partial drying. The first really practical method was devised by Gail Borden, of White Plains, New York, who successfully evaporated milk under reduced pressure, and in 1856 obtained a patent for his process.

Condensed milk is heated to about the pasteurizing temperature but is not sterilized. It is preserved with 40 per cent sugar (sucrose). When not produced under sanitary conditions, it may contain millions of bacteria per cubic centimeter. Condensed milk should contain at least 28 per cent total solids, of which not less than 8 per cent should be fat (United States Government Standard).

Evaporated milk is unsweetened and must be processed by heat in order to preserve it; it is therefore sterile, or nearly so. Evaporated milk should have at least 25.5 per cent total solids, of which 7.8 per cent should be fat (United States Government Standard).

Condensed and evaporated milks may be made from whole milk, skimmed, or partly skimmed milk. These are useful and legitimate products, but they should be labeled as to the grade of milk used, the amount of butter fat, etc. Inspection should be maintained to ensure quality and cleanliness. Condensed and evaporated milks at prevailing prices are relatively expensive when compared to fresh milk. Babies raised on these products are apt to develop scurvy unless given orange juice, tomato juice, or other antiscorbutic.

Dried Milk (Milk Powder).—Milk may be dried in vacuo at moderate temperatures, or on revolving belts or drums in the presence of hot dry air. The presence of the fat has interposed the greatest difficulty to the complete drying of milk. In the Ekenberg process the milk is sprayed under constant pressure on the inner surface of a rotating steam-heated cylinder. The milk is thus dried in partial vacuum at a comparatively low temperature. A more frequently employed process common in the production of cheaper grades consists in spraying the previously concentrated milk on the exterior highly polished surface of revolving steel drums. Here it is almost instantaneously dried at a temperature of 230° F. and then scraped off by sharp knife blades. In the Bénévot-de-Neveu process the milk is first concentrated in a vacuum and then sprayed under great pressure into a large drying chamber where the cloud of finely atomized particles is surrounded by a current of hot air,

³⁴ U. S. Pub. Health Rep., 1920, 25: 35.

and thereby instantly dried. The result is a powder in which most of the physical and chemical properties of the original milk are retained.

The process has been improved in recent years to such an extent that when milk powder of good quality is mixed with water it makes a product that resembles milk in nearly all essential particulars.

Dried milk powder may be made from skim milk, from partly skimmed milk, or from whole milk, rich in cream. The product keeps well. It has practically all the nutritive value of the original milk. Vitamins A and B are not materially affected, but the antiscorbutic properties are diminished about one-half. In other words, drying, pasteurizing and age have about the same effect on this vitamin in milk. For general cooking and food purposes, it is about the equal of liquid milk.

Dried milk has all the uses of milk. It is used as a basis of certain proprietary infant foods; it is employed in admixture with cocoa and sugar; with egg powder and sugar as a custard powder, and in various other food combinations. It is extensively used in the baking and confectionery trades. It is convenient in the household and economical so far as waste is concerned.

Babies have been fed on dried milk exclusively with good results, but not all methods of drying have yet been tested. The answer to this question must await a number of years of patient observation. In any case, antiscorbutic accessories, as orange juice, should be used. Dried milk powder makes a good food for growing children and adults.

One of the most promising of the recent developments consists in the dehydration of the milk fat and the milk solids-not-fat separately. It is claimed that these products are much less perishable when kept separated than when together. Milk or any of its products may be reproduced by simple methods of mixing the two products with water.

The *advantages* of total or partial desiccation, whether evaporated, condensed or powdered milk, are manifest. It reduces weight and bulk and saves carrier charges; it improves the keeping quality of a perishable food; and it suppresses the watery environment necessary for microbial activity.

The possibilities of dried and partly dried milk promise a revolution in the milk industry. It permits milk to be produced in parts of the world where it can be made to best advantage. It saves the surplus at the spring flush. It simplifies the present cumbersome distribution to the householder, and stabilizes the supply for large cities. Transportation difficulties are swept away. Sanitary control of production, and honest labeling are therefore doubly important.

Fresh Milk Products.—Cream, butter, buttermilk, ice cream, sour milk, fresh cheese, and other milk products may convey all the infections contained in the original milk from which they are prepared. It is known that tubercle bacilli pass into butter and may live there for months. It has also been demonstrated that infected cream has been the cause of typhoid fever, septic sore throat, diphtheria, scarlet fever, and other milk-borne diseases. Outbreaks of typhoid fever, scarlet fever and other diseases have been traced to

ice cream. Cold preserves rather than kills bacteria. Therefore, ice cream may be infected either with the milk or cream from which it is made, or directly from a case or carrier.

Milk products are often made from milk that is left over or otherwise unsalable. This should be controlled by an efficient system of inspection.

The infections in fresh milk products may be guarded against by pasteurization. It is comparatively easy to pasteurize cream, for the reason that it may be heated to a higher temperature than is the case with milk without materially altering its physical properties.

Butter.—Butter is made by churning “gravity” cream or “separator” cream. The cream may be fresh, but is usually ripened, that is, partially sour before it is made into butter. Special cultures of microorganisms (“starters”) are sometimes added to ripen the cream for the purpose of giving the butter a particular flavor.

Butter is usually “scored” in accordance with a score card proposed by Woll in which 45 points are allowed for flavor, 25 for grain (body), 15 for color, 10 for salt, and 5 for packing. The amount of fat contained in butter may be determined by the Doran method which is accurate to within one-half per cent. Warm the butter to 40° C., stir thoroughly, add about 10 cubic centimeters of the sample into a graduated sedimentation tube, and whirl in the centrifuge for a few seconds; measure, and record. Now add about 5 cubic centimeters of gasoline; mix, and whirl again for 15 or 20 seconds. The gasoline dissolves the fat which rises. The non-fat portion sinks to the bottom. The latter is measured and the difference represents the amount of fat. The United States regulations require that butter shall contain not less than 80 per cent of milk-fat, and a renovated butter shall not contain more than 16 per cent of moisture.

Natural butter has a refractometer index at 40° C., ranging from 1.4531 to 1.4562, usually about 1.4553. The presence of other fats that have been mixed with the butter may readily be determined by a higher refractometer reading. Coloring matter is often added to butter. The presence of coloring matter may be detected by dissolving the fat in ether and adding to separate portions dilute hydrochloric acid and sodium hydroxid. The first demonstrates the presence of the azo dyes, the second, the vegetable dyes.

Butter turns acid and rancid in time, owing to the conversion of the fat into fatty acids. Rancid butter may be renovated by washing it with skim milk or with water to which bicarbonate of soda or lime is sometimes added to neutralize the acidity. There is no particular health objection to these processes provided such butter is sold as renovated butter.

Fresh butter contains a great number of microorganisms (millions per gram). The total bacterial count diminishes with time. There may be a reduction of 85 per cent in two weeks, and 93 per cent in four weeks. Butter may contain tubercle bacilli, typhoid, and other pathogenic bacilli. Of twenty-one samples of market butter examined in Boston,³⁵ two of them were found

³⁵ *J. Med. Research*, 1914, 30: 69.

to contain tubercle bacilli, being 9.5 per cent of the samples examined. On account of this danger butter should always be made from pasteurized cream and labeled "butter made from pasteurized cream," not "pasteurized butter."

Petri examined 102 samples of butter at Berlin, using 408 animals for inoculation; 16.7 per cent contained tubercle bacilli. Korn found 23.5 per cent of seventeen samples of butter at Freiberg to contain tubercle bacilli.

The frequency with which tubercle bacilli are found in butter is shown in a table collected by Swithinbank and Newman.³⁶ Of 498 samples tested from different sources, 76, or 15.2 per cent, contained tubercle bacilli.

Schroder and Cotton³⁷ have found that living tubercle bacilli will retain their infective properties for at least 160 days in salted butter when kept without ice in a house cellar.

Butter may also convey typhoid bacilli and other pathogenic microorganisms.

Oleomargarin consists of a mixture of edible animal and vegetable fats churned with milk. Since all of the ingredients are in themselves useful foods, the wholesomeness and nutritive value of the finished product is beyond question. *Oleomargarin* consisting exclusively of vegetable oils churned with skimmed milk is deficient in the growth producing vitamins. This objection does not apply to *oleomargarin* prepared from animal fats, as oleo oil is a recognized source of the growth promoting vitamin. The better grade of *oleomargarin* also contains some milk fat.

Objections to the manufacture and sale of *oleomargarin* are based rather on the possibility of fraudulent substitution for butter than on nutritive or sanitary considerations. In the United States, *oleomargarin* is an object of federal taxation, and its manufacture is subject to supervision by the Bureau of Internal Revenue. A tax of ten cents per pound is imposed on *oleomargarin* which is artificially colored to resemble butter and a tax of one-fourth cent per pound on the uncolored product. During the fiscal year ended June 30, 1924, 229,031,000 pounds of *oleomargarin* were prepared: 127,488,000 pounds were prepared from mixtures of animal and vegetable fats, 101,130,000 pounds from vegetable fats exclusively, and 413,000 pounds animal fats exclusively.

Oleomargarin containing fats derived from the carcasses of cattle, sheep, swine, and goats is a meat food product. It is, therefore, subject to Federal Meat Inspection. This inspection insures the use of pure and wholesome materials and the maintenance of cleanliness and sanitary conditions in the establishment. Only animal fats from "U. S. Inspected and Passed" carcasses may be used. *Oleomargarin* prepared exclusively from vegetable fats is not subject to federal inspection. Pasteurization of milk and other dairy products used is required in all establishments operating under Federal Meat Inspection and is regularly practiced in establishments preparing *oleomargarin* from vegetable oils. It will be evident, therefore, that the possibility of the dissemination of disease through *oleomargarin* is exceedingly remote.

³⁶ *Bacteriology of Milk*, Dutton, New York, 1903, p. 221.

³⁷ *Bureau of An. Ind., Cir. No. 153*, p. 38.

Test to Distinguish Butter from Oleomargarin.—Place a piece of the sample, about the size of a small chestnut, in an ordinary tablespoon. In the laboratory a small dish or test-tube may be used. Heat over a flame, first melting the sample to be tested, hastening the process by stirring with a splinter of wood (a match-stick). When melted, increase the heat, bring to a brisk boil, and after the boiling has begun stir thoroughly, not neglecting the outer edges.

Oleomargarin and renovated butter boil noisily, sputter more or less like a mixture of grease and water when boiling, and produce no foam, or very little. Renovated butter produces usually a very small amount. On the other hand, genuine butter boils usually with less noise and produces an abundance of foam.

The refractometer reading gives a more accurate test.

SANITARY CONTROL

Inspection.—An efficient inspection service is a preventive measure that strikes at the root of the milk problem. A good inspection service is expensive, but is worth its cost in providing cleaner and better milk.

A competent system of inspection will help the farmer very much with his problems, and the educational value of such a system is one of its best features. The score-card system is an essential element in a successful inspection service.

The *score card* should be used in inspecting dairies, but dairy scores may not correspond to milk grades as determined by bacteriologic tests. The score card has advantages and limitations. It scores cleanliness and decency, but cannot score intelligence and conscientiousness. It should take equipment as well as methods into account. The system of scoring is instructive. It may serve as a basis of grading and licensing.

Inspection is particularly helpful in tracing the source of infected milk and preventing recurrences. Another important element in any inspection system is the license or permit. All persons producing or handling milk should obtain a license, which should be issued only after the person has demonstrated his capacity to handle milk in a safe and cleanly manner. The license should be renewed at least once every year.

The sanitary inspector cannot prevent occasional unsanitary conditions. No veterinarian can prevent the occasional infection of milk from cattle diseases. The medical inspector cannot prevent the infection of milk from human sources. There is a more effective safeguard to prevent milk-borne infections and epidemics—pasteurization.

PASTEURIZATION

Pasteurization consists in heating milk to a temperature below that of boiling, holding it at that temperature for a definite time, and then chilling

it rapidly. The time and temperature of pasteurization are designed to be sufficient to kill the harmful microorganisms with the least possible effect upon the milk itself.

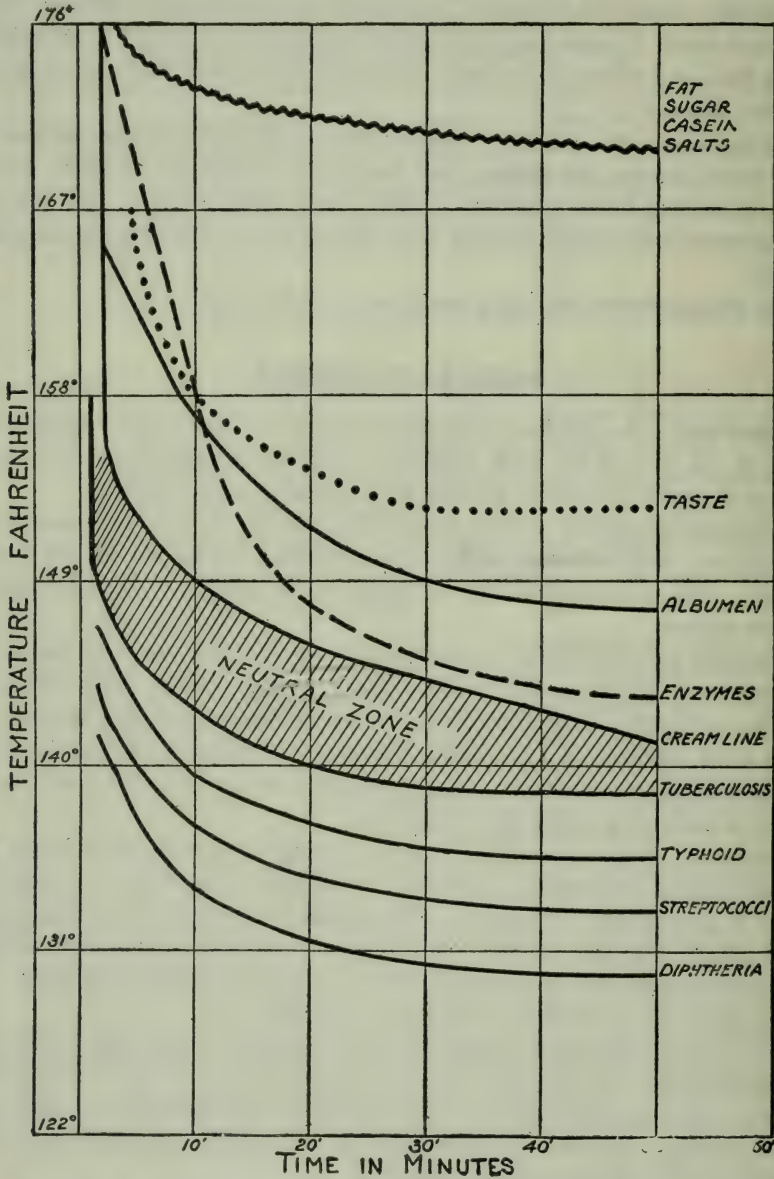


FIG. 58.—TIME AND TEMPERATURE FOR PASTEURIZATION IN THE NEUTRAL ZONE WHICH IS ABOVE THE THERMAL DEATH CURVES FOR THE PATHOGENIC MICROÖRGANISMS WITH MINIMUM INJURY TO THE MILK. (U. S. Pub. Health Bull., 1925, No. 147.)

Pasteurization is a preventive measure of public health importance. It should be defined legally in the sanitary code so that milk labeled "pasteur-

ized" but which has been processed by improper or incomplete methods may be prosecuted as misbranded. The heat of pasteurization does not alter the taste, appearance or digestibility of milk, and does not appreciably diminish its food value, except that there may be a diminution of its antiscorbutic property, which in any case should be offset by the use of orange juice or tomato juice. In fact, pasteurization tends to make the curds smaller and in this way perhaps easier to digest.

Time and Temperature.—It has been abundantly demonstrated that heating milk to 140°F. (60°C.) for twenty minutes is sufficient to kill the bacilli of tuberculosis, typhoid fever, paratyphoid fevers, dysenteries and diphtheria, the streptococci of scarlet fever and septic sore throat, the micrococcus of Malta fever, the virus of foot-and-mouth disease and all other non-spore-bearing milk-borne infections that are of concern to man. The temperature is a factor of the time; that is, for every degree above 140°F., the time may be reduced one minute, so that at 155°F. all the above pathogenic microorganisms are killed in five minutes. The exposure should not be less than five minutes.

A factor of safety is necessary in commercial practice, and it has been found by long experience in great cities handling enormous quantities of milk that 142 to 145°F. for thirty minutes is safe and satisfactory. Milk thus pasteurized protects the public health. Most cities and many states have adopted this definition of pasteurization,³⁸ but some still insist upon a minimum of 145°F. for thirty minutes. The dairy industry objects to heating milk to 145° F. or higher for thirty minutes or longer because the cream line is affected, but there is no sanitary harm from these temperatures.

No pasteurization device has been made that will automatically heat all the milk passing through under practical conditions to a given temperature and hold it precisely on the mark. There is a plus or minus deviation in the best designed heating apparatus of at least 1.5° F. To pasteurize milk between 142 and 145° F., it is necessary to set the thermoregulator at 143.5° F. When the sanitary code requires a minimum of 145° F., the apparatus must be set at at least 146.5° F., and the milk will then range between 145° and 148° F.

As the result of several years extensive experience with commercial pasteurization and of recent researches³⁹ it has been demonstrated that a temperature of 142 to 145° F., with a holding period of thirty minutes, serves the purpose of protecting the public health and preserving the integrity of the milk. Pasteurizing machinery is not fool-proof, and even the best designed apparatus must be operated with intelligent watchfulness. The thermoregulator should be responsive, the thermometers accurate and frequently standardized, for they are apt to be injured by the steam used in cleaning. Automatic records of the temperature and time should be kept for each run.

Advantages and Disadvantages.—Pasteurization prevents sickness and saves lives. Pasteurization is not ideal, but only an expedient. It is advo-

³⁸ *Pub. Health Bull.*, No. 147, U. S. Pub. Health Service, 1925.

³⁹ *Ibid.*

cated because milk is apt to convey the viruses of a number of diseases harmful to man. Pasteurization effectively prevents this hazard. It implies precaution, protection and prevention. It is the best insurance both for the industry and the consumer, and the simplest, cheapest, least objectionable and most trustworthy method of rendering milk safe.

Pasteurization does not claim to replace sanitation and common decency. It cannot atone for filth and should not be used as a redemption process. Stale, weak and dirty milk is still stale, weak and dirty after it has been pasteurized. A pure milk is better than a purified milk. However, no one should drink raw milk that cannot be guaranteed by the health officer as safe and free from danger. Even certified milk or milk of equally high character is only reasonably safe without pasteurization. Less than one per cent of all the milk found upon the market is certified. Therefore, raw milk of this honor class is not a public health problem of any magnitude, although it has been responsible for outbreaks of diphtheria, scarlet fever and other diseases. There is no authentic record of any milk-borne epidemic caused by properly pasteurized milk.

It is sometimes alleged that pasteurization does not destroy nature's danger signal—souring. Milk pasteurized at the temperatures recommended (142-145° F.) sours as a result of lactic acid fermentation just as raw milk does, although somewhat more slowly. Nature has no danger signal for infected milk. Milk may be teeming with typhoid bacilli and other pathogenic microorganisms without its taste, odor or appearance being changed.

Pasteurization is sometimes objected to because it does not destroy heat resisting toxins which are supposed sometimes to be in milk. The occurrence of such poisons is a mere assumption. Even if they exist in milk, they would be in the heated milk as well as in the raw milk. The true bacterial toxins, such as those of diphtheria, tetanus and botulism, are killed at 80° C. for thirty minutes.

One objection to pasteurization has always been the claim that it will put back the cause of clean milk and good dairy methods, because pasteurization will make cleanliness unnecessary and will put carelessness at a premium. Experience has proven the fallacy of this argument; in fact, the general milk supply of large cities has materially improved despite pasteurization.

Pasteurization is not proposed as a substitute for, but as an adjunct to inspection. Inspection gives us cleaner and better, but not necessarily safe milk. Inspectors cannot be present all the time, and furthermore, even if they were Pasteurs, they could not see missed cases and carriers. Pasteurization destroys the dangers inspection cannot see. The combination of inspection and pasteurization corresponds in all respects to the modern principles of furnishing a safe water supply to a large city. The watershed, through inspection, is kept as clean as practicable, but the water is filtered or purified to protect the consumer.

There can be no more objection to the heating of milk for the use of adults or children above the age of one year than there is to the cooking of meat.

Infants should receive breast milk. There is no adequate substitute. When this is not possible, they should have the best and freshest cow's milk that can be obtained. Whether such milk is to be pasteurized, modified or otherwise treated rests with the pediatrician. Pasteurization has the well-nigh unanimous endorsement of sanitarians and pediatricians.

The Effects of Heat upon Milk.—The changes produced in milk by heat depend upon the degree of heat and the length of time of exposure. Milk heated to 145° F. for thirty minutes does not undergo any appreciable physical or chemical change. The boiling of milk, however, produces pronounced changes. In the main, these consist of a partial decomposition of the proteins and other complex nitrogenous derivatives; diminution of the organic phosphorus and an increase of inorganic phosphorus; precipitation of the calcium and magnesium salts and the greater part of the phosphates; expulsion of the greater part of the carbon dioxide; caramelization or burning of a certain portion of the milk sugar, causing the brownish color; partial disarrangement of the normal emulsion, and coalescence of some of the fat globules; coagulation of the serum albumin, which begins at 75° C.; the ferments are killed; some of the vitamins, notably C, are affected.

Boiled milk has a cooked taste which appears at about 70° C. This is due perhaps to the decomposition of certain of the proteins in the milk. The loss of certain gases also alters the taste, so that milk heated in closed vessels has a less pronounced flavor than if heated in open vessels.

Milk heated in the open air forms a pellicle which renews if it is removed. This scum forms when milk reaches about 60° C. It consists of:

Casein and albuminoid.....	50.86 per cent
Fatty matter.....	45.42 per cent
Ash	3.72 per cent

Milk heated in closed vessels does not form a pellicle, even when the temperature reaches the boiling point. It seems that this pellicle is due mainly to the drying of the upper layer of the liquid.

Heat causes a progressive decline in the hydrogen ion concentration of the milk as the temperature rises. Evidently, the heat modifies the balance of colloids in the milk.

It is claimed that heat influences the availability of calcium and phosphorus of the feeding mixture. It appears that heat pasteurization throws the calcium salts more or less out of the solution, and thus they are made less readily available. This does not occur to the same degree in quickly boiled milk, which therefore has its advocates both here and abroad.

Relation of Pasteurization to Scurvy and Rickets.—Milk contains but a moderate and variable amount of antiscorbutic property, vitamin C. The amount of this dietary factor depends upon the quantity contained in the feed of the cow. Stall-fed cows in winter furnish a milk almost devoid of antiscorbutic property. This vitamin is influenced by age, oxidation, and to a

certain extent by heat. Experiments have shown that the temperature of pasteurization recommended decreases this property in milk about one-half. In any case, cow's milk cannot be depended upon to protect children against scurvy, and they should therefore receive orange juice or tomato juice, whether the milk is raw or pasteurized.

Milk at best has but moderate antirachitic property, so-called vitamin D, which is not affected by heat. Rickets therefore cannot be laid at the door of pasteurization. It may readily be prevented by the use of cod-liver oil and the benefits of sunshine.

Care of Pasteurized Milk.—Pasteurized milk must be handled at least as carefully as raw milk. It is just as apt to become infected if exposed. Bacteria grow even more rapidly in heated than in fresh raw milk. The germicidal properties of milk are destroyed by heating above 158° F. (70° C.) but are little influenced at 142-145° F. for thirty minutes (see page 717). There is a plentiful lack of understanding concerning the relative growth of bacteria in raw or heated milk. From a practical standpoint, this question can be disregarded, for the pathogenic germs that concern us particularly are just as harmful if taken in raw milk as in heated milk.

Pasteurized milk should be bottled by machinery immediately following the process, kept cold and delivered promptly. The bottles should first be disinfected with steam or scalding water. All milk, whether raw or pasteurized, should be kept *clean, cold and covered*.

Official Control.—Pasteurization is too important a public health measure to leave to individual caprice. The process should be under official supervision. The sanitary code should clearly define the requirements, and pasteurized milk should be labeled as such, or merely "heated milk," stating the degree of heat and the length of time and the date on which the process was done. Action for misbranding may be taken for milk labeled "pasteurized" but which has been processed by improper or incomplete methods. Milk should not be pasteurized twice.

Milk should be pasteurized at some central station where it may be done scientifically under official surveillance. In other words, it should be done for the householder just as in the case of central water purification plants. If water needs filtration or chlorination, experience teaches that it is inadvisable and expensive to depend on the householder to carry out the process. The same is true with pasteurization.

Methods of Pasteurization.—There are five methods of pasteurizing milk: (1) the flash method, (2) the holding method, (3) continuous flow, (4) the vat method, and (5) in the final container.

Pasteurizing machinery must be well designed, properly run and kept clean and in good order. It should have automatic temperature control and automatic temperature recording devices, and the records should be kept for official inspection. From time to time the thermometer and thermoregulators should be standardized and the process controlled by physical and bacteriological methods to insure its effectiveness. The first gallons of milk that go

through a cold pasteurizer may not be thoroughly heated and should be re-run.⁴⁰

The Flash Method.—The flash method consists of heating milk momentarily to a temperature of about 158° F. and chilling at once. This is done by allowing it to flow in a thin film on the outside of a steam coil, and then over cold metal. It does not give uniform results and is not entirely reliable, and hence does not meet with the approval of the sanitarian. The method, however, is rapid and cheap.

The Holding Method.—The holding method consists of heating the milk to the desired temperature (142-145° F.) and holding it in a suitable tank at that temperature for a given time (thirty minutes). In this way none of the milk escapes the disinfecting power of the heat, and the pathogenic bacteria in question are killed with certainty. This method has proved satisfactory in practice under commercial conditions. From the holding tank the milk should be run directly to the cooler and thence to the bottling machine.

Continuous Flow.—In this method the milk is first heated and then permitted to flow through a series of tanks or coils of metal tubes. The rate of flow is adjusted so that the heated milk remains the desired time in the apparatus. Experiments have shown that there is mixing and irregularity in the time of flow.

The Vat Method.—The vat method requires a double walled vat or cauldron, which is usually enameled or glass lined. The milk in the vat is heated by steam in the double wall. When the temperature reaches the desired point, the milk remains in the vat as a holding device. At the end of thirty minutes, it may be cooled by sending cold water through the double walls. In this way the entire process takes place in one container.

Pasteurization in the Final Container.—In order to heat milk in bottles, they should be well sealed with an effective stopper. The simplest method, which is useful in an emergency epidemic, is to place the milk bottles in a water-bath brought to the proper temperature, held there a sufficient length of time and then chilled. Several types of machinery are in use for this method. One borrowed from the beer industry consists of a revolving drum in a metal cylinder, in which the filled bottles are subjected to a spray of hot water, and before completing the revolution are chilled. Another type of device is by endless conveyors, which carry the bottles through first a trough of hot water and then cold. Pasteurization in the final container is called the perfection of the art. It has the theoretical advantage that contamination after pasteurization is entirely eliminated.

Home Pasteurizers.—Freeman's pasteurizer for heating milk in individual feeding bottles in the home is most serviceable. The modification of Nathan Straus is shown in Figure 59. It is used as follows:

After the bottles have been thoroughly cleaned they are placed in the tray (A) and filled with the milk or mixture used for one feeding. Then put on the corks or patented stoppers without fastening them tightly.

⁴⁰ *J. Med. Research*, 1912, 26: 127.

The pot (B) is now placed on the wooden surface of the table or floor and filled to the supports (C) with boiling water. Place the tray (A) with filled bottles into the pot (B) so that the bottom of the tray rests on the supports (C), and put cover (D) on quickly.

After the bottles have been warmed up by the steam for five minutes, remove the cover quickly, turn the tray so that it drops into the water, replace the cover immediately. This manipulation is to be made as rapidly as possible to avoid loss of heat. Thus it remains for twenty-five minutes.

Now take the tray out of the water and fasten the corks or stoppers airtight. Cool the bottles with cold water and ice as quickly as possible, and keep them at this low temperature until used.

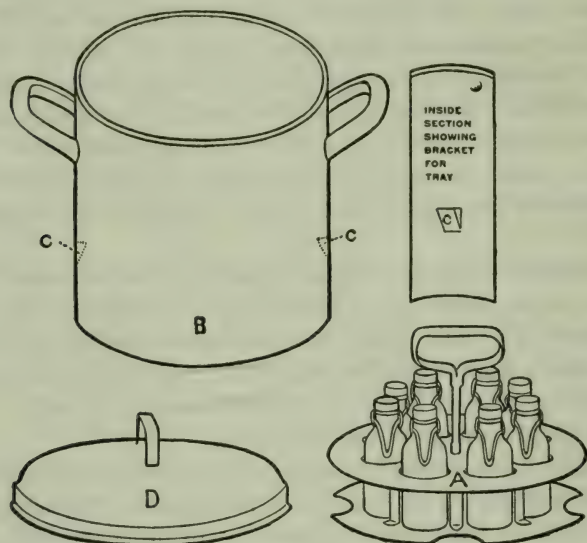


FIG. 59.—STRAUS HOME PASTEURIZER.

Use the milk from the bottles and by no means pour it into another vessel.

The milk should not be used for children later than twenty-four hours after pasteurization.

Emphasis is laid on the fact that only fresh, clean milk, which has been kept cold, should be used.

The Essential Requirements for a Safe and Satisfactory Milk Supply.

—1. Cows should be healthy and free especially from communicable infections, or any febrile disease, or inflammatory condition of the udder.

2. All persons who in any way come in contact with the milk or milk apparatus should be free from communicable diseases and not be carriers. A minimum of human contact should be insisted upon.

3. The milking should be done in clean rooms, the udders washed; the hands of the milker should be clean and dry.

4. The milk should be received into clean, sterilized pails, with a small mouth so as to keep out dust and dirt which falls from the udder and belly of the cow. If strainers are used they should be cleaned and boiled morning and evening. Cans and pails should be cleaned with washing soda or alkaline powder (not soap), rinsed in clean water, and then steamed or boiled.

5. The milk should be chilled to 50° F. or under at once, and kept protected from flies, dust, odors, and contamination, in a clean milk house until collected. A wooden paddle should not be used to stir the milk. If stirred a metal paddle is preferable.

6. The milk in transit to the city should likewise be kept protected and cold, not higher than 50° F., and guarded against tampering en route. The lower the temperature the easier it will be to keep the bacterial count down.

7. All apparatus at the city dairy, such as tanks, clarifiers, separators, pasteurizers, and bottling machines, should be kept scrupulously clean and sterilized daily with steam.

8. PASTEURIZATION at 142° to 145° F. for not less than thirty minutes should be followed by rapid chilling and the milk kept below 50° F. until delivered to the consumer. Milk and cream used for making milk products should also be pasteurized. The need here is just as great as for milk itself.

9. The pasteurized milk should be bottled by machinery in sterilized bottles, well sealed; and delivered promptly to the consumer.

10. All bottles and cans, after use in city delivery, should be washed and sterilized before being returned to the producer in order to prevent the conveyance of infection to the dairy or country farm.

11. The milk must be graded. In this way the producer is paid for care and cleanliness and the consumer has a ready means of knowing the sanitary character and nutritive value of the milk he purchases. The health officer should make frequent bacterial counts, and also sanitary surveys, for the purpose of grading.

12. A sanitary code based upon good milk laws, which include milk products, should be enforced.

13. A system of licensure, inspection and education to insure the above requirements should be followed.

CHAPTER III

FOOD: MEAT, EGGS, FISH, PLANTS, ETC.

MEAT

Sources and Consumption of Meats.—Meat forms an important item of food for human beings throughout the world. Man selects his meat-food diet from a wide range of animal life. However, those animals which live on plant life or on the animals of the lower forms are mostly selected for food by civilized nations, while the less used are those classes which feed on animals of the higher orders as fish, amphibia, reptiles and birds of prey. Herbivorous mammals and certain omnivorous animals are the chief sources of man's meat food, while the flesh of carnivora is mostly rejected with repugnance by civilized peoples. Next to mammals, birds and then fish supply the meat part of the human diet.

In the past fifty years the consumption of beef, veal, mutton, and pork in the United States and England has increased considerably, and to a less extent perhaps in other countries, due to the development of refrigeration processes, curing, canning, and improved facilities of transportation.

PER CAPITA AND PROPORTIONAL CONSUMPTION OF MEAT IN UNITED STATES, UNITED KINGDOM, GERMANY AND FRANCE

KIND OF MEAT	UNITED STATES (Average, 1920-1924)		UNITED KING- DOM (1921)		GERMANY (Average, 1904-1913)		FRANCE (1904)	
	Pounds	Per Cent	Pounds	Per Cent	Pounds	Per Cent	Pounds	Per Cent
Beef	61.1	49.3	66.7	51.6	41.5	36.7	37	46
Veal	7.6	5.0	4.	3.1	8	10
Mutton and lamb .	5.4	3.5	28.6	22.1	2.2	1.9	9	11
Pork	77.8	51.2	30.	23.2	69.4	61.4	26	33
Lard	14.4
TOTAL PER CAPITA	166.3	100.	129.3	100.	113.1	100.	89	100

During the year 1924, the total slaughter for food in the United States of cattle, calves, sheep and swine was approximately 119,980,000 animals or about one and one-tenth animals for each man, woman and child of the population. Argentina and Australia produce large surpluses of beef and mutton. The United States holds approximately one-sixteenth of the world's population and possesses within its borders about one-seventh of the recognized food-producing animals.

In some countries the flesh of horses, dogs, and cats is eaten. In Germany horses and dogs are slaughtered and regularly inspected for human food. The meat of these animals is also used in other countries that have long been flesh hungry. If derived from healthy animals there is no sanitary objection to the use of such meat. Horse meat, when eaten in ignorance of its true character, makes no unpleasant impression. In Paris, Vienna, and other cities large numbers of horses, mules, and donkeys are slaughtered for food.

Structure and Composition of Meats.—Meat is composed of muscular fibers, and the structures intimately associated with them, such as connective tissue, blood-vessels, nerves, lymphatic vessels, and more or less adipose tissue.

The muscle (flesh) of an animal consists of highly specialized tissue, whose chief function is to produce mechanical work through contraction. It is in addition a storage organ in which glycogen, a form of starch, and also fats are stored as reserve foods. It contains but little of cellular structures in the sense that the glandular organs, such as the liver, kidney, pancreas, etc., do. Chemical analysis shows the muscle to consist, aside from the reserve food-stuffs, principally of water, protein and salts. The glandular organs yield a high content of nucleic acid, while the muscle tissue yields but little in proportion to its weight. The inorganic content of the muscle tissue resembles that of the seed of the plant, rather than the leaf, both in amount and in relative proportions among the elements (McCollum).

Meat contains albuminoids and gelatinoids. The latter through action of hot water or steam are converted into gelatin. In addition meat contains the following nitrogenous substances: syntonin, myosin, muscle albumin, serum albumin, and numerous extractives, such as creatin, creatinin, xanthin, hypoxanthin, lactic acid; and small quantities of inosite and glycogen.

The toughness of meat is due to the thickness of the walls of the muscle tubes and excess of connective tissue which binds them together; hence the flesh of young domesticated animals is usually more tender than that of old or wild animals. Freshly dressed meat is alkaline in reaction. Soon after death rigor mortis of the muscles takes place and is accompanied by certain changes in the flesh. Its alkalinity changes to an acid reaction due to the development of sarcolactic acid, and during this process the connective tissue and muscular fibers become softened, thus rendering the muscular structures more tender.

The flavor of uncooked meat varies more or less with the age of the animal, its food, breed and condition at time of slaughter, and the handling of the meat after slaughter. The flesh of sexually mature male animals is usually stronger in odor than the meat of females or young or castrated males. This is particularly true of goats and swine.

Nutritive Value of Meat.—The nutritive value of meat depends mainly upon the presence of proteins and fats. Nitrogenous extractive matters, such as creatin, xanthin, and similar substances, sometimes called meat bases, are formed by cleavage of the proteins, but are of little value as foods. These nitrogenous extractives are present in about the same amount in both red

and white meats; in fact, there is no essential difference between red meat and white meat except the color.

Animal tissues must be differentiated into two groups, namely (1) glandular organs, as the liver and the kidneys; (2) the highly specialized muscle tissue. Beef muscle is very deficient in vitamins A, B and C, whereas liver is a good source of A and B. The proteins of muscle and the glandular organs are complete as sources of amino-acids.

Corresponding with the specialized function and the peculiarities in composition of muscle, we find that the dietary properties of meat are comparable with the seed rather than the leaf of plants; in fact, muscle tissue differs markedly from the seed in only one respect when considered as a foodstuff, namely, in the quality of its proteins. The proteins of meat are complete, those of seeds incomplete. Both meats and seeds are poor in inorganic salts, which are necessary in the diet. Both muscle and seeds are relatively poor in vitamin A as compared with such foods as milk, egg yolk, and the leaves of plants. Meat is an admirable source of protein of good quality, but it is not an economical food. It is appetizing, satisfying and stimulating. The condimental value of meat is very high. A common dietetic error is to eat too much meat. Growth and nutrition cannot be maintained on meat alone because it is so deficient in many of the necessary dietary factors. Carnivorous animals in the wilds make up this deficiency by drinking the blood, gnawing the bones and eating the liver of their quarry.

There is little difference in the composition and nutritive value between the meat of mammals, birds and fish. Meats from different sources and even from the same source differ chiefly in the amount and character of the fat they carry. The flesh of fish is ordinarily not classed as meat, but it has the same muscular structure and similar chemical composition and nutritive value as mammals and birds (see page 759).

Beef Extracts.—Beef extracts are nothing more or less than a soup or soup stock specially prepared from beef. They first became generally known through the researches of Liebig, and are now an important article in commerce. The composition of the ordinary beef extract found on the market contains from 15 to 20 per cent of moisture, from 17 to 23 per cent ash, and from 50 to 60 per cent of meat bases, and contains only a trace of soluble albumin, albumoses and peptone. The chief meat bases which form the principal part of the substance are creatin, creatinin, xanthin, carnin and carnic acid. It is, therefore, evident that meat extracts contain little nutritive matter, although this, being in a state of solution, is probably more readily digested than a similar amount of other nutritives in the form of ordinary meat. These extracts may be useful as stimulants or as condiments, or as a means of speedily introducing a soluble nutriment in the case of disease, where it is extremely important that even small amounts of nutritious material should enter the body.

A distinction should be made between beef extract and beef juice. *Beef juice* is obtained by strong pressure of fresh lean meat which is concentrated

in vacuo, or it may be freshly prepared in the household. Beef juice contains much more albuminous nutrient material than beef extract, provided it is not coagulated by heat and separated out.

Differentiation of Meats.—It was formerly difficult to distinguish horse meat, but the meat of any species can now be readily determined by means of the specific precipitins. The different kinds of meat may be distinguished by physical, microscopical, chemical, or biological tests. Ordinarily meats from different animals may be distinguished by their odor, taste or appearance. Microscopically the fibers resemble one another so closely that this test is not to be relied upon. Meat varies somewhat in chemical composition from different species, from different animals of the same species, and even from different muscles in the same animal. The principal difference in the chemical composition of meats from animals of different species consists in the glycogen and fat content. Thus, horse meat contains considerably more glycogen than beef. The glycogen test, however, is not reliable because it may be changed as a result of bacterial action.

The fats of different animals have different physical and chemical characteristics. The fats crystallize in different forms and have different melting points; also the fatty acids derived therefrom. A careful examination of the fat, therefore, will lead to an approximate degree of knowledge concerning the character of the flesh from which it has been derived. For instance, lard and beef or mutton tallow are easily distinguished from one another.

Safeguarding against Infection.—The prevention of infections and poisoning from meat and meat food products depends, first of all, upon the health of the animal, next upon the mode of death, and finally upon the methods of butchering, preserving and handling the flesh. Careful attention to every detail is necessary throughout all these operations. Cleanliness approaching surgical methods on the part of butchers during the preparation, transportation, and handling of the meat is called for. A careful system of meat inspection is a sanitary protection. Thorough cooking is our ultimate safeguard.

Meat should not be eaten raw, even where there is a carefully conducted inspection by trained experts. Individual cysticerci (tapeworm larvæ) are very easily overlooked, and one is enough to bring forth a tapeworm. It is also not practicable to examine the flesh of all hogs for trichinæ, and even though this were done with care the method does not afford complete protection. It is again emphasized that some of the more serious bacterial infections do not alter the color, taste or appearance of the meat in any way. Raw meat does not have a higher nutritive value than cooked meat.

MEAT INSPECTION

Importance of Meat Inspection.—Food animals may be affected with diseases or parasitic conditions which alter the character of the flesh and make it objectionable for food and possibly harmful, or the meat of a healthy animal may develop noxious qualities after slaughter through deteriorative changes,

or it may carry bacterial or other objectionable and contaminating material through insanitary handling. The border line between health and disease is ill defined. It is doubtful whether any animal slaughtered for food is entirely free from disease or unsoundness. The common or popular error on this point is the broad assumption that every condition to which the term "disease" is applied is noxious and harmful to such a degree as to make unsafe or unfit for food all the flesh of an animal in which any of these conditions is found. The facts of pathology plainly refute such assumption, and in order that these established principles may be accurately applied so far as practicable a skilled inspection of food animals and their products becomes necessary to protect consumers against these possible dangers. Inasmuch as the consumers of meat are unable to make this inspection, each for himself, it necessarily devolves upon the community, the state or the national government to provide skilled inspection so that the common need shall be met and the necessary protection provided.

Economic Value of Meat Inspection.—An efficient meat inspection system is not only of advantage to man, but is the means of detecting and preventing disease among livestock. A sharp outlook at places of slaughter will discover the first appearance of rinderpest, foot-and-mouth disease, Texas fever, or other epizootic, which may then be quickly traced to its origin and effective measures taken for control or eradication. Foci of herd diseases, such as tuberculosis, actinomycosis, and hog-cholera, may thus be located. A meat inspection service is therefore of great economic importance and an effective agency in locating and eradicating dangerous diseases from the food herds of the country.

The practices of meat inspection vary in different countries, depending upon the local conditions. Thus, in some countries, which have long had a scarcity of meat, and the people are, therefore, flesh hungry, much meat is passed for food that would be condemned in the United States. In countries where meat is not very abundant it is even necessary for the officials to keep a sharp watch to prevent the people from eating meat known to be injurious. In America where meat has been plentiful the attitude is very different and many people have a repugnance even to meat known to contain a harmless parasite. As meat becomes scarcer and prices higher this supersensitiveness on the part of some of the public will be overcome and a closer application made of the principles of pathology. The need of conservation, during and since the World War, has placed our national meat inspection service on a more practical basis, without sacrificing good standards.

THE FEDERAL MEAT INSPECTION

Scope of the United States Meat Inspection Law.—The federal meat inspection law, approved June 30, 1906, and amended in 1913 and 1920, provides for the inspection of cattle, sheep, goats, and swine, and, in a restricted way of horses, the meat or products of which animals are subject to interstate or foreign shipment. It is administered by the Bureau of Animal Industry under the direction of the Secretary of Agriculture. The law depends for its

authority upon the interstate and foreign commerce clause of the Constitution of the United States. Under regulations issued by the Secretary of Agriculture, it is required that establishments that slaughter food animals or process meats and transport in whole or in part such articles of food in interstate or foreign commerce shall operate their plants under federal meat inspection, even though only a relatively small part of the output may be actually transported to another state, territory or foreign country. Therefore, a very considerable local benefit in obtaining healthful meat is derived from the conduct of federal meat inspection at plants within the several states.

The Federal Meat Inspection Service.—In brief the purpose of the Federal Meat Inspection Service is to eliminate diseased or otherwise unsound meat from the general food supply; to require that the preparation and processing of foods composed wholly or in part of meat are conducted in a cleanly and sanitary manner; to prevent the use in meat food products of harmful dyes, preservatives, chemicals or other deleterious substances, and to prevent false and misleading labels and statements in connection with meat foods offered for sale in interstate or foreign trade.

During the fiscal year 1924, federal meat inspection was maintained in 906 slaughtering, packing, rendering and meat-preparing plants located in 261 different towns and cities, and included most plants of considerable size or importance in the United States. The year's slaughter in these inspected establishments was in round numbers 9,180,000 cattle, 4,667,000 calves, 11,505,000 sheep, 31,000 goats and 54,416,000 swine, a total of nearly 80,000,000 animals.

In magnitude the Federal Meat Inspection Service stands alone among the meat inspection systems of the world. It is a service in hygiene and sanitation of benefit to the American people as well as to those of foreign countries who receive meats from the United States.

Application of the Federal Law and Regulations.—In general terms the federal system may be regarded as consisting of five continuous and coördinating inspections, namely, (1) sanitation, (2) the antemortem or live-animal inspection, (3) the postmortem or slaughter inspection, (4) the products inspection or supervision of processing and manufacturing meat food products, and (5) the laboratory inspection. A description of the essential features of these follows.

Sanitary Inspection.—Under federal regulations no establishment is granted inspection until certain definite and important requirements as to sanitation have been fully and satisfactorily met. Some of the requirements are adequate light and ventilation, efficient floor drainage, modern plumbing and sewerage, pure water supply, the use of smooth and impervious material in the construction of equipment, and so far as possible the use of such material in the construction of buildings. Modern and sanitary toilet and dressing rooms separate from other rooms, lavatories for cleansing hands, and equipment for disinfecting utensils must be provided. All rooms in which meat and products are stored or handled must be separate from those in which

inedible products are stored, handled or treated. Cleanliness in all operations and in the handling of meats is emphasized.

Veterinary Antemortem Inspection.—In this inspection all animals about to be slaughtered are carefully observed for signs of any disease or condition which might cause condemnation of the carcass or any part thereof during the slaughter inspection. All animals found to be, or suspected of being, so affected are marked for identification, are separated from other animals of the lot, and are slaughtered apart from other animals in order that an especially expert veterinary postmortem inspection may be given. During the antemortem inspection animals which show symptoms of particularly virulent diseases and those in a moribund condition are condemned outright and ordered destroyed.

Veterinary Postmortem Inspection.—The postmortem inspection is really a well-conducted autopsy and is performed by veterinarians during the process of dressing the carcass. The regulations require that the head, tongue, tail, thymus gland, and all viscera, and all parts and blood to be used in the preparation of meat food products or medicinal products shall be held in such manner as to preserve their identity until after the postmortem examination has been completed, in order that they may be identified in case of condemnation of the carcass. Briefly described, this inspection consists of an examination of the principal groups of lymphatic glands of the head, these glands being repeatedly incised in the case of cattle and swine because they are favored seats of tuberculosis in these species; likewise, the various groups of lymphatic glands of the viscera are viewed, palpated, and incised if necessary; the tongue, lungs, heart, liver, spleen and kidneys are examined, and the peritoneal and thoracic membranes, exposed bones, and the joints and surfaces of the carcass are observed for indications of disease or unsoundness. A carcass found affected with disease or other abnormal condition is marked, with its viscera and other detached parts, and all is subjected to an expert veterinary inspection to determine its disposition. Carcasses or parts were condemned by the Federal Meat Inspection Service during the year 1924 for more than forty diseases and conditions. Among these are listed the following: anthrax, tuberculosis, hog-cholera, pyemia and septicemia, actinomycosis, caseous lymphadenitis, Texas fever, cysticercus, emaciation, gangrene, leukemia, icterus, immaturity, pregnancy, sexual odor, tumors, parasitic infestations, abscesses, bruises, dropsical conditions, and asphyxia. A few of these diseases deserve brief mention.

DISEASES FOR WHICH ANIMALS ARE CONDEMNED

Tuberculosis.—Tuberculosis is exceedingly common in cattle and is becoming more and more prevalent among hogs. A preponderating percentage of all carcasses condemned as unfit for food is so condemned on account of tuberculosis. Thus, under Federal Meat Inspection 56,760 cattle, or 0.618 per cent, and 100,110 swine, or 0.184 per cent, were condemned on account of tuberculosis in the fiscal year 1924. In the same year 7,184 cattle and 125,000

swine affected with localized or limited tuberculosis were passed for sterilization after removal of all affected parts. Tuberculosis is important, not alone because so many food animals are infected with it, but because it presents a peculiarly difficult problem for the meat inspector. The fundamental thought in determining whether to pass or to condemn meat of a tuberculous animal is that it should not contain tubercle bacilli, and should not be impregnated with toxic substances of tuberculosis or associated with septic infection. If the lesions are localized and not numerous, if there is no evidence of distribution of tubercle bacilli throughout the blood, and if the animals are well nourished and in good condition, there is no reason to suspect that the flesh is unwholesome, and it is permitted to be used after the removal of the infected portions. Just when tuberculosis should be considered localized or generalized, from the standpoint of meat inspection, is frequently a difficult question to determine. Fortunately, the danger from this source is not very great, as tuberculosis of muscle is exceedingly rare, and the further safeguard of cooking is sufficient to kill the tubercle bacilli, provided the meat is thoroughly cooked throughout. The relation of bovine tuberculosis to human tuberculosis has been discussed on page 160.

Tuberculosis of cattle shows itself in four primary lesions: (1) the retropharyngeal lymph nodes, (2) the lungs and associated lymph nodes, (3) the mesenteric lymph-nodes, and (4) the liver. From the retropharyngeal nodes the process extends to the cervical lymph-nodes and also to the anterior mediastinal lymph-nodes. When this group of glands alone is infected the disease may be considered as localized. From the mesenteric lymph-nodes the infection frequently reaches the peritoneum, and from the bronchial lymph-nodes the pleura. The newly formed growth in the peritoneal or pleural cavities may be enormous in amount. It is often suspended from the omentum in great grape-like masses (Perlsucht), or the intestines may be plastered with tubercles. In these cases the animal otherwise may be in good condition; that is, the disease is still outside the vital organs and the tubercle bacilli have not invaded the blood stream. In Germany it is permitted to cut off such growth and allow the meat to go into consumption. In our country the meat of such animals is rejected.

For practical purposes it is necessary to formulate definite rules for the guidance of the veterinary inspector, and this is done with minute particularity in the regulations of the Bureau of Animal Industry in the case of tuberculosis. In general, if the tuberculous lesions are limited to a single part or organ of the body without evidence of recent invasion of tubercle bacilli into the general circulation, the diseased parts are removed and the remainder of the carcass is passed for use. If the animal suffered from fever before it was killed or is cachectic, anemic, and emaciated, or if the lesions are generalized, especially if they exist in two or more body cavities, or if the lesions are found in the muscles, intermuscular tissues, bones, or joints, or if the lesions are multiple, acute, and actively progressive, the carcass is condemned. Carcasses which reveal lesions more severe or more numerous than those de-

scribed for carcasses to be passed, but not so severe or numerous as the lesions described for carcasses to be condemned, may be rendered into lard or tallow or otherwise sterilized in accordance with the regulations, when the distribution of lesions is such that all parts containing tuberculous lesions can be removed.

In Germany tuberculous and trichinous meat is sterilized and sold as second quality meat in accordance with the third class or "*freibank*" meat system. There is no known sanitary objection to this practice, provided the sterilization is complete and the label represents the true nature of the product.

Anthrax.—All carcasses showing lesions of anthrax, regardless of the extent of the disease, are condemned and immediately incinerated. This includes the hide, hoofs, horns, viscera, fat, blood, and all portions of the animal. The killing bed upon which the animal was slaughtered must then be disinfected with a 1:1000 solution of bichlorid of mercury, and all knives, saws and other instruments that have come in contact with the infection must be boiled or otherwise disinfected.

Hog-Cholera and Swine-Plague.—Carcasses showing well-marked and progressive lesions of these diseases in any organ or tissue are condemned. If the lesions are slight and limited they may be passed for sterilization. Man is not susceptible to hog-cholera.

Actinomycosis.—If the animal is in a well-nourished condition and the disease has not extended from a primary area of infection the carcass may be passed for food after condemnation of the infected organs or parts, but if the disease is generalized the entire carcass is considered unfit for human use and should be condemned.

Tapeworm Cysts.—Carcasses of animals affected with tapeworm cysts, known as *Cysticercus bovis*, are condemned if the infestation is excessive. Carcasses showing a slight infestation may be passed for food after removal and condemnation of the cysts, provided the carcasses as a further precautionary measure to insure complete destruction of any hidden cysts are then held in cold storage or pickle for not less than twenty-one days; the time in storage may be reduced to six days if the temperature does not exceed 15° F. As an alternative to retention in cold storage or pickle, such carcasses may be passed for sterilization.

Carcasses or parts of carcasses found infected with hydatid cysts (*echinococcus*) may be passed after condemnation of the infected part or organ.

Septic and Pyemic Conditions.—All carcasses of animals so infected that consumption of the meat or meat food products thereof may give rise to meat poisoning should be condemned. For the information of the inspector the following conditions are specified: (1) acute inflammation of the lungs, pleura, peritoneum, pericardium, or meninges; (2) septicemia or pyemia, whether puerperal or traumatic or without any evident cause; (3) severe hemorrhagic or gangrenous enteritis or gastritis; (4) acute diffuse metritis or mammitis; (5) polyarthritis; (6) phlebitis of the umbilical veins; (7) traumatic peri-

carditis; (8) any other inflammation, abscess, or suppurating sore if associated with acute nephritis, fatty and degenerated liver, swollen soft spleen, marked pulmonary hyperemia, general swelling of the lymphatic glands, and diffuse redness of the skin, either singly or in combination.

It is required that, immediately after the slaughter of any animal so diseased as to require its condemnation, the premises and implements used must be thoroughly disinfected. The part of any carcass coming in contact with the carcass of any diseased animal or with the place where such animal was slaughtered, or with the implements used in the slaughter, before thorough disinfection has been accomplished, should also be condemned.

DISEASES AND CONDITIONS FOR WHICH CONDEMNATIONS WERE MADE ON POSTMORTEM INSPECTION, BY THE FEDERAL MEAT INSPECTION SERVICE DURING THE FISCAL YEAR 1924 *

CAUSE OF CONDEMNATION	CATTLE		CALVES		SHEEP		GOATS CARCASSES	SWINE		HORSES CARCASSES
	Car-casses	Parts	Car-casses	Parts	Car-casses	Parts		Car-casses	Parts	
Actinomycosis . . .	658	93,201	30	1,641	9	84	...
Anthrax . . .	5	...	4	...	18	17
Asphyxia . . .	8	...	7	1,406
Blackleg . . .	77	13	102	27	281	285	...	7,652	82	...
Bone diseases
Caseous lymphadenitis	876	13	7
Cellulitis	1	3	...	67	135	...
Congestion . . .	9	20	2	...	3	71
Contamination . . .	2	3,067	1	1	12	1,000	4,734	...
Cysticercus . . .	172	1,185	24	2	162	7	...	105	88	...
Dropsical diseases . . .	36	18	11
Emaciation . . .	6,609	...	2,185	...	2,811	...	299	1,316	...	4
Foot-and-mouth disease . . .	821	...	4
Frozen	2
Gangrene . . .	72	...	39	...	3	12
Hog cholera	44,725	13	...
Hydronephrosis . . .	1	1	28
Icterus . . .	123	...	136	...	1,715	...	3	5,971
Immaturity	4,661
Injuries, bruises, etc. . .	4,158	405	894	98	602	194	5	1,861	8,006	...
Leukemia . . .	465	...	31	...	9	155
Melanosis . . .	32	5	113	4	31	161	...	8
Moribund . . .	9	...	12	...	28	140
Necrobacillosis . . .	8	1	2	1
Necrosis . . .	7	1,078	1	28	...	1
Parasitic diseases . . .	17	27	36	30
Phlebitis	127
Pneumonia, peritonitis, enteritis, metritis, pleurisy, etc. . .	7,855	...	2,321	...	4,739	...	4	33,470	...	12
Pregnancy and recent parturition . . .	125	22	425
Septicemia, pyemia, uremia etc. . .	4,393	...	1,260	...	1,314	...	2	24,997	...	8
Sexual odor . . .	1	3	4,514
Skin diseases . . .	1	...	2	...	1	101	7	...
Texas fever . . .	63	...	88
Tuberculosis . . .	56,760	73,794	634	546	13	...	1	100,110	1,099,253	2
Tumors and abscesses . . .	1,436	3,298	56	246	152	48	...	4,314	66,899	2
TOTAL	83,923	176,093	12,736	2,566	12,853	575	321	232,670	1,179,301	36

* Annual Report of the Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, 1924.

Poisons in Meat.—Meat may occasionally be injurious to health from a variety of miscellaneous causes. Thus, an animal that has died of arsenic or

other poisonous substance may contain sufficient of the poison in the tissues to affect the person who eats part of the flesh.

The belief that sickness in man can follow the consumption of the flesh or milk of animals which have previously fed upon poisonous plants is not unfounded. Chestnut states that as much as three grains per liter of formic acid may be present in honey, and that poisons from various plants have been isolated from honey.

"U. S. Inspected and Passed."—Under federal requirements all condemned carcasses and parts are effectually destroyed for food purposes under direct supervision of inspectors. Carcasses and organs passed for food are marked with brands or marks carrying the statement "U. S. Inspected and Passed," or an abbreviation thereof, so that the consumers may know that the meat so marked has been inspected by the federal government. The ink used in marking is harmless.

Products Inspection.—The products inspection begins with carcasses, viscera and fresh meats previously inspected and passed on the postmortem inspection and does not end until the same are shipped from the establishment as fresh meats and products or as processed, manufactured and labeled meats and products. It covers all such processes and operations as rendering, curing, smoking, cooking, mixing, canning, manufacturing and labeling meats and products in the establishment. All meats and products in an establishment are subject to inspection and reinspection at any time to determine whether they have remained sound since the previous inspection, and samples for laboratory examination are collected. In short, the purpose of this inspection and supervision is to see that sanitary conditions are maintained; that the handling of meats and products at all stages is cleanly; that adulteration and the use of harmful substances are not practiced; and that the meats and products shipped are sound, wholesome and correctly labeled.

Importance of Products Inspection.—As regards the importance of the products inspection it may be mentioned that in the fiscal year 1924 federal inspectors found it necessary to condemn 13,156,000 pounds of meats, fats and products because the same had become sour, tainted, rancid, unclean or otherwise unfit for food.

Preservatives Allowed.—The regulations of the United States Department of Agriculture permit the addition to meat or meat food products of the following substances: common salt, sugar, wood smoke, vinegar, pure spices, saltpeter, nitrate of soda, and nitrite of soda. Benzoate of soda may be added to meat and products only when declared on the label with percentage used. Only such coloring matters as may be designated by the Secretary of Agriculture may be used under prescribed limitations and declarations. The adulterants most commonly used in meats are boracic acid, borax, sulphite of soda, and benzoic acid.

Laboratory Inspection.—The government maintains well-equipped district chemical laboratories at convenient points throughout the country. In these laboratories are made analyses and examinations of meats, meat food

products, and of the water supplies, curing materials, spices, cereals, denaturing oils and of other substances. The samples for analysis are collected by Bureau inspectors and forwarded to the district laboratory for examination.

Need of State and Municipal Inspection.—About one-third of all animals slaughtered for food in the United States do not receive the security of federal inspection. In other words, one out of every three animals is slaughtered and consumed locally, and therefore not subject to federal control. The federal system of meat inspection has been extended to all parts of the field authorized for it by law. The government is expending annually nearly \$5,000,000 to perform its part of the responsibility toward supplying the consumers of meat with healthful, wholesome, clean and unadulterated meat and meat food products. A few states and a number of municipalities maintain meat inspection which varies considerably in enforcement. In many places there is practically no control whatever over meat that does not cross the state border. It is even said that there is a traffic in tuberculous cattle and other diseased food animals to such places.

The Public Abattoir.—So long as animals are permitted to be slaughtered in any barn or cellar it is impossible to exercise a proper control over meat and meat products, and conditions which endanger the public health will prevail. The first essential of a good meat inspection service is to concentrate all slaughtering in large central, sanitary abattoirs. This simplifies the inspection and sanitary control, and is a needed measure to protect the consumer. In Germany, England, Australia and New Zealand public abattoirs have been established which belong to the city. These structures are built thoroughly of brick and concrete, are so constructed that they may be kept clean, and are well protected against rats. Each person who wishes to slaughter must obtain a permit and pay rent. In the entire city of Paris there are only three slaughterhouses. The erection and maintenance of well-controlled, modern public slaughterhouses is one of the needs of our country, especially in the smaller towns, and until this reform is accomplished we shall never have a satisfactory solution of the meat problem.

An abattoir must be especially well constructed and kept clean. The same may be said of the trucks, drays, and all objects that come in contact with the meat. Slaughtering and butchering involve more or less blood and refuse matter, hence the necessity of frequent and repeated cleaning. The water-closets, toilet rooms, and dressing rooms should be entirely separated from the departments in which the carcasses are dressed or meat products handled or prepared. Attention must be paid to eliminate all sources of odor that may contaminate the meat or be otherwise objectionable, and every effort must be made to keep out flies and other vermin, especially rats and mice. Dogs should not be allowed around slaughterhouses on account of the danger of spreading the echinococcus and other parasites. The feeding of hogs on the uncooked refuse of slaughterhouses should not be permitted.

The employees themselves must be cleanly and should wear clean outer clothes that may be readily laundered. The federal regulations even prescribe

that employees shall pay particular attention to the cleanliness of their boots and shoes. It is also important to wash the hands before beginning work and to be particular after each visit to the toilet in the slaughterhouse or butcher shops. Persons with communicable infections should not be permitted in any department of the work where the meat or meat products are handled or prepared in any way. It is important that butchers who handle a diseased carcass should thoroughly cleanse and disinfect their hands afterward. Butchers' implements used on diseased carcasses should be sterilized in boiling water and thoroughly cleansed before they are again used. The precautions required in an abattoir and butcher shop are based on the same principles as those in a surgical clinic. Meat that falls upon the floor or otherwise becomes soiled should be required to be removed and the soiled portions condemned. Inflation by air from the mouth should not be permitted; inflation of carcasses and parts by mechanical means is also prohibited by the Department of Agriculture. Only good, clean, and wholesome water and ice should be used in the preparation of the carcasses, and the wagons and cars and all surfaces with which the meat comes in contact should be kept clean and in good sanitary condition. All condemned carcasses, parts of carcasses and meat products should be effectually destroyed for food purposes under special requirements in an official abattoir.

Qualifications of a Meat Inspector.—A corps of thoroughly trained meat inspectors is one of the most important links in the chain of an efficient meat inspection system. A meat inspector should be a qualified veterinarian having special experience and training for his specialty. He must know the anatomy of the various food-producing animals, especially cattle, horses, swine, sheep, and also fowl, and must be acquainted with the normal parts of each. He must be able to distinguish between the various organs of the various species, so that he cannot be imposed upon by those who would like to substitute one for another. He must know how to examine animals during life, in order to determine whether they are healthy. He must know the character of all the infectious diseases which are likely to pass through the district where he is situated. The government recognizes that it requires a high degree of skill to conduct this work, and it has, therefore, placed the meat inspection service under the Civil Service, and, further, will admit veterinarians only if graduates of recognized veterinary colleges.

"BOB-VEAL"

"Bob-veal" is the flesh of immature calves, that is, animals less than two or three weeks old. "Bob-veal" is objectionable only from humanitarian and æsthetic grounds, not from a health standpoint. The prejudice against "bob-veal" is illogical. The meat is flabby, edematous, soft. The connective tissue is gelatinous and is present in greater quantity than in mature animals. The fat is reddish-gray and soapy, the meat less nutritious in value, as it contains a large proportion of water. The digestibility of the protein of "bob-veal" is the same as market veal, namely, 93 per cent. On account of its moist and

soft condition "bob-veal" has a greater tendency to spoil than the flesh of mature animals. Young calves are highly susceptible to a number of infections, particularly diarrheal diseases and infections which enter through the navel. Trouble, however, has seldom been traced to "bob-veal." Bollinger and also Ostertag have reported a few cases of illness due to "bob-veal," but in these instances the meat came from diseased animals. Infections may be guarded against by care, inspection and finally by thorough cooking.

ANIMAL PARASITES

Trichiniasis (Trichinosis).—*Trichinella spiralis*, formerly *Trichina spiralis*, commonly known as trichina, is a round-worm which passes its entire life cycle in man, rat, or hog. Many other animals, such as mice, foxes, guinea-pigs, rabbits, cats, dogs, etc., are susceptible. This parasite differs from many other animal parasites in affecting several genera and in passing its entire life cycle in each host. Trichinosis is rare in animals which do not eat meat.

Trichiniasis (usually called trichinosis) is not a mere medical curiosity. It is a common and important disease, readily preventable. The average mortality is about half that of typhoid fever, in some epidemics, however, rising to 16 or even 30 per cent, as in the Hedersleben epidemic¹ in 1865.

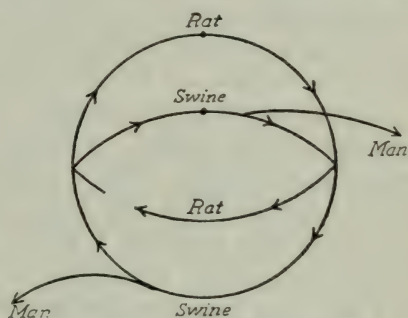


FIG. 60.—*TRICHINELLA SPIRALIS*.
Entire Life Cycle in Each Host.

Life Cycle of *Trichinella Spiralis*.—The larvæ are imbedded in the muscles. When the trichinous meat is eaten the capsules are dissolved in the stomach, the larvæ set free; the freed larvæ enter the intestine, where in about two days they grow into mature worms. The female produces upward of five hundred young, and as she is partially imbedded in the wall of the intestine is able to deposit her embryos directly in the lymph spaces in the intestinal mucosa. The embryos get into the blood stream and are thus distributed to the muscles. They may be found in large numbers in the circulating blood, between the eighth and twenty-fifth days after infection. After settling down in the muscles, the young parasites increase rapidly in size, reaching a length of about 1 millimeter, assume their characteristic spiral form, and become encysted, the formation of the cyst beginning about a month after infection. The adult worms usually disappear from the intestines in five or six weeks, or even sooner if the patient has diarrhea. Calcification of the capsules surrounding the encysted larvæ in the muscles may begin as early as six months after infection. The larvæ may remain alive for many years in calcified cap-

¹ Cited by Stäubli, *Trichinosis*, Wiesbaden, 1909, p. 16.

sules, but sooner or later they die and are absorbed or themselves become calcified.

Sources of Infection.—Some authorities consider rats to be the normal or common hosts of *Trichinella spiralis*, others believe that the hog is the only important reservoir of the parasite, and that in the absence of hogs the parasite would be unable to perpetuate itself among rats. It is a well-known fact that rats about slaughterhouses and butcher shops are commonly infested with trichinæ, and though it is questionable to what extent rats are responsible for the spread of infection to hogs, the possibility of their importance in this respect cannot be disregarded. Clearly important sources of infection for hogs are the carcasses of dead hogs, offal from slaughterhouses, and refuse or garbage containing pork scraps. Infection through feces has never been proved, and it is quite unusual if it ever occurs. Man receives the infection by eating trichinous pork, occasionally dog, cat, or bear meat. Country slaughterhouses, where hogs are fed on human feces and the offal of slaughtered animals and where rats abound, are important factors in the propagation of the infection.

Characteristics of the Disease.—Not all persons who eat trichinous flesh have the clinical disease. A limited number of the worms may not cause noticeable symptoms. The number of encysted larvæ that may be present in severe infestations is very large. As many as 1,200 have been counted in a piece of muscle weighing 1 gram, which would make about 500,000 in a pound. The number of encysted larvæ present in the bodies of persons who have died from trichinosis has been estimated, in various cases, at from 5,000,000 to 100,000,000. The severity of the disease depends largely upon the number of live larvæ swallowed. In man the disease is well characterized in two stages: (1) gastro-intestinal, (2) general infection. The symptoms of the second stage are fever,² intense pain in the muscles caused by the migration of the parasites, edema, and leukocytosis. The count of the white cells may reach 30,000 with distinct eosinophilia. One attack of trichinosis does not confer an immunity. Schwartz³ has shown that experimental animals infected and harboring trichinæ in their muscles are not immune to further infection when fed trichinous meat.

The recognition of trichinosis as a distinct infection is recent (1860). The parasite was named by Richard Owen (1835) and was long regarded as harmless and as a curiosity. The infection was mistaken for typhoid fever, rheumatism, acute miliary tuberculosis, and other diseases of common occurrence. The particular case which finally revealed the parasite as being capable of harm was that of a young woman admitted to the hospital at Dresden suffering from a disease diagnosed as typhoid fever. The patient had agonizing pains in the muscles, and the autopsy revealed the parasite imbedded in vast numbers in the muscular fibers. Leidy, in 1846, had announced the discovery of trichinæ in pork and in the present case an investi-

² *Trichinella spiralis* is the only metazoan parasite that, infecting man, causes fever with constancy.

³ *J. Am. M. Ass.*, 1917, 69: 884.

gation included the examination of some pork of which the patient had eaten four days before the first symptoms appeared, with the result that the same parasites were found. Since then many local outbreaks have been described, more particularly in Germany, where the custom prevails of eating raw or underdone pork, especially in sausage.

Prevalence of Trichinæ.—As an indication of the prevalence of trichinæ it may be noted that microscopists of the United States Department of Agriculture found living trichinæ in 115,812 hogs out of 8,257,928 examined during the period 1898-1906, or 1.41 per cent, from which it would appear that from 1 to 2 per cent of the hogs in this country are trichinous. The disease in man is probably more prevalent than the figures of the clinicians indicate. Careful search at autopsy has shown that many persons have been infected but have recovered. Thus, Williams,⁴ of Buffalo, examined 505 cadavers (largely from almshouses and insane hospitals, so his material cannot be considered to represent a fair average of the population) and found the parasites present in 27, or 5.34 per cent. Osler states that about one-half to two per cent of all bodies at autopsy contain trichinæ.

Prevention.—The disease is practically never recognized in swine during life. The protection rendered by microscopic inspection is quite unsatisfactory. This inspection consists in compressing small fragments of the muscle (diaphragm, tongue, etc.) between two glass plates, which are then examined with a low power of the microscope for the encysted larvæ. That this examination is not an entirely satisfactory safeguard, even in cases where it is done with care and precision, is shown by the history of trichinosis in Germany. Of the 6,329 cases of trichinosis occurring in Germany between 1881 and 1898, over 32 per cent (2,042 cases) were traced by Stiles to meat which had been inspected and passed as free from trichinæ. The microscopic inspection of every carcass for trichina is expensive and open to several practical sources of error.

Our federal meat inspection regulations no longer require a microscopic examination of pork for trichina. The former practice of microscopically examining pork intended for export to foreign countries whose import regulations required microscopic inspection was discontinued in 1906.

The United States Bureau of Animal Industry issues the following warning:

"No method of inspection has yet been devised by which the presence or absence of trichinæ in pork can be determined with certainty, and the Government meat inspection does not include inspection for this parasite. All persons are accordingly warned not to eat pork, or sausage containing pork, whether it has been officially inspected or not, until after it has been properly cooked.

"Pork when properly cooked may be eaten without danger of infection. Fresh pork should be cooked until it becomes white and is no longer red in

⁴*J. Med. Research*, 1901, 6: 64.

color in all portions of the piece, at the center as well as near the surface. Dry-salt pork, pickled pork, and smoked pork previously salted or pickled, provided the curing is thorough, are practically safe so far as trichinosis is concerned, but as the thoroughness of the curing is not always certain, such meat should also be cooked before it is eaten."

The trichinæ are not particularly resistant to heat. The thermal death-point of trichina larvæ is 55° C.⁵ The Bureau of Animal Industry recognizes 137° F. (58.33° C.) as the minimum temperature to which pork and products containing pork are required to be heated when cooked in establishments operating under federal meat inspection. The requirement refers to the temperature actually reached in the interior of the meat (pages 653 and 692).

Trichina larvæ die in less than twenty days at a temperature not higher than 5° F. Ransom⁶ disproved the notion formerly held that the larvæ of *Trichinella spiralis* are very resistant to cold. He recommends that meat should be refrigerated at a temperature not higher than 5° F. for not less than twenty days, a period which allows a probable margin of safety of ten days. Pork products of kinds prepared customarily to be eaten without cooking, for which there is a certain demand in this country from persons under the influence of foreign food customs, are required under the federal meat inspection regulations to be subjected to this process of refrigeration or to some other approved process that will destroy the vitality of any trichinæ that may be present. Certain special curing processes that kill trichinæ are permitted as alternatives to the refrigeration process.⁷ These restrictions are regularly enforced only in establishments operating under federal meat inspection and do not afford protection to consumers of pork products prepared to be eaten without cooking that come from other sources. In general, therefore, *the only safe rule to follow is to cook pork well before eating it.*

The rat and the hog should be regarded as the common reservoir of trichinæ; a persistent warfare should be made against rats in slaughterhouses, butcher shops, markets, and places where hogs are kept (see page 330). Hogs should not be fed uncooked offal from slaughterhouses, and on the farms should not be allowed to eat the carcasses of dead hogs.

The Pork (or Measly) Tapeworm (*Tænia Solium*).—*Tænia solium* passes the larval stage of its life history in the flesh of pork. Its encysted larvæ are known as bladder worms or *Cysticercus cellulosæ*; they are commonly called pork measles. Man eats these encysted larvæ which develop into adult tapeworms in the intestinal tract.

Infection with this tapeworm is particularly dangerous, because the cysticerci may occur in man as well as in the hog. When the cysticerci develop in important parts, such as the eye, brain, etc., death or serious consequences

⁵ Ransom and Schwartz, *J. Agric. Research*, 1919, 17: 201.

⁶ *Science*, 1914, 39: 181; *J. Agric. Research*, 1916, 5: 819.

⁷ Ransom, Schwartz and Raffensperger, Bull. No. 880, U. S. Department of Agriculture, 1920.

may ensue. The infection with this particular tapeworm is fortunately rare in the United States and Canada, but is more frequently met with in the Old World. The adult tapeworm occurs only in man; the larva is found especially in hogs and occasionally in man. This parasite is smaller than the beef tapeworm. The head is armed with a double row of hooks, with which it maintains its hold to the mucous membrane. Each link contains a uterus with seven to ten lateral branches, and the genital pore is marginal and irregularly alternate.

The source of infection in man is practically always the larvæ in under-

cooked or raw pork. Occasionally the cysticerci develop in man; in this case, the infection is contracted from another person through the eggs in the feces. Auto-infection also occurs. Hogs become infected from eating human feces containing the eggs, or from food and drink contaminated with them. To build a privy over the pigpen, as one sometimes sees in the country, means the formation of an endless chain in the biology of this worm.

Tænia solium produces less anemia than the fish tapeworm but may be dangerous because of cysticercus formation in man. This is the only tapeworm

in which this is known to occur. A person infected with *Tænia solium* may reinfect himself through dirty finger nails, unwashed hands, or other uncleanly habits, and it is also comparatively easy to infect others through the feces.

Prevention.—In prevention one must first consider the disposal of feces. Hog carcasses heavily infected should be destroyed; those having a light infection may be thoroughly cooked and the meat eaten. As the larvæ of *Tænia solium* appear to be more tenacious of life than those of *Tænia saginata* and survive longer after the death of their host, the holding of meat for a period of twenty-one days in refrigeration or in

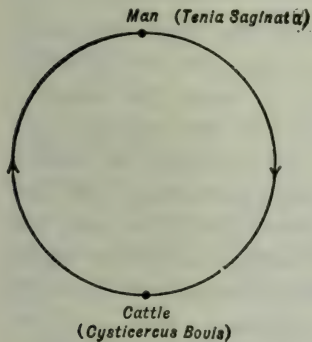


FIG. 62.—BEEF TAPEWORM.

brine as practiced in the case of beef slightly infested with *Tænia saginata* larvæ is a method that cannot be depended on to destroy the vitality of the larvæ of *Tænia solium* in pork.

Tænia saginata.—*Tænia saginata*, also called *T. mediocanellata*, occurs

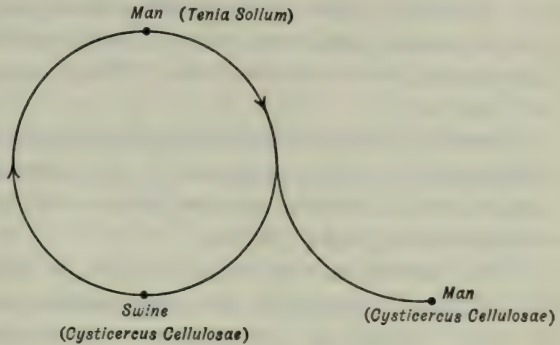


FIG. 61.—TÆNIA SOLIUM, THE PORK OR MEASLY TAPEWORM.

Note that man may infect himself.

only in cattle and man. The tapeworm is rather common in our country, but is not so dangerous as *Tania solium*, though at times it produces a certain degree of anemia and other symptoms. It is often difficult to expel, despite the fact that it has no hooks. In geographical distribution it is cosmopolitan. The adult worm occurs in man; the larva is found imbedded in beef and is known as the *Cysticercus bovis*. The uterus has fifteen to thirty-five slender dichotomous branches on each side. The genital pore is marginal and irregularly alternate.

Ransom⁸ concludes that if measly beef carcasses are exposed six days to a temperature not exceeding 15° F. the parasites die. Ostertag⁹ has found that cysticerci from beef carcasses held in cold storage or in pickle for twenty-one days are no longer viable.

Man becomes infected by eating raw or underdone beef. The larvæ are most frequently detected in the heart, diaphragm, and muscles of mastication, but occur throughout the voluntary musculature. Cattle become infected through the eggs passed in human feces, which contaminate their food or water.

Prevention.—Prevention depends first upon proper disposal of human excrement. An efficient system of meat inspection is an important factor in reducing the prevalence of the beef tapeworm. The cysticerci die in three weeks after killing, hence beef that has been kept in cold storage or in pickle twenty-one days may be regarded as safe. Proper cooking of beef removes all danger of infection with this species of tapeworm.

HYDATID OR ECHINOCOCCUS DISEASE¹⁰

The larvæ of a cestode, *Tania echinococcus*, frequently infest man in some parts of the world, rarely in the United States.

The larvæ are hydatids of a minute tapeworm of the dog. The adult worm in the intestinal tract of the dog is not more than four or five millimeters long and consists of three or four segments of which the terminal one alone is mature. The head is small and provided with four sucking disks, and a rostellum with a double row of hooklets. The terminal or mature segment contains about 5,000 eggs. The eggs are passed in the feces and then infest various animals, particularly the hog and ox, more rarely the horse and sheep. The egg hatches in the digestive tube, liberating an embryo which pierces the mucosa and lodges in the various tissues and organs of the body, where it develops into the larval or cystic stage (hydatids).

The disease prevails especially in those countries where man is brought in close contact with the dog, but more especially when, as in Australia, the dogs are used to herd sheep. The dog gets the larvæ from infested sheep. In the

⁸ *J. Parasitol.*, 1914, 1: 5.

⁹ *Ztschr. f. Fleisch-u. Milchhyg.*, 1897, 5: 127.

¹⁰ Echinococcus disease is not contracted by man from meat, but is conveniently considered in this chapter with the other tapeworms.

dog the tapeworm reaches maturity in the intestinal canal, and the eggs are passed in the feces to infect sheep, man, and other animals. In this country *Tenia echinococcus* is rarely found in the dog, because dogs are not often examined for its presence, but it is not a very rare parasite, as its cystic stage is rather frequently encountered in the liver of hogs by federal meat inspectors. The larva imbeds itself in the tissues and there develops a cyst—the hydatid cyst. This may occur in the liver, lungs, abdominal organs, nervous system; in fact, in almost any part of the body. The cysts grow in size as the larvæ multiply, forming daughter cysts and grand-daughter cysts.

Prevention.—The first essential in prophylaxis is to protect the dog against infection. This resolves itself into a good system of meat inspection, sanitary slaughterhouses, proper disposal of offal, and the keeping of dogs away from slaughterhouses, butcher shops, rendering plants, and the like. If offal is used as food for dogs, hogs and other animals it should first be thoroughly boiled.

The eggs of the worm reach the mouth of man directly and indirectly from the dog in various ways—through drinking water, through food soiled with dog feces, through dirty hands. Dogs lick their anal region and also lick fecal matter, and hence may directly transmit the eggs to man through licking and fondling. In an infested region, drinking water should be boiled; likewise all vegetables. Fruits and berries, especially those from near the ground, should be thoroughly washed before being eaten. Sheep and other herbivora become infected from dogs in ways entirely similar to those of man, and the principle of prophylaxis is the same.

The number of dogs should be diminished, especially stray dogs, which show a high percentage of infection. The control of the number of dogs and their habits would also help with the rabies problem. The better management of local slaughterhouses, of which there are still many in this country operating without inspection, would not only help control echinococcus disease but also trichinosis.

FISH

In nutritive properties, there is little difference between the muscle of fish and that of beef. In other words, fish is meat although ordinarily not so regarded. Drummond¹¹ found that the coagulable proteins of muscle tissue of the cod, herring and canned salmon have a nutritive value as high as those derived from beef. Fish also resembles meat in that both are poor in vitamin B.

The percentage of fat in different species of fish varies greatly. The unique quality of fish oils as a source of vitamins, especially A and D, suggest the importance of fish in the diet. The livers of fish do not serve as a storehouse of glycogen as do the livers of mammals, but are loaded with fat. All liver oils from fishes thus far studied are excellent sources of growth-promot-

¹¹ *J. Physiol.*, 1918, 52: 95.

ing vitamins. The liver of the cod is a veritable storage battery of radiant energy, which gives to cod-liver oil its well-known antirachitic properties.

Fish poisoning or ichthyotoxismus is most frequent in Russia, Japan, and the West Indies, and other seacoast countries in which fish forms a large part of the diet. It occurs especially in warm countries.

Physiological Fish Poisoning.—Some fish are always poisonous, that is, normally contain a substance toxic to man; usually the poison is developed only during the spawning season. Various species of the *tetrodon* and *diodon*, which includes the puffers, balloon fish and globe fish, frequently cause serious and fatal poisoning in Japan. The most poisonous is perhaps the fugu. In Tokio alone, 680 fatal cases out of 933 were reported occurring in 1885 to 1892 from the so-called "*fugu*." In China and Japan such fish are sometimes taken for suicidal purposes. The active principle in fugu poisoning resembles curare. The poison is found mainly in the head, liver and ovaries, and called "fugin." It is not destroyed by boiling. Its chemical nature has not been determined. The symptoms produced are: dyspnea, cyanosis, dilatation of the pupils, relaxation of the sphincters, paralysis of speech, dizziness, salivation, and vomiting. Death may result in one or two hours.

Few fish containing physiological poisons are found outside of the tropics. Some fish, such as shad and smelts, are preferred during spawning season. However, during spawning season the roe of different members of the sturgeon family, of the pike, and the barbel have been said to cause pronounced and even fatal intoxication; the symptoms resemble those of gastro-enteritis. Anchovy belassa and the meltite of the Indian Ocean are both said to be poisonous, the former causing death when only small amounts are taken, the latter causing violent vomiting. The Greenland shark causes an intoxication in dogs similar to that caused by alcohol. A certain degree of tolerance can be produced by feeding graded amounts. The roe of the European barbel produces the so-called "barbel cholera," while the roe of the pike is said to be poisonous during the spawning season. The toxic symptoms are said by Pozzi-Escot to occur in not less than twenty-four hours after ingestion. The smooth puffer (*Lagocephalus levigatus*) is considered by fishermen to be the most poisonous of the fishes of Brazil. Very little is known concerning the nature of physiological fish poisons.

Bacterial Poisons.—Bacterial poisoning from fish occurs. The fish may be diseased, or when caught may be healthy, but the bacteria gain access and grow throughout the meat as the result of contamination or imperfect preservation. Bacterial diseases among fish are rather common and often occur as epizootics. In almost all the reported instances of injurious action resulting from bacteria the fish has been eaten raw. Bacteria may form poisonous substances in fish closely resembling botulism. Fish caught by the gills in nets die slowly and decompose rapidly. They are of inferior flavor and value and are more liable to be injurious than fish taken from the water and killed at once; under such circumstances they remain firm and retain their flavor longer than those that die slowly. In some parts of the world live fish in tanks

are offered for sale in the markets. This procedure cannot be commended from a sanitary standpoint, for the tanks are apt to become dirty and the fish liable to sicken and die slowly, so that the object of purveying only live, fresh, and wholesome fish is largely defeated. It is well known that fish decompose readily and should, therefore, be handled in a cleanly manner and used as fresh as possible. When refrigerated the temperature should be low (page 676).

"Fish poisoning" is doubtless sometimes due to a toxin produced by *Clostridium botulinum*, or a similar anaërobe. Konstansoff¹² isolated an anaërobic organism from fish which had caused poisoning, and which he called *B. ichthyismi*. This organism produced a strong toxin which affects chiefly the nervous system. When administered to white mice per os, subcutaneously, intravenously or intraperitoneally, it gives rise to the same symptoms and produces the same pathologic lesions as result from ingestion of the poisonous fish. This organism is quite similar to *Clostridium botulinum*, but Konstansoff concludes, after a careful comparison of the two organisms, that *B. ichthyismi* is a distinct entity. The symptoms caused in mice are diminution of secretions and excretions, dilation of the pupils, ophthalmoplegia, clonic and tonic muscular spasms, retention of urine and feces, and motor paralysis, which spreads gradually over the entire body, death resulting from respiratory paralysis. The heart is resistant to the action of the poison, being only slightly decreased in rate. There is, therefore, marked similarity between botulism and this form of ichthyotoxismus. Poisoning from fish contaminated with *B. enteritidis* or one of the closely allied members of the colon-typhoid group is very seldom recorded. Hypersensitiveness to fish, in the sense of anaphylaxis, is rather common.

The Fish Tapeworm.—The principal animal parasite conveyed through fish is the tapeworm, *Diphyllobothrium latum* or *Dibothriocephalus latus*, which infects man wherever fresh fish forms a large part of the diet. The larval stage or plerocercoid is found in the muscles and organs of various fresh water fish, particularly pike, perch, and several members of the salmon family, and when partaken of by man develops into the adult tapeworm in the intestines. The adult worm is also found in cats and dogs that feed upon fish.

The fish tapeworm produces a severe anemia resembling pernicious anemia. The head is armed with hooks and attaches itself to the mucous membrane

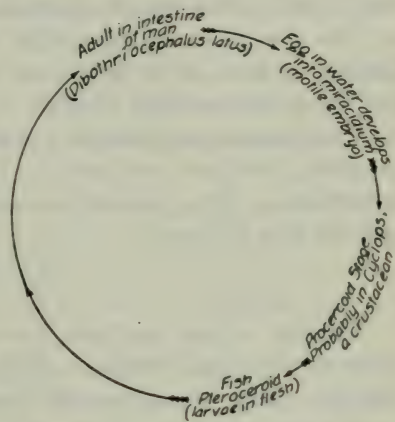


FIG. 63.—DIBOTHRIOCEPHALUS LATUS.
THE FISH TAPEWORM.
Produces serious anemia.

¹² Vestnik. Obshtsh, Hig., 1915, 5: 766.

of the bowels. Faust and Tallquist consider that the anemia is due to an hemolytic action caused by oleic acid found in the head of the fish tapeworm. Another opinion is that it results from the absorption of toxic products from dead worms or portions of worms that have lost their vitality before expulsion from the digestive tract. Each link of the fish tapeworm has a rosette-shaped uterus in the median line and a special uterine pore from which eggs are constantly discharged and may readily be found in the feces. It is through the pollution of the streams with sewage containing the eggs that the fish become infected. The larvæ that hatch from the eggs, however, first have to pass through a procercoid stage in small crustacea (cyclops), and the infected crustacea are then ingested by the fish. Most of the cases of fish tapeworms occurring in the United States are in immigrants, but twelve native cases have been reported and the parasite appears to have become established in this country, particularly in the Great Lakes region.

The prevention of infestation with the fish tapeworm consists in proper disposal of feces, so as to prevent the contamination of fresh water streams, and the proper cooking of fish.

Paragonimus westermani, a fluke (distome) common in Japan and other countries, produces very serious lesions in the lungs, clinically resembling tuberculosis. The parasite is contracted through fresh water crabs,¹³ and crayfish (see Flukes).

SHELLFISH

Shellfish include mollusks, as oysters, clams, mussels; and crustaceans, as lobsters, crabs, and shrimp. When we eat clams, oysters and soft crabs, we take the entire animal except the shell. This insures the consumption of unrecognized food substances which may be important supplementary factors in our diet. Sea foods are of special significance in that their iodine content is higher than that of land plants or animals. They are nutritious, savory and digestible, and are important additions to the diet.

The conditions which render such food injurious are much the same as those discussed in connection with fish. No disease of shellfish is known to affect man. Snails convey flukes, but are not important as articles of diet. Shellfish may be perfectly good and wholesome when fresh, but may become contaminated and injurious on keeping, especially if not properly preserved. It is very seldom that they are responsible for illness, albeit they have a bad reputation. This is due in large part to the fact that hypersusceptibility to these foods is rather common. In such instances the trouble is not with the food but with the person who eats it.

Man has made shellfish a danger to himself by polluting the water in which they live with sewage. On being transferred to clean water they may purge themselves in time of typhoid and other injurious bacteria.

¹³ Nakagawa, *J. Parasitol.*, 1916, 2: 111; Kobayashi, *Mitt. med. Fachschule zu Keijo*, 1918, 97.

OYSTERS

Bacteria in Oysters.—It is now well known that oysters, and doubtless other mollusks, while in polluted water, may take up large numbers of different kinds of bacteria, and that these remain alive and virulent for a long time. Herdman and Boyce found 17,000 colonies from an oyster obtained from the neighborhood of a drain pipe. Ordinarily oysters from open waters contain less than one hundred colonies. Oysters contain fewer bacteria during the winter months (December to March), when they probably hibernate.

Gorham¹⁴ has shown that during cold weather (40° to 45° F.) oysters rest or hibernate; the ciliary movement ceases, and feeding does not occur, and the oysters become relatively free from sewage organisms even when lying in sewage-polluted beds.

Oysters reflect the bacteriology of the water in which they live and grow. The bacteria are found both in the oyster and in the oyster liquor. Oysters from clean water contain few bacteria and no *B. coli*; oysters from polluted water contain many bacteria and numerous *B. coli*. The number of *B. coli* is expressed by a "score" in accordance with the method of the American Public Health Association.¹⁵

Scoring.—The shell liquor of five or more oysters is mixed and added in varying quantities to fermentation tubes containing lactose bouillon. One cubic centimeter of the mixed shell liquor is planted into each of five tubes; 0.1 cubic centimeter into each of five tubes; and 0.01 cubic centimeter into each of five tubes.

The presence of *B. coli* in each fermentation tube, if confirmed, is to be given the following values, which represent the reciprocals of the greatest dilutions in which the test for *B. coli* is positive:

If present in 1.0 cubic centimeter but not in 0.1 cubic centimeter, the value of 1.

If present in 0.1 cubic centimeter but not in 0.01 cubic centimeter, the value of 10.

If present in 0.01 cubic centimeter, the value of 100.

The addition of these values for the five fermentation tubes gives the value for the sample and this figure is the score. For example, if *B. coli* is found in all the tubes planted with 1 cubic centimeter and 0.1 cubic centimeter, but not in 0.01 cubic centimeter, then the score is 50 and the oyster is passed. If, however, *B. coli* is found in addition in one of the tubes planted with 0.01 cubic centimeter, then the score is 140 and the oyster is condemned.

Purification.—*Floating in Clean Water.*—Oysters from polluted waters may retain the typhoid bacillus for a long time. When placed in clean water the numbers rapidly decrease, so that the hazard from this source is diminished. The time of purification depends upon the temperature and quality

¹⁴ *Am. J. Pub. Health*, 1912, 2: 24.

¹⁵ *Am. J. Pub. Health*, 1922, 12: 573.

of the water and its amount. Klein found typhoid to persist in oysters from two to three weeks.

Krumwiede, Park, *et al.*,¹⁶ call especial attention to the prolonged persistence of minimal numbers of *B. typhosus* under different conditions. They state that apparently the only safe oyster is one which has been protected from contamination with fecal pathogens for at least some months prior to harvesting. They found *B. typhosus* to persist in shell oysters stored at refrigerator temperatures for forty-nine days, although the oyster died on the forty-first day. On shells of stored oysters the bacilli apparently are dead after fourteen to forty days. Repeated changes of fresh water result in a rapid diminution reaching about 99 per cent after three changes of water. A dead oyster serves as a culture medium and the number of *B. typhosus* will increase if the temperature is satisfactory.

Jordan¹⁷ found that the longevity of *B. typhosus* in the oyster juice of both shucked and shell oysters in storage varies with the temperature. In general, the temperature best suited for the preservation of the oyster tends to prolong the life of typhoid bacilli in the oyster. He found them to survive twenty-two days in shucked oysters and sixty days in shell oysters. In living shell oysters during dry storage at 45° F., typhoid bacilli survived for a longer period than the oysters. Kinyoun¹⁸ aroused oysters from hibernation, fed them with *B. typhosus*, and then stored them. *B. typhosus* was being recovered fifteen days after the oysters had been fed with the organism, when the tests were discontinued.

Chlorination.—Wells,¹⁹ in 1916, first called attention to the possibility of purifying polluted oysters by holding them in chlorinated sea water. The method was even used on a commercial scale at Inwood, Long Island, New York. The chlorination was produced either by electrolysis of the sea water or by adding bleaching powder. Carmelia²⁰ found that oysters treated by two changes of chlorinated water, covering a total period of from eighteen to twenty-four hours, practically sterilizes the water in which the oysters are floated and results in such marked bacteriological improvement in the bivalves that they may be regarded as safe for food, unless the initial infection has been excessive. Park and Krumwiede,²¹ however, find there is no advantage whatever in floating oysters in chlorinated water. The degree of purification following three changes of fresh sea water approximated that observed with a similar number of treatments with chlorinated sea water. We must conclude therefore that it is impractical to disinfect oysters by chlorination or by any germicidal substance, for the reason that if the hypochlorite is strong the oysters close up tight, and if weak the chlorin is soon used up and in any event does not penetrate. Chlorination is useless for shucked oysters.

Oysters and Disease.—Both typhoid and cholera have been convincingly traced to infected oysters. The oysters may become infected where they grow

¹⁶ *Am. J. Pub. Health*, 1926, 16: 263.

¹⁷ *U. S. Pub. Health Rep.*, 1925, 40: 819.

¹⁸ *U. S. Pub. Health Rep.*, 1921, 36: 876.

¹⁹ *J. Am. M. Ass.*, 1925, 84: 1402.

²⁰ *U. S. Pub. Health Rep.*, 1916, 31: 1848.

²¹ *Am. J. Pub. Health*, 1926, 16: 263.

or during the process of "fattening" or "floating," which consists in soaking them in fresh water for the purpose of making them more plump and increasing their size. In the language of the fishermen, this is called "floating," "plumping," "drinking," or "laying out." On account of the difference in osmotic pressure the water enters the cells of the oysters and certain mineral salts pass out. While the oyster increases in size and weight it is at the expense of the natural salt, mostly sodium chlorid, which the oyster contains. Floating is practiced by the majority of oyster growers, partly from necessity, for purchasers do not seem to realize that an oyster in its natural condition is never very thick and has a slightly greenish color.

Outbreaks of Typhoid Fever Traced to Oysters.—At Middletown, Connecticut, Professor Conn²² was the first to show that the outbreak of typhoid fever at Wesleyan University during 1894 was due to raw oysters eaten at fraternity banquets. Since then, many outbreaks have occurred (see page 113).

The New York Epidemic of 1924-25.—The widespread epidemic of 1924-25, which radiated from New York, exceeded in magnitude any heretofore recorded. As a rule typhoid outbreaks involve few people in a limited area, but this one involved at least 1,500 cases and caused over one hundred deaths. It followed the shipment of infected oysters for a radius of much over 1,000 miles. It included New York, Washington and Chicago particularly; and there was an excess prevalence of typhoid fever probably due to infected oysters in Buffalo, New York; Cincinnati, Ohio; Grand Rapids, Michigan; Memphis, Tennessee; Pittsburgh, Pennsylvania; Providence, Rhode Island; Rochester, New York; Scranton, Pennsylvania; Yonkers, New York; and many other cities, extending even to San Francisco, California. The infected oysters were distributed between October 25th and December 20th, and the disease prevailed three to four weeks later. This corresponds to the seasonal prevalence of oyster-borne typhoid, which occurs mostly in the fall and early winter. When oysters hibernate during cold weather, they are less subject to contamination. The trouble was traced to oysters distributed by a producing company operating within the vicinity of West Sayville, New York, and it is probable that the oysters came from one bed or float and constituted a small proportion of the total.

Other Outbreaks.—The increased prevalence of typhoid fever in Atlantic City, N. J., during the summer and autumn of 1902 was traced by Pennington²³ and others to the use of oysters and clams floated in Penrose Canal, which was highly polluted with sewage.

Bulstrode,²⁴ during 1902, reported twenty-one cases of typhoid fever and 118 cases of gastro-enteritis from a total number of 267 guests who had eaten raw oysters at the mayoralty banquets at Winchester and Southampton, England, on November 10th of that year. The oysters in question were imported from France and "laid down" or floated for a few days in sewage-polluted

²² *Rep. State Bd. of Health of Conn.*, 1894, 243-264.

²³ *Phila. M. J.*, Nov. 1, 1902, 634.

²⁴ *Suppl. App. A.*, 32nd Ann. Rep., Local Gov. Bd., England, 1902-3.

"drinking" grounds at Emsworth. One patient who developed a fatal case of typhoid ate only one infected oyster, while others ate only two or three of these oysters.

During the period from 1894 to 1902, inclusive, Newsholme, of Brighton, England, investigated 241 cases of typhoid fever which he ascribed to eating infected shellfish.²⁵

During the year 1902, Thresh and Wood²⁶ reported in the county of Essex, England, four cases of typhoid fever and twenty-one cases of illness due to eating Portuguese oysters sold on August 14th and 21st of that year.

Soper,²⁷ 1905, showed that twenty-one out of thirty-one cases of typhoid fever at Lawrence, Long Island, New York, could be traced to eating oysters and clams which had been floated or grown in Jamaica Bay, near Inwood, Long Island.

Netter²⁸ reported thirty-three cases of typhoid fever due to eating oysters from Cetto in 1907. The cases were very virulent in character, seven of the thirty-three resulting fatally.

Stiles²⁹ investigated an outbreak following the Minisink banquet, held at Goshen, New York, on October 5, 1911, and showed conclusively that the "Rockaway" oysters served on that occasion were responsible. There were seventeen well-defined cases of typhoid fever, with one death, and eighty-three cases of gastro-enteritis (diarrhea) traced directly to eating "Rockaway" oysters from Jamaica Bay, floated at Indian Creek, near Canarsie, Long Island, New York.

Fuller³⁰ reviewed the literature on this subject which covered more than twenty separate outbreaks due to infected shellfish up to 1904.

In Great Britain more than in other European countries, shellfish transmission of typhoid fever is regarded as quite frequent. In 1896, Newsholme, then health officer in Brighton, published careful studies showing that 30 per cent of the typhoid infections occurring in that city were due to oysters and other shellfish. For Belfast, the investigations of Mair showed that the extensive increases of typhoid fever from 1897 to 1909 was due in a large measure to infection from cockles gathered along a shore not far from the main sewer outlet.

Prevention.—The only safe rule is to use oysters and other shellfish from water which, upon bacteriological examination, would be safe for drinking purposes. Not only should the oyster beds be clean, but special care must be taken to prevent contamination of the water in which oysters are floated, because many oyster-borne outbreaks have been traced to this source. Under these circumstances, and with cleanly methods of handling, oysters are reasonably safe without chemical disinfection.

New York passed a law April 2, 1925, requiring purification of the water

²⁵ *Brit. M. J.*, 1903, 2: 295.

²⁷ *Med. News*, 1905, 86: 241.

²⁹ *Bull. 156. Bur. Chemistry, U. S. Dept. of Agric.*, Sept., 1912.

²⁰ *Rep. U. S. Bur. Fisheries*, 1904, p. 189.

²⁶ *Lancet*, 1902, 2: 1567.

²⁸ *Lancet*, 1907, 1: 551.

in which oysters are placed for drinking or floating, and prohibiting storage in water of less than a prescribed salinity. Other states have similar requirements. Health authorities are paying deserved attention to the sanitary conditions in oyster shucking and packing plants. Oysters can be infected from a typhoid carrier.

The prevention of typhoid and similar infections through oysters, clams, and other shellfish consists in regulating the location of the beds and in transferring doubtful oysters to a clean situation in clear sea water until the bacteria have perished or have been washed away. How long this may take is somewhat doubtful; there is a marked quantitative reduction in the number of typhoid bacilli, but some persist a long time. The only safe rule, therefore, is to forbid the use of oysters which have been exposed to contamination within several months. Thorough cooking will kill all these non-spore-bearing bacteria.

Prevention also needs adequate official sanitary supervision, regulation and protection of the industries engaged in the production, preparation and distribution of shellfish and other foods eaten raw.

OTHER SHELLFISH

Clams.—There is epidemiological evidence tracing typhoid outbreaks to clams. That the infection may be so conveyed is clear from bacteriological studies. Clams are often collected from polluted beaches. Perhaps they are not frequently the cause of trouble because usually cooked before being eaten. The consumption of raw clams, however, is increasing.

Mussel Poisoning.—*Mytilus edulis*, the common mussel, is a source of poisoning in England and on the Continent. Mussel poisoning is a comparatively rare occurrence. Savage³¹ has summarized the cases reported in Great Britain, finding that from 1827 to 1909 there were sixty-one cases, eight of which were fatal—a case mortality of 13 per cent. Figures for the Continent and America do not seem to be available, but in all probability they do not exceed those of Great Britain.

The cause of "mussel poisoning" is not known. It is highly probable that the trouble comes from bacteria of intestinal origin, for all the mussels which have caused illness have invariably come from sewage polluted waters.

Three clinical types have been described: (1) an erythematous type, which is doubtless anaphylactic and not an instance of actual "poisoning"; (2) a gastro-enteric or intestinal type; (3) a paralytic type. The difference in the last two types of poisoning is one of degree, the symptoms apparently varying with the virulence and amount of the poison and the susceptibility of the individual.

The paralytic type suggests curare. This is less frequent and more dangerous than the gastro-enteric types. It may be compared with botulism, but differs in rapidity of onset, the nature of the symptoms, and in the fact that

³¹ *Rep. to the Loc. Govt. Bd. on Pub. Health and Med. Subjects*, No. 77, 1913, p. 5.

boiling does not destroy the poison. Death has occurred in fifteen minutes after eating boiled mussels. A notable example of mussel poisoning occurred at Wilhelmshaven in 1885. A large number of dock laborers and their families were poisoned shortly after eating cooked mussels; three died. The mussels were examined by Brieger and Salkowski, who isolated several basic substances or "ptomains," one of which, mytilotoxin, was poisonous to animals, causing similar symptoms.

Mytilotoxin ($C_6H_{15}NO_2$) is said to produce the same symptoms in animals as found in "mussel poisoning" in man. Novy considers this a true instance of a heat-resisting alkaloidal-like poison or ptomain in a sense analogous to mushroom poisoning. Cats and dogs eating poisonous mussels are said to suffer with symptoms similar to those seen in man, namely, paralysis, coma, and death. Rabbits have been poisoned by giving them the water in which the mussels have been cooked. Thesen³² isolated from mussels a toxic substance which resembles mytilotoxin in some ways, but he has not been able to convince himself that the two substances are identical. Savage is impressed with the incompleteness of our knowledge concerning mussel poisoning and thinks the whole subject needs reinvestigation by our modern methods.

The prevention of mussel poisoning, on the basis of present information, rests primarily upon the prevention of sewage pollution of waters in which the mussels grow. In spite of differences between healthy and poisonous mussels, there seems to be no ready means by which the consumer can tell whether or not they are toxic. Cooking does not seem to render them safe.

Snails.—Of interest in connection with mussel poisoning is the so-called *snail poisoning*, which has come to be associated with a certain marine snail (*Murex uradatus*). It is not known whether this snail, which is sometimes used for food, contains, under certain conditions, a substance which is poisonous to man, whether pathogenic bacteria are present, or whether preformed bacterial toxins are the cause of the symptoms in man.

EGGS

Perhaps no article of diet of animal origin is more commonly eaten in all countries and served in a greater variety of ways than eggs. Eggs are used in nearly every household in some form or other. It has been calculated that on an average they furnish 3 per cent of the total food, 5.9 per cent of the total protein, and 4.3 per cent of the total fat used per man per day. When we speak of eggs we ordinarily mean hen's eggs, but the eggs of ducks, geese, and guinea fowls are used to a greater or less extent; more rarely turkey's eggs and sometimes those of wild birds. Plover eggs are prized in England and Germany, while in this country the eggs of sea birds, such as gulls, terns, herons, and murre, have long been gathered for food. Other eggs besides those of birds are sometimes eaten. Turtle's eggs are highly prized in most

³² *Arch. f. Exper. Path. u. Pharmacol.*, 47: Hefte 5 u 6.

countries where they are abundant. The eggs of the terrapin are usually served with the flesh in some of the ways of preparing it for the table. Fish eggs, especially those of the sturgeon, are preserved in salt under the name of caviar. Shad roe is also a familiar example of the use of fish eggs as food. The eggs of alligators, lizards, serpents, and some insects are eaten by races who lack the prejudices of western nations.

Very large quantities of eggs are now broken out, mixed, frozen, or dried. These products are largely employed by bakers and others who use eggs in quantities (see page 680).

Hen's eggs vary considerably in size and appearance. The shell constitutes about 11 per cent, the yolk 32 per cent, and the white 57 per cent of the total weight of the egg. The eggshell consists mainly of carbonate of lime, and when freshly laid is covered by a mucous coating. The egg white consists of 86.2 per cent of water, 12.3 per cent nitrogenous matter, 0.2 per cent fat, and 0.06 per cent ash. The yolk consists of 49.5 per cent water, 15.7 per cent nitrogenous matter, 33.3 per cent fat, and 1.1 per cent ash.³³ These are averages; different eggs vary somewhat in composition from each other. It is noteworthy that eggs contain practically no carbohydrates and are poor in calcium. The chick gets its calcium by absorption from the shell.

Nutritive Value.—The egg contains all the chemical complexes necessary for the formation of the chick during incubation. Eggs therefore furnish everything needed for the full development of the embryo. The egg is indeed a complete food, but not one which produces the optimum results when employed as the sole source of nutriment. There is an interesting contrast between eggs and milk. Aside from the calcium content of the white and yolk of the egg, which is much lower than that of milk, the contents of the egg resemble milk in a general way in nutritional value. The high content of milk sugar in the latter, and the almost complete absence of carbohydrate from the egg, cause them to differ considerably in the physiological results which they produce on animals when each is fed as the sole source of nutriment. Egg, when fed alone, encourages much more than milk the development of putrefactive organisms in the alimentary tract. The shell of the egg consists principally of calcium carbonate, and during incubation this is to some extent dissolved and absorbed for the formation of the chick. When eggs serve as human food the shells are discarded. There are distinct differences in the chemical natures of the constituents of eggs as contrasted with milk. The principal protein of egg yolk, like that of milk, contains phosphorus, but the fats of milk are phosphorus free, whereas phosphorized fats (that is, lecithins) are very abundant in egg fats. There is an abundance of lactose in milk, whereas the egg contains but a trace of sugar. The fats of eggs are fully comparable with butter fat as a source of vitamin A. The proteins of eggs are of high biologic value. The yolk is poor in vitamin B, but rich in antirachitic properties; it is even curative, but definitely less so than cod-liver oil.

³³ *J. Biol. Chem.*, 1910, 7: 110.

Classification.—In addition to fresh and refrigerated, eggs are classified in the trade as “rots,” “spots,” “checks,” “ringers,” “chickens,” “dirty shells,” “heated,” or “incubated,” etc. Eggs are assorted by inspection and candling. Candling consists in holding them before a bright light; the egg is translucent and the movable yolk may clearly be discerned, as well as the air space which is always at the larger end. A practiced eye quickly detects eggs that are not first quality. Rotten eggs are distinguished as “red rots” and “black rots,” depending upon the kind of putrefaction. By “spots” are understood eggs that contain opaque spots under the light. These spots usually consist of local growths of mold that have penetrated a crack in the shell, although they may be due to coccidia, embryos, or foreign bodies. “Checked” eggs are those which have slight cracks or nicks in the shell. “Ringers” contain small embryos of about two days’ growth, which are flat, disk-like, and reddish in appearance. “Chickens” contain embryos of larger growth. Eggs with dirty shells are undesirable more from æsthetic than other reasons. The dirt usually consists of hen excrement. A “heated” egg is a shrunken egg, that is, an egg that has been exposed to the summer temperature for several days. Some water is lost by evaporation through the porous shell, the air sac on the end has increased considerably in volume, and in many instances the embryo is partly developed; therefore, heated eggs are also known as incubated eggs. Many of the eggs gathered during the hot months of summer, especially in July and August, belong to this category. These eggs are much less desirable than the spring and fall layings. Eggs are also graded as to size, the very small eggs being undesirable, commanding a lower figure in the market. Further, eggs are classified as strong- or weak-bodied, depending upon how they “stand up” when broken out.

Bacteria in Eggs.—Eggs as they come from the hen frequently contain bacteria, worms, gravel, blood clots, and foreign bodies of various kinds. Practically all eggs contain bacteria, although numerous observers report occasionally that an egg is sterile. As a rule, these observations are based upon planting a small part of the egg. If the entire egg is planted a growth is almost invariably obtained. Thus, in eighteen freshly laid eggs which I examined every one of them contained bacteria in the yolk; two of them contained *Bacillus coli*. Curiously enough, there are practically always more bacteria in the yolk than in the white; the white contains some bactericidal property, probably similar to that possessed by fresh blood. The bacteria doubtless gain entrance to the egg while in the oviduct. Pernot³⁴ examined the eggs from over the size of a pea to the perfect egg and found bacteria at every stage. It is well known that the bacteria may also get into an egg through the shell, as it is porous and permeable. When the shell is moist and dirty the chances of growth and mold piercing it are increased. Eggs laid in the summer time (July and August) contain many more bacteria than those laid in the spring, fall, and colder months. Summer eggs do not keep

³⁴ Bull. 103, Oregon Agric. Coll. Exper. Station.

as well as winter and spring eggs. Sterile eggs keep better than fertilized eggs.

Eggs and Disease.—Of all foods, so far as known, eggs are less liable to convey disease or contain harmful properties than any other single food of animal origin. The literature is singularly free of instances of sickness attributed to eggs. There is no known infection of the hen transmissible to man through its egg. Eggs do not agree with some people, who have an "idiosyncrasy," so that a very small quantity will bring on symptoms resembling anaphylaxis. This condition is doubtless an instance of specific hypersusceptibility to egg protein. There are several cases on record in which this hypersusceptibility has been cured by the administration of pills or candy containing at first infinitesimal amounts of egg-white, gradually increasing the amount. The entire treatment should extend over a period of months. In this way an "immunity" may be established in man precisely analogous to the desensitization which may be established by repeated injections of an alien protein into guinea-pigs. The treatment is also applicable for other food for which there is a specific idiosyncrasy.

PLANT FOODS

Man is a parasite living on the plant kingdom. Our chief source of heat and energy is obtained from the vegetable world. The tissue-building constituents of our foods also are met in plants and are either taken directly or indirectly. The animals which furnish food to man function as expensive converters of the energy of plants into a form directly available for our uses. A cow must eat a liberal plant ration daily during several years before the milk and meat which she furnishes are ready for the market.

For purposes of nutrition, plant foods are classified as seeds, roots and tubers, and leafy vegetables; also fruits and nuts. Each class has distinct dietetic qualities.

THE NUTRITIVE VALUE OF PLANTS

Seeds.—Seeds are storage organs and can be classed together so far as their food value is concerned. The bulk of the food consists of starch. The proteins of seeds are incomplete, because they lack some of the amino-acids necessary to rebuild human protein. Seeds are also poor in mineral elements, especially calcium, sodium, and chlorin. McCollum examined wheat, corn, rice, oats, barley, rye, Kaffir corn, millet seed, flaxseed, pea, and both navy and soy beans. All, with the exception of millet seed, were below the optimum in their content of the dietary factor, vitamin A. Seeds are also deficient in vitamin B and in antiscorbutic properties. When seeds alone are used as the sole source of nutriment, it is not possible to secure appreciable growth in young animals.

Wheat is the most important seed grain used as food by all western peoples. The chief protein of wheat (gliadin) is superior to those of any other

grain. The prominent place of wheat in the diet of mankind is justified by the results of experiments on animals. Wheat, however, is poor in calcium, phosphorus, sodium, chlorin and iodine. The wheat germ contains most of the proteins which are of good quality, is exceptionally rich in vitamin B and contains some vitamin A. The oil of the germ contains vitamin E, which is essential for reproduction. In making white flour, the germ and bran are both removed.

Wheat flour is remarkable because it is more notably deficient in dietary factors than any other single food entering into the diet, except sugar, starches and fats, which are marketed in the pure state. Wheat flour consists essentially of starch. Its proteins are of relatively poor quality and its mineral components are conspicuously lacking in calcium, sodium, chlorin, iron and phosphorus. Wheat flour is very deficient in all vitamins. The merits of the flour rest essentially upon the peculiar glutinous properties of its proteins, which make possible the formation of dough. This is tenacious and permits of leavening and the production of light, spongy bread. Bolted flour has exceptional keeping qualities, largely because it contains no oil to become rancid. Wheat flour, despite its shortcomings, has become our most important energy-yielding food.

Rice, next to wheat, is the most important cereal grain in the diet of more than half of the human race. It is used especially in the wettest parts of the world. The practice of polishing rice had its origin in the desire to improve its keeping quality. The demand for white rice and the artificially established liking for white flour and white corn meal are illustrations of the failure of the instinct of man to serve as a safe guide in the selection of food. The æsthetic sense is appealed to by products of low biologic values.

Other Seeds.—The proteins of corn have a slightly lower value in nutrition than those of wheat. The oat kernel is comparable to wheat in its dietary properties in nearly all respects. The legume seeds, such as peas and beans, are richer in protein than any of the cereal grains, but nutrition experiments on animals show clearly that there is something lacking in their molecules, which limits the extent to which they can be converted in the body into tissue protein.

Leaves.—Leaves can be classed together as foodstuffs of similar character. They resemble one another just as do the seeds in other nutritive properties. Leaves are especially rich in just those elements in which the seed is poorest, namely calcium, phosphorus, sodium and chlorin. They are also a source of vitamin A. Thin leaves are more complete foods than thick ones. The thin leaves are cellular structures. The thick leaves contain reserve food material and some, like the cabbage, have been modified to storage organs containing reserve food and comparable to that stored in the endosperm of the seed. The fleshy leaves therefore tend to have in some degree the dietary properties of the seed.

The leaf is the synthetic laboratory of the plant. It builds up proteins, starch, sugars and fats, through the action of chlorophyll and sunlight upon

CO₂, which is absorbed from the air, together with water and mineral salts which are absorbed from the soil through the roots. The surfaces of the leaf are a mosaic of living cells. They contain all the chemical complexes which are necessary for the nutrition of the animal cells, and are qualitatively complete foods. In general, leaves are analogous to cellular organs of animals, such as liver, pancreas and kidneys, in dietary properties. The leaf proteins appear, from the data available, to supplement and enhance in some degree the value of the seed proteins with which they are combined. The leaf supplements, therefore, all the nutritive deficiencies of the seed, but not necessarily in a highly satisfactory manner.

Peoples who have employed the leaf of the plant as their sole protective food are characterized by small stature, relatively short span of life, high infant mortality, and by contented adherence to the employment of the simple mechanical inventions of their forefathers. Contrast with the success and vigor of people using milk, page 695.

Tubers and Roots.—After the seeds, the tubers of certain plants constitute one of the most important classes of energy-yielding foods. The potato and sweet potato are by far the most important representatives in this group in Europe and America, but several other kinds of tubers are widely used as human food in the Orient. The potato and other tubers are classed with the seeds from the standpoint of nutrition, because they consist mainly of reserve material.

Fleshy Roots.—Fleshy roots, such as the sugar beet, sweet potato and carrot, are similar in a general way to the potato, respecting both dietary properties and biological functions. They have a cellular layer at the periphery, and the interior is loaded with reserve foodstuffs. Steenbock and Gross found that carrots and sweet potatoes, both of which contain yellow pigment, are far better sources of vitamin A than are any of the roots which do not have a yellow color, as rutabaga, dasheen, beet, parsnip, potato, mangle and sugar beet. Feeding tests have shown that the properties of the beet resemble those of the seed and tuber rather than those of the leaf. The fleshy roots and the potato and the sweet potato have an inorganic content which resembles that of the seed in a general way.

Fruits.—The chief uses of fruits in the diet depend upon their salt content, their laxative properties, and their antiscorbutic value. Fruits do not rank high as sources of energy to the body; that is, their caloric values are rather low in comparison with most of the common food products. Fruits are almost without exception devoid of fats and are poor in protein. They are good sources of mineral salts. Some of them contain carbohydrate, such as the sugars in the orange and the banana. They all have a high percentage of water. The citrus fruits are rich in organic acids. They are highly palatable and exert a favorable influence on the excretory processes of the kidneys and the intestine. Many of them are rich in vitamin B and antiscorbutic vitamin. The addition of fruits and vegetables to the diet tends to establish a proper acid-base equilibrium, as they neutralize the meat and bread portion of the diet.

Nuts.—Nuts are the seeds of plants and should not be regarded as simple relishes or accessories, for they are staple articles of food. With the exception of the chestnut, they are rich in proteins and fats but poor in carbohydrates, and are an available source of vitamin B.

The proteins of nuts are similar to those of meat and fish. The protein of cocoanut contains all the essential amino-acids. Since the protein of nuts is fairly high in lysin, it is a valuable supplement to the grain proteins. Brazil nuts contain excelsin, which in itself is ample for all the requirements of protein metabolism in normal nutrition, according to Osborne and Mendel.

Nuts, however, lack a satisfactorily balanced mineral and vitamin content, being poor in calcium and vitamins A and C, which are absolutely essential to normal human growth and nutrition. The homely peanut is relatively rich in basic amino-acids in which the cereal proteins are relatively poor. Nuts vary considerably in composition. Chestnuts are starchy; cocoanuts and walnuts are rich in fat; while almonds, Brazil nuts, butternuts and peanuts are rich in both proteins and fat. Nuts in general, being rich in protein and fat, are comparable with meats as food.

Vegetable Oils.—Animal experiments by various investigators have shown that without exception vegetable oils, such as olive oil, cottonseed oil, maize oil, soy bean oil, cocoanut oil, sesame oil, etc., are essentially without value as a source of vitamin A. Vegetable oils are readily digestible and a good source of energy.

For a further discussion of the nutritive value of plants see *The Newer Knowledge of Nutrition*, by McCollum and Simmonds, The Macmillan Company, New York, 1925.

HOW PLANTS MAY INJURE HEALTH

Poisonous Plants.—Many plants contain a poison, such, for example, as aconite, strychnin, ricin, abrin, muscarin, and a long list of other substances normally present. According to Chestnut,³⁵ there are about five hundred species of plants in North America which are said to be poisonous. Most of these are rare specimens, and never eaten by man; some are poisonous only during certain seasons of the year; others are poisonous only when introduced parenterally into the human body; still others, such as the poison ivy, are injurious externally only to susceptible individuals, while many of them are known to be poisonous only to domestic animals. Of these latter, the larkspur (*Delphinium*), the water hemlock (*Cicuta maculata*), the lupines, the laurels, and the death camus (*Zygadenus*) are prominent examples.

Chestnut estimates that in the United States there are only about thirty species of plants which have been associated with poisoning in man, and furthermore most of such cases must be considered as extremely rare accidents. Instances of such accidents have been recorded by Jordan,³⁶ who cites the

³⁵ *Science*, 1902, 15: 1916.

³⁶ *Food Poisoning*, University of Chicago, 1917, p. 4.

mistaking of the American false hellebore (*Veratum viride*) for the marsh-marigold; the use of the fruit of the Kentucky coffee tree (*Gymnocladus dioica*) in mistake for that of the honey locust; the use of daffodil bulbs for food; the substitution of the mountain laurel (*Kalmia latifolia*) for winter-green, and the mistaking of the water hemlock (*Cicuta maculata*) for other edible roots. Jordan feels that poisoning from the latter cause is probably more frequent than supposed, saying that in one year, in New Jersey alone, ten cases, two of which were fatal, occurred. Ford³⁷ reports accidental poisoning in man following ingestion of the tutu plant, one of the *Eoriariae*, found chiefly in New Zealand. Roberto and Jelmoni³⁸ record an instance of poisoning following the ingestion of the berries of *Taxis baccata*, the European yew tree. Guerrero, De la Paz and Guerrero³⁹ report cases of poisoning from the use of a decoction of "Sanki," the fruit of *Illicium religiosum*, Siebold; they call attention to the great similarity between *Illicium religiosum* and *Illicium anisatum*, which, they say, is extensively used by the Filipinos as a stimulant, stomachic and carminative. The manuals on poisonous plants cite many other instances of accidental poisoning, due to the substitution, usually by children and the ignorant, of poisonous plants for similar food plants. A good example of this is the eating of castor beans, which are poisonous on account of the ricin they contain. Three or four beans cause violent gastrointestinal symptoms; five or six are fatal to a child, and twenty for an adult.

There is a great variation in the toxicity of plants, and tropical plants are more often poisonous than those of cooler climates, as is the case with fish, insects, snakes, and other animals.

Mineral substances in plants rarely cause poisoning. Lead, in grass, has been shown to be the cause of symptoms of lead poisoning in cows, and plants manured with superphosphates which contain arsenic may absorb enough arsenic to cause sickness.⁴⁰ Similarly, there is possibility of poisoning due to the use of insecticidal sprays, washes and powders, on vegetables and fruits (see page 277).

Acids are of more common occurrence. Prussic acid occurs free in some plants, as a glucosid in some others, especially in those of the rose and apple family. The bitter cassava (*Manihot utilissima*), from which ordinary tapioca is derived, contains prussic acid in considerable amount, and cannot be eaten in the fresh state. The prussic acid is dissipated by heat. Cases of poisoning due to this cause are not known.

Oxalic acid is quite common in many plants, and illustrative of its poisonous qualities is the outbreak of so-called "ptomain poisoning" reported in New York,⁴¹ which was shown to be due to soup prepared from "Qchav" or

³⁷ *J. Pharmacol. & Exper. Ther.*, 1910, 2: 1.

³⁸ *Chem. Zentr.*, 1916, 1: 1088; *Chem. Abst.*, 1917, 2: 2829.

³⁹ *Philippine J. Sc.*, 1916, 11: 203.

⁴⁰ *Science*, 1902, 15: 1016.

⁴¹ *Weekly Bull.*, N. Y. C. Dept. of Health, Sept. 16, 1916.

"Szchav" leaves, more commonly known as sour grass, a species of sorrel. Two grains of oxalic acid were found in each ounce of the leaves, and four grains in each ounce of the stems of this plant. The soup which was eaten contained about ten grains of oxalic acid per pint. Robb⁴² reports a fatal case of oxalic acid poisoning due to eating dried rhubarb leaves. Marked exhaustion, hemoptysis, early cardiac failure, and greatly delayed coagulation time of the blood were the prominent symptoms observed in this case. The oxalic acid content (as oxalates) of the stalks of rhubarb varies from 1.5 per cent to 40 per cent,⁴³ but there seems to be no figure available for the amount in the leaves. Arbenz⁴⁴ found that rhubarb contains 3.2 grams of oxalic acid per kilogram. Many other foods contain oxalic acid, but in harmless amounts. The poisonous qualities of the loco weed (*Astragalus mollissimus*), common to the western states, have been attributed to an acid present in the plant, called by some "loco" acid. Marsh has recently demonstrated that "trembles" is associated with the rayless goldenrod (see page 724).

Oils.—Some of the common vegetable oils, such as the oils of chamomile, cloves, cinnamon, sassafras, etc., may be poisonous in excessive amounts. Chestnut⁴⁵ cites an instance of the death of a child following the ingestion of two nutmegs, while Jordan⁴⁶ calls attention to an outbreak of sickness in Germany in 1911 due to the inclusion of maratti-oil (from the tropical plant *Hydrocarpus*) in a commercial substitute.

Carotinemia.⁴⁷—A diet rich in carotin, which is the coloring matter contained in carrots, spinach, egg yolk, and oranges, may produce a yellow discoloration of the skin, which resembles jaundice, except that the sclera are not involved. Carotin is also present in squash, and pigmentation of the skin has long been observed among vegetarians in Japan, where unusual amounts of this vegetable are used as a partial substitute for rice among farmers. There are no symptoms other than the pigmentation. The carotin and xanthophyll pigments derived from food are the sources of the coloring matters of milk fats and body fats, of egg yolk, of the corpus luteum, nerve cells, and other structures.⁴⁸

Parasites.—Certain vegetables, such as lettuce, celery, water cress, radishes, and similar plants, eaten raw, may convey typhoid fever, cholera, dysentery, both amebic and bacillary, the eggs and larvæ of animal parasites, and other agents of infection. This usually occurs from the use of night-soil as fertilizer, or from infected water from a foul source.

All vegetables which are eaten raw should be washed thoroughly beforehand. This cannot be depended upon to remove all the bacteria, but does

⁴² *J. Am. M. Ass.*, 1919, 73: 627.

⁴³ U. S. Dispensatory.

⁴⁴ *Chem. Abst.*, 1917, 2: 2374.

⁴⁵ *Science*, 1902, 15: 1016.

⁴⁶ *Food Poisoning*, University of Chicago, 1917, p. 16.

⁴⁷ Hess and Meyers, *J. Am. M. Ass.*, 1919, 73: 1743.

⁴⁸ Palmer and Eckles, *J. Biol. Chem.*, 1914, 17: 191, 211, 223, 237, 245; 1915, 18: 261, 1916, 19: 27.

wash away manure and other impurities, as the excrement of domestic animals which have been roaming in the garden. The larvæ of worms have been transmitted to man in this manner.

A fungus developing in rye is responsible for ergotism. Molds and smuts which grow on plants used by man for food may give rise to serious difficulty due largely to mechanical obstruction although there is some reason to believe that poisonous substances may be formed, for example, the sulphocyanic acid formed by *Aspergillus niger*.⁴⁹ Ernst⁵⁰ reports a case of minor infection, suggestive of pulmonary tuberculosis, in which the source of the mucor was probably corn.

Toxins.—Bacteria growing in plants may develop toxins. Botulism is the only known example in this category. This poison may form in beans, corn, peas, asparagus, beets, olives, and a variety of other plant foods containing protein.

Susceptibility, Idiosyncrasy, Anaphylaxis.—A number of plant foods, such as strawberries, tomatoes, oatmeal, etc., cause urticarial eruptions and other manifestations of anaphylaxis in susceptible individuals. Hay fever is another instance of hypersusceptibility, brought on by the pollen of various plants.

POISONING FROM PLANTS

Rhus Poisoning.—*Rhus* poisoning, also known as *Rhus dermatitis*, or *Dermatitis venenata*, is caused by an irritating resinous substance in the sap of numerous plants. The various plants which may provoke such irritation in susceptible subjects are at least sixty or seventy in number. The most common and best known of this group belong to the genus *Rhus*. *Rhus toxicodendron*, or poison ivy, is distinguished from other suspected creepers of a similar appearance by its possession of three leaflets instead of five. *Rhus diversiloba*, or poison oak, which grows especially in the western part of the United States, is a shrub or small tree. *Rhus venenata*, known as poison sumac, poison dogwood, and poison elder, is a shrub or small tree, growing in swampy places in the United States and Canada as well as in Japan.

Of the six varieties of the rhus family that grow wild in Japan, *Rhus toxicodendron* and *Rhus vermicifera* are the most injurious. Among the plants which less frequently cause dermatitis are the nettle (*Urtica doica*), the primrose (*Primula obconica*), cowhage (*Macuna pruriens*), smartweed (*Polygonum punctatum*), balm of Gilead (*Podophyllum*, *Balsamum gileadense*), oleander (*Nerium Oleander*), and rue (*Ruta*).

The part of these plants to be feared is the resinous sap. This sticky sap, exuding from all parts of an injured plant, when it comes in contact with the skin, causes intense irritation, which is characterized by its acute character, frequently beginning between the fingers, associated with swelling, and often

⁴⁹ *Science*, 1902, 15: 1016.

⁵⁰ *J. Med. Research*, 1918, 29: 143.

large vesicles and blebs, the exudate from which is non-toxic. The dermatitis occurs in sharply defined patches, elongated streaks, and other irregular shapes, corresponding with the original area of contact. It does not follow the nerve trunks. It seldom attacks the scalp or the inside of the hands. The original areas of contact are most affected and the parts of the skin to which the poison has been transferred from the original sites of contact are usually less severely affected. Together with the local lesions, there is a leukocytosis and constitutional disturbances, such as fever, coated tongue, loss of appetite, constipation, and a trace of albumin in the urine.⁵¹ The attack may subside in from four to six days, depending on the amount of the irritant and the sensitiveness of the skin. Idiosyncrasy plays an important part. Some persons are exquisitely susceptible; others resistant.

It is now quite certain that the toxic principle in rhus poisoning is not volatile, as was once supposed; in other words, contact is necessary, although not actual contact with the plant itself, for the sap may be carried indirectly by clothing, tools, insects, smoke, etc., to the skin of persons far from the actual neighborhood of the plant, thus explaining those mysterious "recurrent" cases of rhus poisoning. Sap thus carried loses its toxic properties by oxidation, the loss being more rapid at body temperature and moist atmosphere.

Japanese lacquered ware when new has caused a dermatitis in a large number of persons. The sap of the lacquer tree produces typical rhus poisoning. Susceptible individuals may be affected by passing under a lacquer tree, or by simply going by a lacquer-ware shop. This does not mean that the poison is volatile, for in such instances the sap is transferred in some mechanical way.

McNair⁵² has shown that smoke from the heated leaves of poison oak causes dermatitis if blown on the wrist. If, however, the smoke is filtered through glass wool, it is no longer irritating, showing that the smoke is only a mechanical carrier of the poison, thus confirming the oft-repeated observation that poisoning may result from exposure to smoke of camp fires, etc. The exact nature of the chemical substance is not known, but it is now clear that it is resinous in nature, and that it is absent from the pollen and plant hairs. The toxic substances in the several plants are identical or very closely related. Pfaff⁵³ isolated a fixed oil, *toxicodendrol*, of which 1/1,000 milligram in two drops of oil will set up localized edema and vesication.

The dermatitis may sometimes be averted, even after handling these plants, by the free use of an alkaline soap and water, or alcohol, containing a little dissolved sodium hydroxid. The poison is soluble in alcohol and alkalies. Gasoline may also be used. An aqueous solution of sodium bicarbonate is less effective. The washing must be prompt and thorough or else it will only tend to spread the irritating poison.

⁵¹ McNair, *J. Infect. Dis.*, 1916, 19: 419, 429.

⁵² *J. Am. Chem. Soc.*, 1916, 38: 1417.

⁵³ *J. Exper. M.*, 1897, 2: 181.

Ergotism.—Ergotism is a form of food poisoning brought on by the prolonged use of meal or bread made from grain contaminated with ergot, a parasitic fungus (*Claviceps purpurea*), which is a disease of rye and occasionally of other grains. The chief source of the poisoning in man is from rye, in which case the fungus may entirely replace the grain. The fungus grows in the grain as a hard, blackish mass called the sclerotium, the presence of which may be suspected by the color of the meal which is grayer than usual and often shows violet-colored specks.

The composition of *ergot* is complex and still not completely understood. Several active principles have been extracted. One of these, *ergotoxin*, has a powerful action on the tissues. Ergot also contains several bases, such as *tyramin* and *ergamin* (histamin), which are also found in putrefying meat and are derived from the amino-acids tyrosin, histidin and leucin by the loss of the carboxyl group.

Chronic poisoning with ergot may take either of two forms, *gangrenous* or *convulsive*. The gangrene is evidently due to vasomotor constriction and contraction of the unstriated muscle, and usually involves the limbs, especially the fingers and toes, but occasionally the ears and nose. Sometimes the whole arm or leg becomes cold and anesthetic, dark in color, and then dry, hard and shrunken, and falls off with little or no pain and no hemorrhage. In the milder cases there is only skin necrosis. In the convulsive form, the first symptoms are depression, weakness and drowsiness, often headache and giddiness, painful cramps in the limbs and itching of the skin. The spasms are prefaced by muscular pains and cramps and may continue only a few hours or perhaps for days. In severe cases paroxysmal convulsions set in, often epileptiform. This form was often fatal in early times. As a general rule, the gangrenous type prevailed almost exclusively in the epidemics in western Europe, while in eastern Europe the convulsive form was usual.

Ergotism occurred in epidemics from the use of bread containing ergot after poor harvests and especially in wet seasons. The last large epidemic in the United States occurred in New York in 1825. The history of ergot is given by Kobert.⁵⁴ The disease was formerly endemic in Europe, but of late years epidemics have become rare except in Russia. Some of the "plagues" of mediæval Europe may have been due to ergot poisoning. Hirsch reported some twenty-eight outbreaks in the nineteenth century.

Lathyrism.—Lathyrism or vetch poisoning is a rather rare condition met with in some parts of Europe, notably Austria and Italy, in northern Africa, and in India. It is associated with wretched conditions of living. The vetch seed is ground in the form of meal and used as a partial substitute for that of wheat. The seed is popularly known as chick-pea. The vetch seeds are obtained chiefly from *Lathyrus sativus* and *Lathyrus cicera*. The eating of bread prepared from meal containing the seeds of the lathyrus is followed by sudden and severe pains in the lumbar region, girdle sensation, spastic

⁵⁴ *Dorp. Stud.*, 1889, 1: 1.

motor paralysis of the lower extremities, tremor, and fever. The nature of the poison is not known, but it is probably of the nature of a toxalbumose, of which ricin and abrin, the poisons of the castor bean and the jequirity bean respectively, are well-known examples.

Favism.—Favism (*fava*, bean) is the name applied to a disease which has been reported in Italy. It is attributed to the use of beans as food, or even just smelling the blossoms of the bean plants. Pammel⁵⁵ cites instances of individuals who cannot inhale the odor of morphin, turpentine, tobacco, the flowers of the common bird cherry, the haw or the tuberose, without becoming ill. Gasbarrini⁵⁶ quotes the work of Fermi and concludes with him that favism is the result of an acute, toxic hemolysis. It is said to occur only where beans are cultivated on a large scale, and then usually only during the spring of the year. Of 1211 cases which were studied by Fermi in Sardinia, 752 were ascribed to ingestion of the beans, and 459 to inhalation of the odor or pollen of the bean plants. The onset of symptoms occurs a day or so after ingestion or from two to six hours following inhalation. It is distinguished by acute, febrile anemia with jaundice and hemoglobinuria, but there may also be abdominal pain, nausea, vomiting and diarrhea. Fermi noted an hereditary tendency in about 20 per cent of his cases, which in some instances could be traced through several generations. This hereditary predisposition is said not to become apparent in some cases until adult life. It is also stated that in some instances one exposure seems to confer a permanent immunity but others suffer at each exposure. The cause of the disease is unknown. Bacterial activity and fungi have been held responsible but no proof of either hypothesis has been brought forward. The reported seasonal prevalence, the incidence following inhalation of the bean pollen and the hereditary predisposition are suggestive of sensitization and anaphylactic reaction.

Mushroom Poisoning.—The ill effects from eating mushrooms are usually due to mistaking the poisonous for the edible species, and in America this is usually done by children, immigrants or the ignorant.

The number of species of poisonous mushrooms which are capable of causing death is not very great, perhaps twenty or thirty. *Amanita* and *volvaria* are the most poisonous genera, and are the ones usually involved in the fatal accidents.

Ford⁵⁷ estimates that from twelve to fifteen deaths occur annually in the United States from *amanita* poisoning. In September, 1911, in the vicinity of New York, following heavy rains, twenty-two deaths were reported. Jordan⁵⁸ believes that mushroom poisoning is probably increasing in this country due to an increasing use of mushrooms for food.

Amanita phalloides and *Amanita muscaria* are exceedingly poisonous, dangerous and seductive species, responsible for most of the deaths from toadstool

⁵⁵ *Manual of Poisonous Plants*, Part I, 1910, p. 7.

⁵⁶ *Policlin.*, Nov. 14, 1915; abstr. *J. Am. M. Ass.*, 1915, 2264.

⁵⁷ *J. Infect. Dis.*, 1906, 3: 191.

⁵⁸ *Food Poisoning*, University of Chicago, 1917, p. 18.

eating. *Amanita phalloides*, because of its white color, is mistaken for the common mushroom, *Agaricus campestris*. *Agaricus campestris* does not grow in the woods, neither has it white gills, nor white spores, nor a volva at the base of the stem. No dependence, however, should be placed upon color, size, shape or general appearance. It requires a trained mycologist to distinguish one species from another.⁵⁹

The first historic instance of mushroom poisoning occurred in the family of the Greek poet, Euripides, who lost, in one day, wife, daughter and two sons. Among others whose lives have been sacrificed through ignorance may be mentioned Pope Clement VII, the Emperor Jovia, Emperor Charles VI, Berronill of Naples, the widow of Tsar Alexis, and the Princess of Conti. Poisonous fungi have figured prominently in many of the accidental and craftily malicious tragedies of history.

Poisonous mushrooms contain at least four classes of poison: (1) a "toxin" represented by amanitotoxin;⁶⁰ (2) muscarin, an alkaloidal-like substance, resembling pilocarpin; (3) a hemolytic poison; and (4) a number of poisons more or less ill defined, such as the Pilz-toxin of Harmsen. These poisons do not all occur in any one species, but are found singly and in various combinations in the different genera and species. Ford⁶¹ gives a new classification of mycetismus, or mushroom poisoning: (1) gastro-intestinal, (2) choleric form, (3) nervous form, (4) hemolytic, and (5) cerebral form.

Amanita phalloides, the "white or deadly amanita," is the cause of the greatest number of cases of mushroom poisoning, if we include in this group *A. verna*, *A. bulbosa*, *A. alba*, *A. virescens*, *A. mappa*, and many other species known by various names in different localities. Fatal poisoning takes place when the fungi are eaten raw or cooked. Two or three deadly amanitas are sufficient to cause profound illness with fatal outcome in an adult. Plowright reports the death of a child of twelve from eating a third of the pileus of a small raw plant.

In 1891, Kobert isolated from *Amanita phalloides* a substance having a powerful hemolytic action which he called *phallin*. For some time it was believed that phallin was the essential poison of *Amanita phalloides*, but Ford⁶² showed that there is another substance present which is much more toxic, and to which most of the symptoms can probably be traced. This is supported by the fact that boiling *Amanita phalloides* mushrooms destroys its hemolytic power, but fails to neutralize its toxic action. This substance Ford has called "amanitotoxin" and states that it has no hemolytic action, but rather produces hemorrhage and causes necrosis and fatty degeneration of the parenchymatous organs. He also succeeded in producing an antihemolysin which completely neutralizes the blood-laking properties of phallin. Clark, Marshall and Rown-

⁵⁹ The subject has been discussed since 1886 in the *Bulletin de la Société Mycologique de France*.

⁶⁰ Ford, *J. Exper. Med.*, 1906, 8: 437.

⁶¹ *Tr. Ass. Am. Phys.*, 1923, 38: 225.

⁶² *J. Infect. Dis.*, 1906, 3: 191.

tree⁶³ found that in cases of *Amanita phalloides* poisoning, the pathological lesions consist chiefly of central necrosis of the liver, epithelial necrosis of the kidney, acute enteritis and colitis, the kidneys being the seat of the most marked changes. They conclude that nervous and mental changes observed are probably uremic in character, and not due to some peculiar "neurotoxin."

The symptoms of poisoning by *Amanita phalloides* usually do not develop for from six to fifteen hours after ingestion, the onset being marked by very sudden, severe abdominal pain, intense thirst, nausea, retching, vomiting and very profuse water evacuations, sometimes containing blood and mucus. A state of collapse may soon develop. There is usually a rapid loss of strength and flesh, and the patient develops a peculiar yellow color. The pupils are usually contracted; the breath is quite fetid and the mucous membranes dry and glazed, and there may be bleeding from the gums. Visual disturbances leading to confusion, delirium and convulsions may develop, but convulsions are usually due to a mixed intoxication, which in turn is due to *Amanita muscaria* being mixed with *Amanita phalloides*. After three or four days in children, and usually six or eight in adults, the patients sink into a profound coma from which they do not often awake. Ford⁶⁴ states that the case fatality rate of *Amanita phalloides* poisoning is from 60 to 100 per cent. The danger is much less in the case of *Amanita muscaria*.

Muscarin is the active poisonous principle of *Amanita muscaria* (*Agaricus muscarius*, "fly" amanita). Muscarin $(\text{CH}_3)_3\text{NOH} \cdot \text{CH}_2\text{OH}$ is a syrupy, alkaloidal-like substance obtained in crystallizable form as a hydrochlorid. It was first isolated by Schmiedeberg and Koppe in 1869. Chemically it evidently is an ammonia substitution compound and is classed with the ammonia bases. It may be prepared synthetically by the oxidation of cholin.

The physiological action of muscarin resembles pilocarpin very closely. It stimulates the myoneural junctions between the nerves and epithelial cells. Atropin is an almost perfect physiological antidote for muscarin, paralyzing the myoneural junctions, and is used with more or less success in mushroom poisoning.

The symptoms of muscarin poisoning come on quickly, often within fifteen minutes after eating the mushroom (*Amanita muscaria*). They consist of salivation, excessive perspiration, a flow of tears, nausea, retching and vomiting, pain in the abdomen, and violent movement of the intestines, causing profuse watery evacuations. The pulse is sometimes quickened, sometimes very slow and irregular. The pupil is contracted, respiration often quickened, and dyspneic. Dizziness and confusion of ideas are often complained of, but mental symptoms are not so conspicuous as those from the peripheral organs. Mental symptoms, such as hallucinations, delirium and convulsions are attributed to the Pilztoxin of Harmsen. Eventually the respiration becomes slower, great muscular weakness supervenes, but consciousness remains more or less clear until the breathing ceases.

⁶³ *J. Am. M. Ass.*, 1915, 64: 1230.

⁶⁴ *J. Infect. Dis.*, 1906, 3: 191.

The peasants of the Caucasus prepare an intoxicating beverage from *A. muscaria* which produces wildly riotous drunkenness. Death from muscarin orgies is not uncommon in this part of Russia. Similar species in North-eastern Asia are also used as an intoxicant. The poison is excreted in the urine which is sometimes later consumed for its intoxicating effect. It is probable that a sort of tolerance to muscarin is developed among the habitual users of the *muscaria* decoction.

The alkaloid is soluble in water and poisoning may be prevented by soaking the mushrooms in water slightly acidulated with vinegar before they are cooked.

Hemolysin.—Kobert, in 1891, obtained a hemolytic substance by alcoholic precipitation from *A. phalloides*. This substance he named *phallin*; it is an extremely complicated substance, having the nature of glucose; that is, it contains sugar in its molecule. It is not always present in *A. phalloides* and is probably not an essential poison in this mushroom, for its activity is destroyed at 70° C. and also by the action of the gastric juice. A high-grade immunity can be established in animals to the hemolytic substance. Ford obtained an antihemolysin which completely neutralizes the blood-laking properties of this poison.

The hemolysin probably plays a small rôle, if any, in human intoxication. *A. rubescens*, considered by the majority of mycologists to be an edible mushroom, contains a powerful hemolysin. On the other hand, a hemolytic poison is found in *Helvella* or *Gyromytra esculenta* which occurs rarely in this country. The active principle is helvellic acid (Boehm and Külz) which produces in dogs all the signs of hemolytic intoxication similar to those sometimes found in man.

Morner gives eight pages of references to mushroom poisoning.⁶⁵

Potato Poisoning.—Outbreaks of poisoning attributed to potatoes have occurred largely among troops. Schmiedeberg⁶⁶ reported outbreaks involving 357, 90, 125 and 101 men, respectively, and Pfuhl⁶⁷ recorded an outbreak involving fifty-six soldiers. The trouble was attributed to potatoes, sometimes new, sometimes old sprouting ones, and sometimes potato salad.

The onset of symptoms occurs usually only a few hours after eating the potatoes. Symptoms reported have been headache, abdominal pains, nausea, vomiting, diarrhea, prostration, dizziness, drowsiness, and slight delirium. Fever has usually been absent. In some cases, dilatation of the pupils is reported. The symptoms only very rarely become threatening and practically all the victims recover rapidly.

These cases were long regarded as solanin poisoning, but it is now plain that this does not account for most outbreaks, some of which at least are bacterial in origin.

Solanin ($C_{42}H_{75}O_{15}N_4$) is a glucosidal alkaloid closely resembling the

⁶⁵ *Upsala Läkaref. För.*, 1914, 24: Nos. 1 and 2.

⁶⁶ *Arch. f. Exper. Path. u. Pharmacol.*, 36: 1895.

⁶⁷ *Deutsche med. Wchnschr.*, 1899.

saponins in reaction, and found in many species of *Solanum*, such as *S. nigrum* (black nightshade), *S. dulcamara* (bitter sweet), and *S. tuberosum* (potato). The solanin content is highest in the potato peel, decreasing towards the center of the potato. It has also been held to occur chiefly in immature, sprouting or diseased potato parts which ordinarily are discarded before cooking.

Toxicologic tests on man have demonstrated that 0.2 gram of isolated solanin may provoke untoward symptoms. Seldom would that amount occur in the quantity of potatoes which an adult might consume in the course of a day. Owing to variations in purity, it is not possible to state how much solanin would be necessary to cause poisoning.

Meyer⁶⁸ showed that the solanin content of potatoes varied with the season, ranging from 0.04 gram per kilo during the winter to 0.116 gram during the summer. In vigorously sprouting and diseased potatoes, he found as much as 1.34 grams of solanin per kilo. Wintgen⁶⁹ also found a fluctuating solanin content in sound potatoes (0.017 to 0.08 gram per kilo), but the quantity was always small. Even in sprouted potatoes, he observed no increase in the solanin content, provided the sprouts were carefully removed. He was unable to find an increased amount in diseased potatoes. Wintgen was also unable to confirm the statement of Weil⁷⁰ that the solanin in potatoes was increased by the activity of two organisms (*Bacterium solaniferum non-colorabile* and *Bacterium solaniferum colorabile*). Harris and Cockburn,⁷¹ in an outbreak in Glasgow involving sixty-one people, showed that the incriminated potatoes contained 0.41 gram per kilo of solanin, an amount which they interpret as being five or six times the normal content.

Rothe⁷² studied a recent outbreak in Leipzig. The symptoms were abdominal pain, vomiting and diarrhea. An analysis of the same lot of tubers disclosed 0.43 gram of solanin per kilogram, which is about ten times the quantity found ordinarily in potatoes. The conclusion is drawn that the possibility of solanin poisoning must be reckoned with, especially when potatoes prematurely harvested are used as food.

It now appears probable that most instances of potato poisoning are not due to solanin, but are bacterial infections. Thus, Dieudonné⁷³ reports an outbreak of poisoning attributed to potato salad, in which the solanin content was only 0.021 gram per kilo. The symptoms were identical with those usually attributed to potato poisoning. *Bacillus proteus* was present in considerable numbers in the potato salad, and when grown on sterile potato medium, a highly poisonous culture was obtained which was fatal to mice in twenty-four hours. In most of the reported cases, the potatoes were prepared at least several hours before they were eaten, and had been left in places favorable to bacterial

⁶⁸ Arch. f. exper. Path. u. Pharmakol., 1895, 36.

⁶⁹ Ztschr. f. Untersuch. d. Nahrungs-u. Genussmittel, 1906, 12.

⁷⁰ Arch. f. Hyg., 1900, 38.

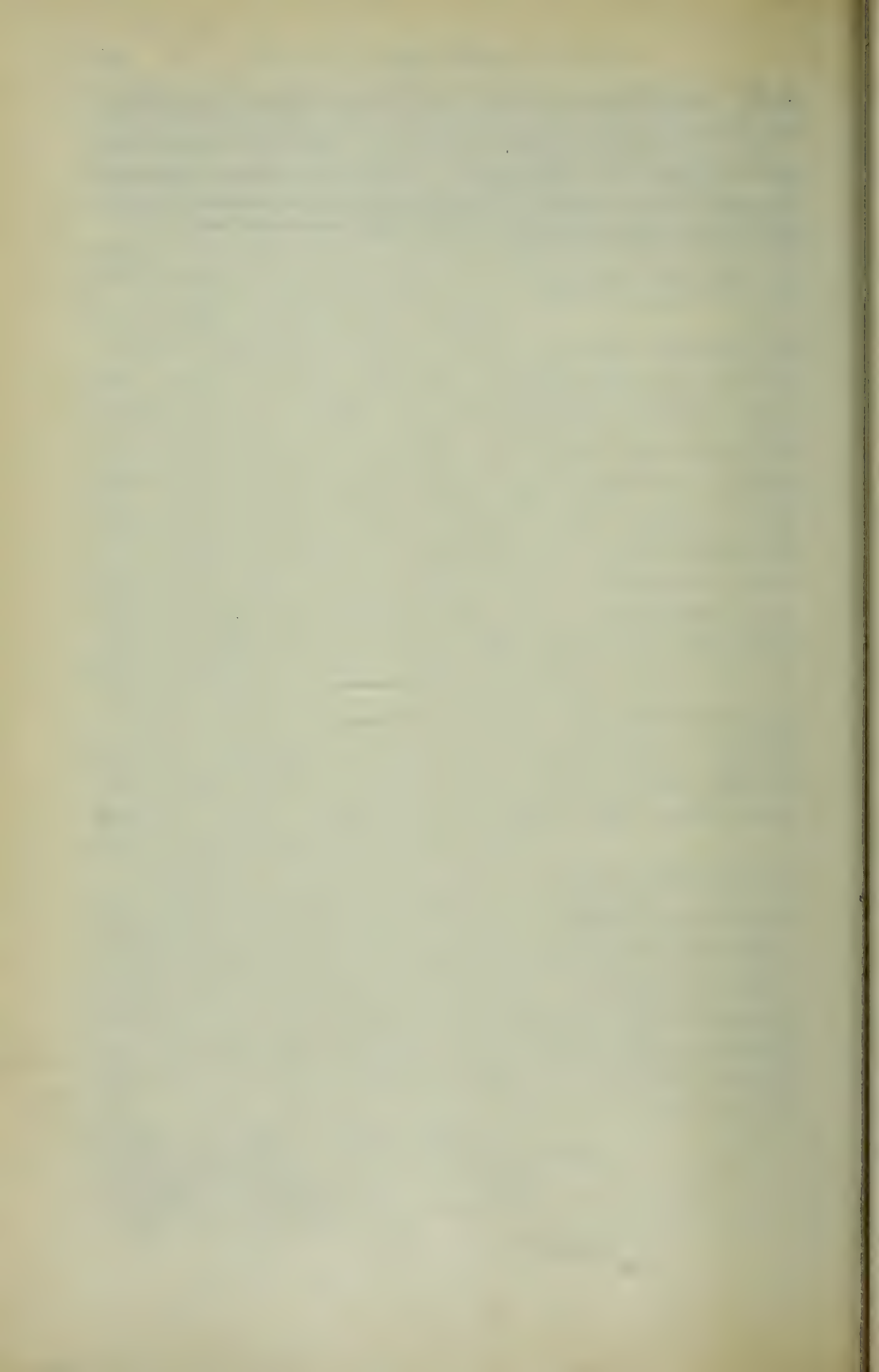
⁷¹ Analyst, 1918, 43: 7; Chem. Abst., 1918, 12: 1403.

⁷² Deutsche f. Hyg., 1919, 88: 1.

⁷³ Deutsche mil.-arztl., 1904.

growth. Potatoes make an excellent nutrient culture medium, even for typhoid and paratyphoid bacilli, *B. enteritidis* and many other pathogenic varieties.

The facts concerning solanin in potatoes are given as an example of the fact that many of our standard articles of diet contain poisonous substances, ordinarily in minute amounts. Extractives that are pharmacologically active can be extracted from practically every one of our wholesome foods.



SECTION VII

AIR

CHAPTER I

COMPOSITION OF THE AIR

The air constitutes a gaseous ocean in which we live; it consists of a vast mixture of gases at least fifty miles high.¹ Ordinarily we speak of this gaseous envelope of the earth as the *atmosphere*, and the water resting up the surface of the earth as the *aquasphere*, while the solid structure of the earth is called the *petrosphere*. Between the *atmosphere* on the one hand and the *petrosphere* and *aquasphere* on the other hand is the region of most abundant life, and this is spoken of as the *vivosphere*.

The importance of fresh air was almost completely ignored in practical life, but recently, thanks to the antituberculosis propaganda, its value has been recognized. While recent studies have shown that the air is not to be feared as a frequent medium for conveying specific infections, it has been demonstrated that an abundant supply of fresh air is necessary to perfect well-being. Statistical studies seem to prove that, of the predisposing causes to sickness which are usually in action, impurities of the air are perhaps the most important. This has been stated over and over again in the case of horses in stables, cattle in stalls and dogs in kennels, as well as of men confined in badly ventilated barracks, jails, and other places.

Many other factors are now known to be a greater menace to health than the "bad" air of crowded places; sanitarians, however, have come to regard an abundant supply of pure fresh air, well conditioned, as one of the real essentials for health and maximum efficiency. Many of the ill effects attributed to bad air are really due to crowding. Crowding causes overheating, which is the chief cause of discomfort in badly ventilated rooms. Circulating air, of proper temperature and moisture, carries off body heat and contributes to comfort. Crowding forces the occupants into close personal contact and thus favors the spread of many infections. It is a well-recognized principle in military hygiene that in a crowded barracks, with good or bad air, there will be an excessive amount of pneumonia, sore throats, colds and other inflammatory affections of the upper respiratory passages, which at times become epidemic.

¹ Fifty miles is its practical limit, and anything beyond that distance is in an extremely tenuous state.

While fresh air is so necessary to perfect well-being, nevertheless some people get along with surprisingly little, and that often vitiated, provided over-heating is avoided. Many people sleep huddled up, with their faces completely covered as though they would suffocate. In Holland many people sleep in an arrangement not unlike a closet, despite which they seem to retain rugged health. Dogs, sheep, and animals sleep huddled up, with their faces completely covered, sometimes in caves or dens where the air must be very bad. It is evident that the factor of safety must be very large, also that the question of habit plays a conspicuous rôle, for persons accustomed to good fresh air are rendered truly miserable when confined to a close, stuffy room.

Functions.—The two chief functions of the air that are especially concerned with health are (1) interchange of gases in respiration, and (2) regulation of bodily temperature. Further, it should be remembered that the combustion of the food we eat depends upon the oxygen of the air we breathe, and that digestion and metabolism are stimulated and improved by an abundant supply of fresh air or rendered sluggish and retarded by prolonged exposure to vitiated air.

Composition.—The atmosphere is now known to contain the following gases in the following approximate proportions measured at 0° C. and at 760 mm. pressure:

Gas	Volumes, per Cent	Weight, per Cent
Oxygen	20.93	23.2
Nitrogen	78.10	76.8
Carbon dioxid	0.03	...
Argon	0.94	...
Helium, krypton, neon, xenon, hydrogen, hydrogen per- oxid, ammonia, ozone	traces	...

Atmospheric air, in addition, contains water vapor in varying amounts, dust, radioactive substances, etc.

The air is a mixture of gases and not a chemical compound! The proofs of this are manifold: (1) The gases do not exist in the air in the proportion of their combining weights or any multiple of them; (2) on mixing the gases in atmospheric proportions there is no heat evolved; (3) the composition of air within limits is variable; (4) when water dissolves air it dissolves each gas according to its partial pressure and its own proper coefficient of solubility. Thus, air contains more nitrogen than oxygen, but, oxygen being more soluble, water takes up 1.87 parts of oxygen to 1 part of nitrogen.

The composition of the air shows wonderful uniformity all over the earth's surface wherever examined. This is due to the enormous amount of atmosphere and the mixing influences of air currents. However, in confined spaces where the air is not in motion, especially where decomposition of organic matter is taking place or where active combustion is going on, or in the presence of animal life, the composition of the air varies considerably.

The difference in composition between inspired and expired air, expressed as volumes per cent at 0° C. and 760 mm., is as follows:

Air	Oxygen *	Nitrogen	Carbon Dioxid
Inspired	20.93	79.04	0.03
Expired	17.	79.	4.

* Including argon and other inert gases.

The expired air is also warmer, is increased in volume, and contains more moisture, but fewer particles, such as dust and bacteria. Under normal conditions of quiet respiration the expired breath contains no bacteria.

Oxygen.—About one-fifth (20.93 per cent by volume, 23.2 per cent by weight) of the atmosphere consists of oxygen, which in many respects is its most important element. Slight differences are noted; thus, the air of towns contains somewhat less (20.87 per cent by volume) than at mid-ocean. The slight differences that have been noted in the percentage of oxygen have no special health significance. It may drop to 15 per cent or may rise to 50 per cent or even higher without any very apparent alteration in the vital functions. An atmosphere containing only 11 to 12 per cent of oxygen becomes dangerous, and 7.2 per cent results in death. In submarines, 16 per cent is the signal to replenish oxygen from the tank. A candle goes out at 16 per cent and an acetylene flame at 12½ per cent.

Haldane ² and his collaborators have shown that oxygen deficiency (anoxemia) increases the rate of respiration, whereas accumulating carbon dioxid increases the depth of respiration without essentially altering the rate. But when the carbon dioxid increases in appreciable amounts, both the depth and rate are influenced until panting occurs. Long exposures to an atmosphere containing a great excess of oxygen may act as an irritant. Karsner and Ash ³ found that rabbits show little effect when exposed for eleven days to an atmosphere containing oxygen between 60 and 70 per cent, but above this point irritation of the lungs and pneumonia supervene.

The amount of oxygen absorbed depends rather upon the needs of the body than upon the amount in the air. About 20 per cent of the oxygen in the inspired air is removed by respiration.

Alveolar air normally contains about 16 per cent of oxygen, and the red blood-cells, as they leave the lungs, are practically saturated with it. The amount taken up on their next trip through the lungs depends on how much they have given up to the tissues in the meantime, not upon how much is available for their use. The normal 16 per cent of oxygen in the alveolar air is automatically maintained by the action of the carbon dioxid on the respiratory center, but on account of the chemical affinity of the hemoglobin for oxygen the blood-cells may still take practically their full capacity of oxygen

²*J. Physiol.*, 1905, 32: 225; 1919, 52: 420.

³*J. Lab. & Clin. Med.*, 1917, 2: 4.

when it is reduced to 12 per cent or less in the alveolar air. In other words, a large excess of oxygen is constantly maintained in the air of the lungs. While it is one of the chief functions of respiration to supply oxygen to the body, neither a surplus nor a deficiency of it in the air, unless the alteration is extreme, has any effect on the respiratory movements. Breathing will not be lessened nor more oxygen taken up because more of it is breathed into the lungs; nor will the oxidation processes in the body be affected in any way, unless other influences are simultaneously brought into play. Indeed, Henderson reminds us that it is necessary to go only a short distance up into the mountains to come under an atmospheric pressure such as to reduce the oxygen supply considerably. Yet mountain air is especially healthful. Except in extreme conditions the amount of oxygen in the closest halls crowded with people practically never falls below 20 per cent. The amount of oxygen in the air apparently has little or nothing to do with the stimulating or depressing properties of the atmosphere breathed in ordinary life.

The constant percentage of oxygen is due in part to the enormous amount of it. Flügge estimates that at the present rate at which the oxygen is used by respiration and combustion it would take eighteen thousand years to reduce it by one per cent, even if not replaced by vegetation. The lungs, of course, at no time after the first breath contain air with the full percentage of oxygen. This is owing to the fact that the lungs do not completely empty themselves, and the residual air remaining in the lungs accumulates carbon dioxid and loses oxygen.

Oxygen is the element in the air that sustains all life. It is absorbed by the lungs, passes into the blood, combines loosely with the hemoglobin of the red blood-corpuscles, and is thus carried to all the tissues and cells of the body. Oxygen in combination with the hemoglobin forms an unstable compound—oxyhemoglobin—which gives the bright red color to arterial blood. The oxygen bound with the hemoglobin in arterial blood consists of from 18 to 22 per cent of the volume of the blood. The amount of oxygen absorbed varies with the age, condition of health, and activity. According to Professor Foster, the average person inhales in twenty-four hours about thirty-four pounds of air, which corresponds to a little over seven pounds of oxygen. As the lungs absorb about one-fifth of the oxygen inhaled, it appears that the average amount of oxygen absorbed daily is nearly two pounds. Oxygen also exists dissolved in its gaseous form in blood, saliva, bile, urine, and other fluids of the body, but only in minute amounts.

The amount of oxygen in the air may readily be measured in the Pettersson-Palmquist or Haldane apparatus. The oxygen is absorbed by 10 per cent pyrogallie acid in a saturated solution of KOH (sp. gr. 1.058); the difference in volume before and after absorption represents the amount of oxygen.

The variations in the amount of oxygen in the air ordinarily are not of sufficient magnitude to have hygienic significance, and such analyses are not made in routine examinations of air.

Nitrogen.—The nitrogen in the air may be regarded as a diluent, and as inert so far as its direct action upon man is concerned. There is no appreciable difference in the amount of nitrogen contained in inspired and expired air. Nitrogen does not “dilute” the oxygen, and thus regulate respiration in the same way that it controls the rate of combustion of substances in the air. Nitrogen is of more direct importance to plants, as certain genera are able to fix some of the atmospheric nitrogen through the action of bacteria, as *B. radiculicola*, in the root nodules of legumes. While nitrogen in the atmosphere seems to be an indifferent element and has no hygienic significance, it is a constant and important constituent of all protein matter. The amount of nitrogen dissolved as a gas in the blood and body juices increases proportionately with the pressure (P. Bert), and may lead to gas emboli, as in caisson disease.

Argon.—Argon, discovered in 1894 by Lord Rayleigh and Professor Ramsey, is quite inert chemically; that is, it has not been made to combine with any other element. It comprises from 0.75 to 1 per cent of the atmosphere. Argon has not been demonstrated in the body; it is apparently indifferent, and, so far as our present knowledge goes, has no hygienic significance.

Ozone.—Ozone, described by Schönbein in 1840, is rarely found in the air in greater proportions than mere traces, but it is so potent chemically that even small quantities may be of importance. At Montsouris, after years of observation, the largest quantity of ozone found in outside air was 1 part in 700,000. Ozone may be regarded as a normal constituent, though by no means constant in air. It is generally absent in the air of large towns and cities, and almost never present in the air of inhabited rooms. It is most abundant at sea and near woods.

Atmospheric ozone is formed in nature during electric discharges, by the oxidation of phosphorescent substances; and perhaps by the respiration of plants; also by friction of large masses of water, such as the sea against the air.

Ozone consists of three atoms of oxygen instead of two, contained in the molecule, thus: $3\text{O}_2 = 2\text{O}_3$. It is one of the most powerful oxidizing agents known, and in small amounts is exceedingly irritating; in large amounts it is fatal to life. Ozone is one of our most active bleaching agents, and in proper concentration is one of the most potent germicides known, and is used to sterilize water, to disinfect bandages, and for other purposes.

It requires at least thirteen parts of ozone per million in the atmosphere to influence bacteria. Such large proportions are never present under natural conditions. Comparatively small amounts are irritating to the respiratory mucous membrane. Thus, Hill and Flack⁴ have studied the action of pure ozone (free of contaminating oxids of nitrogen), and find it irritating in the proportion of one part per million. Exposure for two hours to a concentration of fifteen to twenty parts per million endangers life. Hill and Flack conclude

⁴ *Proc. Roy. Soc., Lond.*, B. 1911, 84: 404.

that there is no harm in breathing weak concentrations of ozone, such as can scarcely be perceived by a keen sense of smell.

Bohr and Maar found that any considerable concentration (even less than one part per million) diminishes the oxygen intake and the carbon dioxide output. The symptoms produced by exposure to ozone in addition to irritation of the mucosa are headache, restlessness, drowsiness, depression and coma.

Since ozone in concentration of one part per million parts of air is certainly injurious, and since this amount of ozone will not destroy inorganic odors nor kill bacteria, nor purify organic matter, it should not be used as a substitute for room ventilation. It cannot purify air in offices, schools, and other occupied spaces. Ozone destroys organic odors. Its action is similar to that of chlorin. Ozone is a deodorizer of powerful stenches, such as those from garbage incineration and fat rendering.¹ When the odors for chimneys, etc., cause a public nuisance, ozone and chlorin have commercial usefulness. Ozone masks but does not destroy the smell of inorganic substances, which do not react with it chemically. Ohlmüller⁵ demonstrated that ozone in considerable strength was incapable of killing dry bacteria within the time limits of his tests. Jordan and Carlson⁶ and also Konrich⁷ found that ozone ranging from 3 to 4.6 parts per million exerts no surely germicidal action, and that the alleged effect of ozone on the ordinary air bacteria, if it occurs at all, is slight and irregular even when amounts of ozone far beyond the limit of human physiologic tolerance are employed.

Human beings are injuriously affected by amounts of ozone far less than are necessary to produce even a slight bactericidal effect. Ozone, therefore, has no place in practical disinfection of occupied places.

The exaggerated claims of the deodorizing properties of ozone are not justified. Ozonizing machines can conceal faults in ventilation while not correcting them. These conclusions have been reached by Jordan and Carlson, Erlandsen and Schwarz, Hill and Flack, and Konrich, Sawyer, and others. Ozone is a poison rather than a purifier.⁸ See also pages 1023 and 1380.

Hydrogen Peroxid (H_2O_2).—Hydrogen peroxid may be found in appreciable traces in rain and snow. One liter of rain or snow water contains about 0.182 milligrams of hydrogen peroxid. This higher oxid gives many of the reactions of ozone, being a very active oxidizing agent, and care must be exercised not to confuse them.

Ammonia.—The ammonia in the air comes largely from the decomposition of organic matter. It is produced in sufficient quantities in a manure heap to be recognized by its odor and to sting the eyes. Ammonia may be regarded as one of the normal constituents of the atmosphere, as it is constantly present in slight traces; it varies in distribution, more being found in the lower stratum of air near the soil. It exists both in the free state and also combined as nitrate and carbonate. Daily analysis of the air at the observatory at

¹ *Arb. a. d. k. Gsndhsamte.*, 1893, 8: 229.

⁷ *Ztschr. f. Hyg.*, 1913, 73: 443.

⁵ *J. Am. M. Ass.*, 1913, 61: 1007.

⁶ *J. Am. M. Ass.*, 1913, 61: 1013.

Montsouris for five years gave, as a mean for ammonia, 2.2 milligrams per 100 cubic meters. There is less after rain, because it is absorbed by the water during its passage through the atmosphere.

"Albuminoid ammonia" (protein nitrogen), according to Angus Smith, is a measure of the sewage of the air; that is, the amount of organic impurities, both living and dead.

Inorganic Acids.—The atmosphere at times contains nitric, sulphuric, and other acids. These are derived from electric discharges, but mainly from the combustion of coal and from industrial processes. Sulphuric acid or sulphates in the air, according to Angus Smith, is a measure of manufacturing activity and also of decomposition. In other words, it is part of the oxidized and, therefore, purified sewage of the air. Traces of sulphuric and sulphurous acids exist in the air. The sulphates and sulphites are usually present as ammonia salts. These substances are usually present in such small amounts that they are appreciable only when washed into rain or snow. A liter of rain water may contain from 0.7 to 2.99 milligrams of sulphuric acid. More of this acid is found in the air about industrial centers than in the air over country or sea. The sulphuric acid in the air comes mainly from the sulphur in coal. The small amounts of acids in the air may be determined by hydrogen ion methods.

Carbon Dioxid.—Carbon dioxid (CO_2) is a very important constituent of the atmosphere. The amount of this gas in the air is relatively small—normally about 0.03 per cent, usually expressed as three parts in ten thousand. When we consider the immense bulk of the atmosphere the total amount of carbon dioxid is very great. It is estimated that there is more carbon in the form of carbon dioxid in the air than there is in all other forms on the earth. Formerly the amount of carbon dioxid in the air was stated as four parts in ten thousand, but repeated analyses with improved methods have shown that the correct amount is three parts or slightly more.⁹ There is apt to be more carbon dioxid in the air just above the soil than at a height of eight or ten feet. This is not because the carbon dioxid is heavy and settles, but because the soil air usually contains more of this gas. Air collected at great heights by balloons has just the same percentage of carbon dioxid as air at sea level. The air over the sea contains somewhat less than air over the land. Carbon dioxid in the air comes from the combustion and oxidation of organic matter, from respiration, from fermentation, from chemical action in the soil, and from mineral springs. The exhaled breath contains about 4 per cent of carbon dioxid.

Even a small alteration in the percentage of carbon dioxid, either up or down, would throw out of adjustment a long established balance, and this would alter the climate of the earth; it is believed, in fact, that the glacial periods were due to a slight decrease in the amount of carbon dioxid in the air.

⁹ Average of many analyses by F. G. Benedict is 0.031. *Carnegie Publications*, No. 166, 1912.

The carbon dioxid in the air is the source from which green plants with the assistance of sunlight obtain their carbon, and is thus indirectly the source of the carbon in the bodies of animals. The normal variations in the carbon dioxid of air in the open are too small to be of sanitary importance, and it is only when stagnant or inclosed air is polluted by combustion and respiration that we find accumulations which may have a bearing upon health. In narrow courts and in smoky air the free atmosphere may contain 0.7 to 0.8 per cent. In moving picture theaters the carbon dioxid may rise to 42 and even 72 parts in 10,000. Workshops may contain from 32 to 53 parts of carbon dioxid per 10,000, and breweries as much as 5 per cent and more. Its significance varies with its source. Enormous volumes of carbon dioxid are constantly being poured into the atmosphere. Manchester adds 8,000,000 cubic meters of carbon dioxid a day from the chimneys of industrial establishments. Even then the air of the city averages only 0.0385 per cent carbon dioxid, while the air of the country averages 0.0318 per cent—a very slight difference. It is estimated that from all sources 500,000,000 tons are discharged annually into the atmosphere. The reason that the carbon dioxid does not accumulate and increase is that it is constantly removed, especially by growing vegetation. Plants absorb enormous amounts under the influence of light and chlorophyl to build carbohydrates. It has been estimated that an acre of tree land withdraws in one season about $4\frac{1}{2}$ tons of carbon dioxid. Much of the gas is also absorbed by water, which at ordinary temperatures takes up its own volume. The ocean acts as a great regulator to keep the amount of carbon dioxid in the air constant.

The amount of carbon dioxid produced by respiration varies with the vitality, size, and activity of the individual. During violent exercise almost ten times as much carbon dioxid may be discharged as during sleep. On the average a man discharges about 0.6 of a cubic foot of carbon dioxid per hour and a woman about 0.4 of a cubic foot. During ordinary activity a man produces, in round numbers, one cubic foot per hour. An ordinary gas jet burns about six cubic feet of gas per hour and produces about three cubic feet of carbon dioxid. Therefore, so far as carbon dioxid is concerned, a man vitiates the air less than a gas jet.

Amount and Function of Carbon Dioxid in Alveolar Air.—Haldane and Priestly¹⁰ have shown that the regulation of breathing is largely dependent on the concentration of carbon dioxid in the air cells of the lungs, that is, the alveolar air. The concentration of carbon dioxid in the arterial blood is determined by the proportion of this gas in the air cells. The nerve cells in the respiratory center are stimulated by the carbon dioxid in the blood. Fixed acids will also stimulate respiration. Thus, beta oxybutyric acid is the cause of dyspnea in diabetic acidosis. An important factor in the stimulation of the respiratory center is the hydrogen ion concentration of the blood; any fixed acid which accumulates, or any excess of carbon dioxid, will vary the hydrogen

¹⁰ *J. Physiol.*, 1905, 32: 225.

ion concentration and thus stimulate the respiratory center. Ordinarily, this stimulation is largely due to the acid base equilibrium of the blood-plasma. The blood carries the carbon dioxid away from the tissues in exchange for the oxygen which it brings. This is internal respiration, to contrast with the reverse process which takes place in the lungs. The sodium bicarbonate and other buffers of the blood-plasma stabilize the hydrogen ion concentration of the blood.

The carbon dioxid which is being constantly formed in the body is carried to the lungs by the venous blood. It escapes from the blood into the air cells of the lungs and its escape is impeded or accelerated according to the resistance it meets in them. This resistance depends on the proportion of carbon dioxid in the alveolar air, since the tension of this gas in the blood can only fall as low as it is on the other side of the membrane separating the blood stream from the air cell. The arterial blood leaves the lungs with essentially the same pressure of carbon dioxid that is found in the alveolar air. In this way the alveolar carbon dioxid regulates the carbon dioxid tension in the blood and so controls the respiratory movements. This is true under normal conditions, but Haldane, Meakens and Priestly¹¹ have shown that if the oxygen in the air breathed falls to about 11 per cent, then the low percentage of oxygen stimulates the respiratory center to rapid, shallow breathing with increased pulmonary ventilation. Even at an oxygen tension of 18.1 per cent, Ellis found the respiratory volume increased.¹²

The breathing is so regulated as to maintain the percentage of carbon dioxid in the alveolar air at a pressure of about 5.5 per cent (5.3 to 6.3 per cent) of an atmosphere. If the pressure falls below this, in the normal individual respiration is lessened or stopped until the loss is regained. If it goes above the usual figure respiration is increased until the normal is restored. We are not aware of this mechanism, which is automatic.

Normally, the carbon dioxid tension of the alveolar air varies between 38 and 45 millimeters of mercury or 5.3 to 6.3 per cent. If abnormal acids are present in the blood, carbon dioxid is driven off from the plasma and as the tension falls in the blood it falls likewise in the alveolar air. A low carbon dioxid tension in the alveolar air during rest therefore indicates an accumulation of fixed acids in the blood, a so-called "acidosis." If the carbon dioxid tension lies between 32 and 28 millimeters of mercury, a mild acidosis is present and below 25 millimeters of mercury the acidosis is severe.

Haldane and Douglas¹³ found that from lying in bed to walking five miles per hour the amount of carbon dioxid expired was increased twelve times, and that the alveolar ventilation was likewise increased twelve times, so that the percentage of carbon dioxid in the alveolar air remained practically constant. Henderson¹⁴ found no material change in the composition of the

¹¹ *J. Physiol.*, 1918, 52: 420.

¹² *J. Physiol.*, 1919, 50: 267.

¹³ *J. Physiol.*, 1912; *Abst. in Brit. Med. J.*, Nov. 16, 1912, 1411.

¹⁴ *Am. J. Physiol.*, 1912, 29: 436.

alveolar air on going from rest to strenuous exercise. The increased production of carbon dioxide was perfectly compensated for by increased breathing.

There can be no doubt that there is a wide range of physiologic response on the part of the respiratory function to meet changing external as well as internal amounts of carbon dioxide. Thus, when more carbon dioxide is formed in the body the respiration is automatically increased in like proportion, and in this way the alveolar carbon dioxide is kept at a uniform level of about 5 per cent. The same thing happens when we breathe an atmosphere containing an excess of carbon dioxide. The volume of air breathed is then increased in such a degree as, if possible, to keep the carbon dioxide in the alveolar air normal. Haldane and Priestley¹⁵ found that with 2 per cent of carbon dioxide in the inspired air the pulmonary ventilation is increased 50 per cent; with 3 per cent it is increased about 100 per cent; with 4 per cent, about 200 per cent; with 5 per cent, about 300 per cent; and with 6 per cent, 500 per cent. With the last the alveolar tension of carbon dioxide is, of course, above the normal, and this fact is signified by severe panting; but up to 3 per cent in the inspired air the increase of breathing is scarcely noticed, unless muscular work is done, when the increased internal production of carbon dioxide calls for a still greater increase of the pulmonary ventilation. The adjustments are automatic and go on without our consciousness, unless an excessive increase of breathing is demanded.

Since even under the most favorable conditions we cannot avoid drawing back into the lungs some of the air that has just breathed out, not much hygienic importance can be attached to the slight variations in carbon dioxide content which ordinarily occur in the air of rooms. See pages 878 and 883.

Carbon Dioxide as an Index of Vitiatio.—For years the amount of carbon dioxide in the air has been generally adopted as the most convenient index of the total conditions which are usually prejudicial to health and comfort. The efficiency of ventilation also for years was usually determined by an estimation of carbon dioxide.

Carbon dioxide in itself is not irritating or poisonous. Large volumes may be taken in beverages or inhaled without noticeable effects. Effects are scarcely felt on the human system when the carbon dioxide reaches 2 or 3 per cent. The result of a concentration of 2 per cent carbon dioxide is simply to cause an automatic increase of 50 per cent in depth of breathing, such as occurs in moderate exercise. Respirations increase with the percentage, in depth only, until about 5 per cent, when there is distinct panting, and at 7 or 8 per cent the dyspnea becomes distressing; and headache, nausea, and chilliness may be noted. Observations made by W. G. Anderson in my laboratory show that these symptoms are more acute when the carbon dioxide is added to the air rapidly. Animals soon die when the percentage reaches 35 or 45 per cent in an artificial atmosphere. Man soon becomes unconscious and suffocates in an atmosphere containing 30 per cent of carbon dioxide.

¹⁵ *J. Physiol.*, 1905, 32: 225.

Benedict and Milner¹⁶ observed seventeen subjects kept for varying periods from two hours to thirteen days in a small chamber with a capacity of 189 cubic feet, in which the air was recirculated, but controlled chemically and physically. The carbon dioxid was usually over eight to nine times normal (over 35 parts per 10,000), often over one hundred parts, and sometimes 240 parts per ten thousand, and yet there was no discomfort so long as the chamber was kept cool. The air of submarines often contains 2.5 per cent carbon dioxid, which is breathed for hours without noticeable harm. In the ventilation of mines, the upper limit of carbon dioxid permitted is 2 per cent which is about the maximum in which persons can carry on severe muscular work.

Pettenkofer, in 1858, proposed ten volumes of carbon dioxid in ten thousand volumes of air as the limit for inhabited rooms. De Chaumont (1875) found that an unpleasant odor becomes perceptible in air containing six volumes of carbon dioxid in ten thousand, and fixed this as the limit, which for many years were accepted by sanitarians. It was soon learned, however, that the percentage of carbon dioxid may rise much higher before ill effects become perceptible. Carnelly, Anderson and Haldane in 1887 concluded that for the very crowded elementary schools a lower limit than thirteen volumes was not practical. Haldane and Osborne in 1902 recommended a limit of twelve volumes for factories and workshops at the breathing level, and that when gas or oil is used for lighting the proportion should not exceed twenty volumes. The general consensus of opinion to-day is that ten volumes in ten thousand is well upon the safe side, although, so far as carbon dioxid itself is concerned, more might be permitted without fear. Carbon dioxid is by no means the most mischievous of the constituents of vitiated air. It is not merely a waste product. It is one of the important hormones of the body. It helps to regulate the action of the heart, influences the tonus of blood-vessels, and stimulates the respiratory center.

It is certainly erroneous and unscientific to rely upon determinations of carbon dioxid in the air of a room as the sole measure of its conditions for respiration. Carbon dioxid never accumulates sufficiently in any ordinary room to become in itself serious; further, the amount of carbon dioxid in the air of a room gives no indication whatever of the moisture, the temperature, or the motion of the air of that room. Determinations of carbon dioxid are now used in ventilation as an index of poor circulation, that is, of stagnant air pockets, especially in auditoriums and theaters. Carbon dioxid is used as a measure of results, and not for the initial requirements which are based on thermal rather than gaseous estimations. While the amount of carbon dioxid, then, gives us a rough index of the degree of vitiation of the air, it affords no information concerning its physical conditions, which are of special importance.

The significance of carbon dioxid upon health is further discussed on page 878.

¹⁶ *Bulletin* No. 175, U. S. Dept. of Agriculture, 1907.

Methods of Determining Carbon Dioxid.—For the ordinary purposes of a sanitary analysis it is not necessary to make an accurate analysis of the carbon dioxid in air, such as the chemical analyst or the student of metabolism would make in scientific research. As the carbon dioxid in itself is not poisonous and is only an imperfect index of other impurities, and as its significance varies with its source, sufficient information may be gleaned for sanitary purposes from methods that give results relatively comparable.

The most accurate method of determining carbon dioxid in the air is that described by Petterson, and used in the Petterson-Palmquist, the Sonden or the Haldane apparatus. Both the Petterson-Palmquist and the Haldane methods are convenient, practical, and sufficiently accurate for all ordinary purposes. The method of Cohen and Appleyard is reasonably accurate and very convenient. The methods of Wolpert and Fitz give only rough estimates.

CHAPTER II

PRESSURE, TEMPERATURE, AND HUMIDITY

PRESSURE

Normal Atmospheric Pressure.—The pressure of the atmosphere at sea level is fifteen pounds to the square inch, or, as indicated in the barometer, it will sustain a column of mercury thirty inches or 760 millimeters. A man of average size living at sea level is exposed to a total pressure of about 34,000 pounds—more than fifteen tons. This great pressure must evidently have physiological importance. All the tissues and fluids of the body are subjected to this pressure and are in equilibrium with it. The interchange of gases on which life depends is largely a phenomenon of pressure. The pressure of the air also keeps the heads of the bones in their sockets without muscular action, and doubtless performs other functions less obvious. The small variations in pressure such as occur day by day at sea level have no evident physiological effects.

Diminished Atmospheric Pressure.—A diminution in atmospheric pressure is equivalent to breathing rarefied or diluted air. This is met with mainly in aviation or on mountain heights. The symptoms are due to the low partial tension of the oxygen. If oxygen gas is added to the inspired air at low barometric pressures, no discomfort is experienced. The dangers of rarefied air are those of oxygen deficiency—*anoxemia*. An adaptation response takes place under these conditions; thus, the acidosis increases the rate of breathing; and the number of red blood-cells increases, thus increasing the oxygen carrying capacity of the blood. The most important physiological effects of diminished atmospheric pressure are due to a diminution in the amount of oxygen absorbed, hence the breathing is deeper and the pulse rate quickened. As the altitude increases there is a lowered tension of oxygen in the alveolar air and a diminished tension of carbon dioxid. While the rate of respiration may be variously influenced in different circumstances, the depth of respiration is almost invariably increased. This of itself not only facilitates the oxygen supply, but also increases the elimination of carbon dioxid.

A compensatory increase in the number of red blood-cells takes place as a result of prolonged residence in high altitudes. Thus, assuming the average number of red blood-cells per cubic millimeter at sea level to be about 5,000,000, at Davos (elevation 1,560 meters) the number of red blood-cells averages 5,500,000 to 6,500,000. At Cordilleras (altitude 4,392 meters) the average number of red corpuscles is 8,000,000. A similar change in the

blood has been produced by keeping rabbits and guinea-pigs in rarefied air at sea level. According to Bürker, only a comparatively small increase takes place, amounting to 4 or 5 per cent, at altitudes of five or six thousand feet. The same moderate results have likewise been noted lately for much higher altitudes. Gregg, Lutz and Schneider¹ showed that where barometer pressure is rapidly reduced, as in aviation, there is a response by increased hemoglobin in the blood usually within twenty minutes.

At a height of eighteen thousand feet the pressure of the atmosphere is only half the pressure at sea level, thus:

<i>Altitude, Feet</i>	<i>Height of Barometer, Inches</i>
0	30
910	29
1,850	28
2,820	27
3,820	26
4,850	25
5,910	24
7,010	23
8,150	22
9,330	21
10,550	20
13,170	18
16,000	16
18,000	15

The highest dwelling place continuously occupied is the Observatory El Mirti, in the Andes, at 5,880 meters. The Observatory at Arequipa is at 6,100 meters. Thok Jalung is a village in the Himalayas at 4,980 meters. In Peru, Bolivia, and Northern Chili a very large part of the population live at about 3,000 meters. Potosi, which has numbered 100,000 inhabitants, is at 4,165 meters; Cerro de Pasco at 4,350 meters supports a native population of some 10,000, the mines of Villacota at 5,042 meters; the railway from Callao to Oroya culminates in a tunnel at 4,760 meters, almost the height of Mont Blanc. An annual fair is held at Gartok, at 4,598 meters, in the Himalayas, to which thousands annually come.²

Persons living in high altitudes believe that pneumonia is invariably fatal, and that relief is obtained by removal to a lower level. There is a high incidence of club fingers in persons living at high altitudes. This is doubtless a cardiovascular phenomenon similar to that seen in chronic and valvular heart lesions.

¹ *Am. J. Physiol.*, 1919, 302.

² Leonard Hill, *Recent Advances in Physiology*.

It is evident that man may become adapted to breathing a rarefied air at great heights, which would overcome persons if the change were made suddenly from sea level. Lindhard actually resided for twenty-six days in a pneumatic cabinet, becoming "acclimated" gradually to a reduced barometric pressure of 450 millimeters, at which he lived for two weeks.

Mountain Sickness.—The symptoms of mountain or aviation sickness are lassitude, headache, usually frontal and growing in severity, and sometimes nausea. The extremities feel cold, the pulse is weakened, respiration becomes deeper and more frequent. The face is pallid, but the lips and nails are cyanotic. Soon a helpless condition of weakness comes on, which renders the least muscular effort irksome and productive of shortness of breath, dizziness and palpitation. There is impairment of the senses of sight, hearing and touch, dullness of the intellectual faculties and a strong desire to sleep. Acclimatization or adjustment at ten thousand to twelve thousand feet may take place in two or three days in some persons. Sometimes symptoms are more severe, with vomiting, gastro-intestinal instability, fever, syncope, marked cyanosis and an increase in all the other symptoms which persist. The symptoms produced by a marked diminution in atmospheric pressure vary with circumstances. The effects are increased by cold, active muscular exertion, or improper clothing.

The direct effects of increased altitude are incomplete saturation of the blood with oxygen, and a resulting decrease of respiration which causes a loss of carbon dioxid. The chief compensations for these are an increase in the number of red blood-corpuscles per cubic millimeter of blood and a total increase in the volume of the blood; an increase in the quantity of the hemoglobin contained in each cell; deeper, and after a time, slower breathing; an increase in the size of the chest wall; a lowering of alkali reserve to compensate for decrease in the tension of the carbon dioxid in the blood; a lowering of the blood-pressure, which is, however, chiefly of a temporary nature and due to a temporary diminution of the blood volume, and vasomotor and other influences.

Schneider³ sums up our present knowledge of compensation to anoxemia as due to the following three factors: (1) increased respiration, (2) chemical alterations in the blood, and (3) increased hemoglobin. Since the alkalosis resulting from augmented breathing interferes with the passage of oxygen from the blood to the tissues, it cannot be questioned but that the restoration of the normal hydrogen-ion content, by the elimination of the excess of alkali, constitutes a compensatory process of almost if not equal importance with the increase in breathing.

Barcroft and his associates⁴ in a series of careful observations made at Cerro de Pasco in Peru confirmed the established fact that "seroche" was

³ *Physiol. Rev.*, 1921, 1: 631.

⁴ "Report to the Peru High-Altitude Committee," by J. Barcroft, C. A. Binger, A. V. Bock, J. H. Daggart, H. S. Forbes, G. Harrop, J. C. Means and A. C. Redfield, Jan. 23, 1923, *Phil. Tr., Lond. Ser. B.*, 1923, 206.

due to anoxemia, and that the compensatory mechanism is complex and not entirely clear.

The limit at which life may be sustained is about 26,000 feet, at which height consciousness is lost.⁵ At this height the barometric pressure of the air is 251 millimeters, which represents a pressure of oxygen of 52 millimeters, which is the equivalent of 6.8 per cent oxygen at sea level. Paul Bert remained twenty minutes in a pneumatic chamber with a pressure of only 248 millimeters without serious inconvenience.

Increased Atmospheric Pressure.—While man is often exposed to rarefied air, he is seldom subjected to increased pressure except under artificial conditions, such as in diving bells, diving suits, and caissons. The increase in atmospheric pressure in the deepest mines has little physiological significance. Divers and workers in caissons are not subjected to more than about $4\frac{1}{2}$ atmospheres, and work under such pressure for only a few hours at a time. When a diving bell is lowered ten meters into the water the air contained in it is compressed to one-half its original bulk, and the pressure of the air is accordingly doubled. Each ten meters' depth means an additional pressure of one atmosphere. At a depth of thirty meters, about one hundred feet, a diver is exposed to a pressure of four atmospheres or about sixty pounds per square inch. Bert exposed dogs to a pressure of ten atmospheres, and then slowly released them without harm.

While 2 per cent of carbon dioxide is the ordinary limit in which persons can do severe muscular work without discomfort, the limit necessarily decreases as the pressure increases. This is due to the increased partial pressure of the gas. Thus, with persons working under a pressure of two atmospheres, the limit will be one per cent instead of two per cent. This is of practical importance in the ventilation of diving bells and caissons.

The physiological effects of an increased atmospheric pressure are mainly due to an increase in the amount of atmospheric gases (especially nitrogen) which are taken up by the blood, and also an increase in the chemical absorption of oxygen by the blood. The serious consequences usually result from too rapid decompression. The nitrogen is absorbed by the tissues, especially by fat and lipid tissues contained in the nervous system. As the pressure is released gas bubbles form. Hence corpulent persons are more liable to suffer than lean; those with sluggish circulation suffer most. Gradual decompression gives a chance for the gas to escape from the lungs and be expelled without the production of gas bubbles.

Caisson Disease.—The effects produced by compressed air in caissons are: (1) those caused when the men are undergoing pressure, and (2) during or after decompression.

⁵ The aviators, Fleming and Steyer, in June, 1911, attained an elevation of 8,910 meters, but experienced grave symptoms which urgently called for the use of oxygen inhalations. With the aid of oxygen aviators have flown above thirty thousand feet—nearly six miles. Major R. W. Schroeder, in 1918, reached an altitude of 36,020 feet. Callizo, a French aviator, October 10, 1924, made an altitude record of 39,586 feet.

The symptoms produced by an increase of atmospheric pressure are a slowing of the respiration, which is evidently compensatory, but on account of compression of intestinal gases the respirations are deeper; the pulse is slower, and evaporation of water-vapor hindered. The voice may be altered; pains in the ear are common, due to pressure upon the drum, and may be obviated by swallowing air and thus passing it up the eustachian tube into the middle ear. Sometimes the ear drum ruptures; headache and dizziness may also occur. During compression the blood keeps absorbing the gases of the air until the tension of the gases in the blood becomes equal to that in the compressed air. As soon as this equilibrium has been attained relief from immediate troubles is secured.

It is during and after decompression that the greatest danger to health and even risk to life occur. The most frequent symptom is excruciating pains in the muscles and joints, called by the workmen "bends." These pains may continue for a few hours or for two or three days. Occasionally there is bleeding at the nose; also severe abdominal pain, and vomiting, nausea, vertigo, dyspnea, and unconsciousness. Death may result from internal hemorrhage, or paralysis may ensue—the so-called diver's palsy.

The effects of increased atmospheric pressure and too rapid decompression were carefully studied in 1878 by Bert, who showed that the lesions are caused by the escape of gases of the atmosphere which have been taken up in excessive amounts, and are released in the blood and tissues when the pressure is diminished. The blood-vessels may contain gas emboli, which may lodge in vital parts and cause sudden death, or the delicate capillaries may break, leading to hemorrhages with resulting paralysis. Air emboli may be distressing or dangerous if they occur in the labyrinth of the ear, in the spinal cord, in the brain, or in the heart or other vital parts.

The prevention of caisson disease consists in gradual decompression. Haldane⁶ advocates decompression by stages, instead of gradually, because men can be thus brought out more quickly and safely. The Bureau of Mines⁷ found helium an excellent substitute for nitrogen in diving. Due to its low molecular weight and increased diffusing power, it was found to lessen danger from the "bends" and permit quicker decompression time. In caisson work it is impracticable on account of the cost.

Sometimes the symptoms come on several hours after the workman has left the caisson. As soon as symptoms come on the workman should at once be hurried back into the compression chamber until equilibrium between the internal and external pressures is restored. He may then be allowed to pass through the decompression chambers, but very gradually. A medical air-lock should be provided at the works, well heated, and furnished with bunks and emergency supplies. Education is an important preventive against the dangers of compressed air.

⁶ *Respiration*, Yale University Press, 1922.

⁷ *Rep. Investig.*, Ser. 2670, Feb. 1925.

MOVEMENTS OF THE ATMOSPHERE

Moving air is necessary for the maintenance of health and is a prime requisite of good ventilation. The motion of the air serves the twofold purpose of bringing us a fresh supply and taking away the sewage-polluted air from our immediate vicinity. Moving air also favors evaporation and helps to prevent heat stagnation by keeping the temperature within normal limits. Paul, Heymann, and Erclentz, in Flügge's laboratory, and also Leonard Hill in England, emphasized the importance of moving air in assisting the heat regulation of our body. They believe that this is a much more important function of moving air than simply the bringing of fresh air or the carrying away of the products of respiration. In still air the body soon becomes surrounded by a warm, moist aërial layer which causes an overheating of the surface of the body and results in the familiar symptoms of "crowd poisoning." In a still atmosphere we are soon surrounded by a blanket of stagnant and impure air, whether indoors or outdoors.

Much of the benefit of mountain, seaside, and other health resorts is attributable to the breezes that blow almost continuously at such places. The health of large cities located upon the seacoast or the shores of great lakes is favored by the quantities of moving air with which they are frequently flushed. A healthful climate is always a breezy climate within reasonable limits. Much of the benefit of driving, of fanning, and of rocking-chairs is due to the motion of the air thus engendered.

If the air in a poorly ventilated room can be kept in motion, say with an electric fan, many of the ill effects of a vitiated atmosphere are avoided, for the products of respiration are diluted, and evaporation and heat interchange are favored. Thus, Leonard Hill placed eight students in a small sealed chamber which held about three cubic meters. He states that "at the end of half an hour they had ceased laughing and joking and their faces were congested. The carbon dioxid had gone up to 4 or 5 per cent. Three electric fans were then turned on, which merely whirled the air about just as it was. The effect was like magic; the students at once felt perfectly comfortable, but immediately the fans were stopped they again felt as bad as before." The effect of moving air varies with its velocity and especially with the temperature. This is discussed below.

In nature the atmosphere is kept in almost constant motion as a result of differences in temperature. Thus, the hotter air in the tropics rises and divides into two currents, which flow toward the north and south, while heavier, colder air rushes along a lower level from the north and south to take the place of the lighter currents. The cold currents from the poles are known as the trade winds, and the upper, warmer currents to the poles as the anti-trades. The upper currents to the poles run northwest and southwest; while the lower currents from the poles run northeast and southeast.

The chief cause of periodic winds, such as daily sea breezes and mon-

soons, is the difference in the heating of the air above land and above sea. On a small scale the same principle is seen at play in theaters, churches, cathedrals, and public buildings. The great mass of people crowded together heats the air about them and it ascends; cool air rushes it from the aisles to take its place, hence the almost unavoidable drafts in such places.

Measurement of Air Currents.—The velocity of air currents is customarily measured by means of anemometers. These instruments require a considerable velocity of air and should never be used without a carefully prepared table of corrections whereby their readings may be adjusted.

Portable anemometers measure velocities between about two hundred and two thousand feet per minute, while the fixed cup type of the weather bureau has a much wider range. For velocities of about 1,500 feet per minute and above, the Pitot tubes and Venturi meters apply, as in ventilating ducts. Below two hundred feet per minute, Hill's dry katathermometer gives good results. Anemometers are not sufficiently sensitive to measure currents below about 150 to 200 feet per minute.

It often becomes desirable in sanitary investigations, particularly in studies of ventilation, to determine the strength and direction of currents of air which are too delicate to be measured by means of anemometers. Lighted candles will show the direction of slight air currents, the flame being deflected in the direction in which the current is moving. More delicate than this is the method of noting the course taken by smoke from a joss-stick or cigarette.³ Engineers often use NH_4Cl or H_2SO_4 mist.

Perception of Air Currents and Drafts.—When a current of air at the temperature of 55° to 60° F. moves at a rate of one mile per hour, there is no perceptible draft. The rate of movement in relation to our perception is as follows:

Air moving at 1.5 feet per second—1.0 mile an hour—imperceptible.
 Air moving at 2.5 feet per second—1.7 miles an hour—barely perceptible.
 Air moving at 3.0 feet per second—2.0 miles an hour—perceptible.
 Air moving at 3.5 feet per second—2.3 miles an hour—draft.

The movement of warm air is less perceptible than the movement of cool air (for Drafts see page 247).

The effect of the movement of the air upon health is so closely bound up with humidity that this relationship is discussed on page 816.

TEMPERATURE

The temperature of the air depends mainly upon solar and terrestrial radiation. The air absorbs vast quantities of heat from the sun, and as the heat of the earth is radiated into space a certain amount is again absorbed by the atmosphere. Accordingly, the air both keeps the heat out and keeps

³ For a further discussion of this subject see *Air Currents and the Laws of Ventilation*, by W. N. Shaw.

it in. It makes the days cooler and the nights warmer. "It is a parasol at noon and a blanket at night." Except for it there would be much more violent changes in temperature (Macfie).

The power of the air to absorb heat and to store heat depends largely on its humidity and impurities in suspension; that is, on the amount of water vapor it contains, for water vapor is opaque to heat rays. The water vapor is also a great reservoir of latent heat. When water evaporates a tremendous amount of latent heat is carried up into the atmosphere with it and again becomes actual heat when the vapor condenses. The quantity of heat thus stored up in water vapor is almost incredibly great.

Air expands $1/491$ of its volume for each degree rise of temperature; air at 32° F. and thirty inches barometric pressure is usually taken for unit of volume. A cubic foot of dry air at 32° F. and thirty inches barometer weighs 566.86 grains; at any other temperature, therefore, its weight can be ascertained by dividing by its increased volume.

The temperature of the air has a very important bearing upon health. Man has an almost incredible power of adapting himself to wide variations of temperature. Workers in foundries have sometimes to endure a heat of 250° F. and even higher for short periods of time. Temperatures of -75° F. are met with in polar expeditions. This is a range of some 325° F. The reason that man, as well as other animals, is able to maintain a constant body temperature when exposed to such great variations of atmospheric temperature, is due not only to the physiological mechanism which regulates heat production and elimination, but to the layers of air immediately in contact with the skin. We wear clothes to protect ourselves from external heat or cold, but still more do we wear air for that purpose. That is why warm temperatures are better borne when the air is in motion, which facilitates evaporation, than when the air is still, while extremes of cold are better borne when the air is still, for then we become clothed in a warm blanket of air. The effect of heat upon health, however, cannot be considered alone, for it depends on the humidity as well as on the movement of the air. Extremes of heat and cold are much more trying when the air is humid than when the air is dry.

Exposure to high temperatures for a considerable time gives a concentration of the blood due to excessive evaporation of body water; an increased oxygen capacity, which can be explained by the concentration of the blood; a slightly increased oxygen content of the blood due to increased pulmonary ventilation; a rapid fall in carbon dioxid content and capacity; an increased hydrogen-ion concentration (alkalosis) due to the excessive pulmonary ventilation with a constant pumping out of the carbon dioxid without a compensatory loss of alkali from the blood. These are the forerunners or concomitants of incipient heat exhaustion. Concerning the concentration of the blood, there is a fairly high factor of safety, for it is not until it approaches 25 per cent that pathological symptoms occur from this cause.⁹

⁹ E. B. Flinn and E. L. Scott, *Am. J. Physiol.*, 1923, 66: 191.

The direct action of heat alone as a cause of infant mortality has been greatly underestimated. The harmful effects of heat must not be measured so much by the maximum or even the mean temperatures of the outside air, but by the indoor temperatures. Indoor temperatures may continue high in spite of remissions in the temperature of the external air. The withering effects of heat upon infants, and adults, too, are increased by the still, moist air found in overcrowded, narrow streets, and poor ventilation of houses.

Exposure of the body to dry, cold air has an effect similar to a cold bath; that is, there is an increased loss of bodily heat, followed by a demand for more. This demand is met by increased oxidation of tissue, metabolic processes become more active, and there is improvement in nutrition. It stimulates the chemical mechanism of the body for the regulation of bodily heat. In this way cold acts as a stimulant and tonic.

It is of first importance that the arrangements for heating rooms, offices, schools, etc., should be so regulated that the temperature never exceeds 21° C. (70° F.); especially should this control be exercised in public rooms, such as schools, etc. As a rule, the temperature of heated rooms should be 17° to 20° C. (62.6° to 68.2° F.) or under. Periodic variations of temperature are as desirable inside of buildings as in the outside temperature. The temperature of the air influences the temperature of the body, and also the general status of the vasomotor mechanism. Experiments clearly indicate that in the daily life of the school and factory the overheating of air seriously affects both health and efficiency. The atmospheric temperature also has a direct effect on diseases of the respiratory tract. Hill and Muecke,¹⁰ in England, and the New York State Commission on Ventilation¹¹ have shown that in going from a hot room to a cold room the membranes of the nose became paler and less moist, while the inferior turbinates contract. Such an action may in part account for the well-known seasonal prevalence of infections of the respiratory tract in the winter weather.

The effect of temperature upon health is so closely interwoven with humidity that this relationship is discussed on page 816.

Methods of Recording Temperature.—*Mercurial or bimetallic thermometers* are best suited to take the temperature of the air. The most accurate mercurial thermometers for this purpose have an elongated bulb of mercury at one end and a ring at the other, through which a cord can be tied; the scale should be etched upon the glass. A good thermometer of this type generally is accurate to about one-half to one-fifth of a degree. Thermometers placed upon a backing of metal, card, or wood, with the scale painted upon the backing, are more ornamental than accurate. They usually possess a decided lag and are, therefore, not trustworthy. Thermometers should be suspended freely in the atmosphere or at least placed in a current of air sufficient to insure good ventilation about the mercury column.

¹⁰ *Lancet*, 1913, 184: 1291.

¹¹ *Tr. Am. Climatol. & Clin. Ass.*, 1915; also Josephine Baker, *Am. J. Public Health*, 1918, 8: 19; also S. Mudd and S. B. Grant, *J. Med. Research*, 1919, 40: 53.

Registering thermometers are of two principal types: those which record maximum and minimum temperatures, and those which make a continuous record of the changes of temperature that occur.

The maximum and minimum temperatures furnish but limited information, and, as such self-recording thermometers are almost invariably mounted upon a backing, they consequently have a considerable lag. They are only dependable where fluctuations in temperature are not rapid. Under these circumstances they may be used to record the highest and lowest temperatures.

For an intelligent understanding of the sanitary condition of any room or inclosed space neither single determinations nor maximum and minimum records are sufficient. Recording thermometers should be placed at various selected points and records should be obtained covering a period of several days. The best type of recording thermometers are thermocouples or elongated mercurial thermometers with capillary type of bulb, both of which are very sensitive and accurate.

The temperature of the wet bulb thermometer should always be taken to determine the drying and cooling (psychrometer and katathermometer). For methods of recording temperatures with the katathermometer, see page 825.

HUMIDITY

Aqueous Vapor.—Water in its gaseous state is always present in the atmosphere. Water vapor is the most variable of the normal constituents of air, and also one of the most important, on account of its influence upon health. It is usual to consider water vapor apart from the other gases of the atmosphere, although it is just as much a gas as oxygen or nitrogen, and conforms to the general laws that govern the behavior of gases. Unlike other gases present in the air, water vapor condenses at comparatively low temperatures. As water vapor weighs only about three-fifths as much as air, dry air is heavier than moist air under equal conditions of temperature, pressure, etc. It is customary to speak of air "holding" water vapor. As a matter of fact, the air has nothing to do with it, for it should always be clearly observed that the presence of water vapor in any given space is independent of the presence or absence of air in the same space. The amount of aqueous vapor which a space contains depends entirely upon the temperature and not upon the presence of the pressure¹² of the air. At 32° F., for instance, the air can "hold" 1/160 of its weight of water vapor, at 59° F. 1/80 of its weight, at 86° F. 1/40 of its weight. Roughly, every 27° F. increase of temperature doubles the amount of water vapor the air can hold in proportion to its weight. In this way the heat of the atmosphere is self-protective, for it loads the air with water vapor, which in turn absorbs much of the heat.

¹² A high barometer retards evaporation, while a low atmospheric pressure accelerates it. All volatile liquids evaporate instantly in a vacuum. The rate of evaporation varies with temperature and pressure, but a given space will ultimately hold the same amount of gas independent of the presence and pressure of other gases.

The latent heat is again given off on condensation. The actual amount of water vapor which a cubic foot of air can hold at different temperatures is shown in the following table:

Temperature		Grains of Water Vapor
10°	F.	1.1
20°	"	1.5
30°	"	2.1
40°	"	3.0
50°	"	4.2
60°	"	5.8
70°	"	7.9
80°	"	10.0
90°	"	14.3
100°	"	19.1

As the temperature rises in arithmetical progression the power to retain vapor increases with the rapidity of a geometric series having a ratio of two.

The amount of water vapor in the air may be expressed either by: (1) Its *vapor tension*. The tension of the water vapor in the air is expressed in inches or millimeters of mercury. If a drop of water is placed in a vacuum, say in a barometer tube, some of the water vaporizes and the mercury is depressed, owing to the tension of the water vapor. The amount that evaporates, as well as the tension, depends upon the temperature. (2) Its weight per unit volume of air, i. e., the *absolute humidity*. (3) The ratio of the amount of water vapor in the atmosphere to the amount it could hold at the temperature in question if saturated; that is, the *relative humidity*. Complete saturation of the air with moisture is stated at 100, and lesser amounts by percentages. (4) The amount of water vapor in the air may also be found from its *dew-point*. The dew-point for any temperature and humidity is the temperature to which the air may be cooled when precipitation takes place.

The vapor tension or the absolute humidity indicates how much water vapor the air contains, while the relative humidity is an expression of how much vapor it might contain. The amount of water vapor which air can hold when saturated at different temperatures has been calculated and recorded in Glaisher's hygrometric tables.¹³ It is, therefore, very easy, by referring to these or to the tables in the U. S. Weather Bureau—*Bulletin Number 235*—to calculate the relative humidity if we know the actual humidity or the dew-point or *vice versa*. The moisture in the air is usually expressed in terms of relative humidity.

¹³ The standard hygrometrical tables in use the world over are those prepared by James Glaisher, F. R. S., of the Royal Observatory, Greenwich, England.

The amount of moisture which out-of-door air ordinarily contains varies from about 30 per cent or less to saturation.

In meteorological tables, giving climatic particulars of any town or

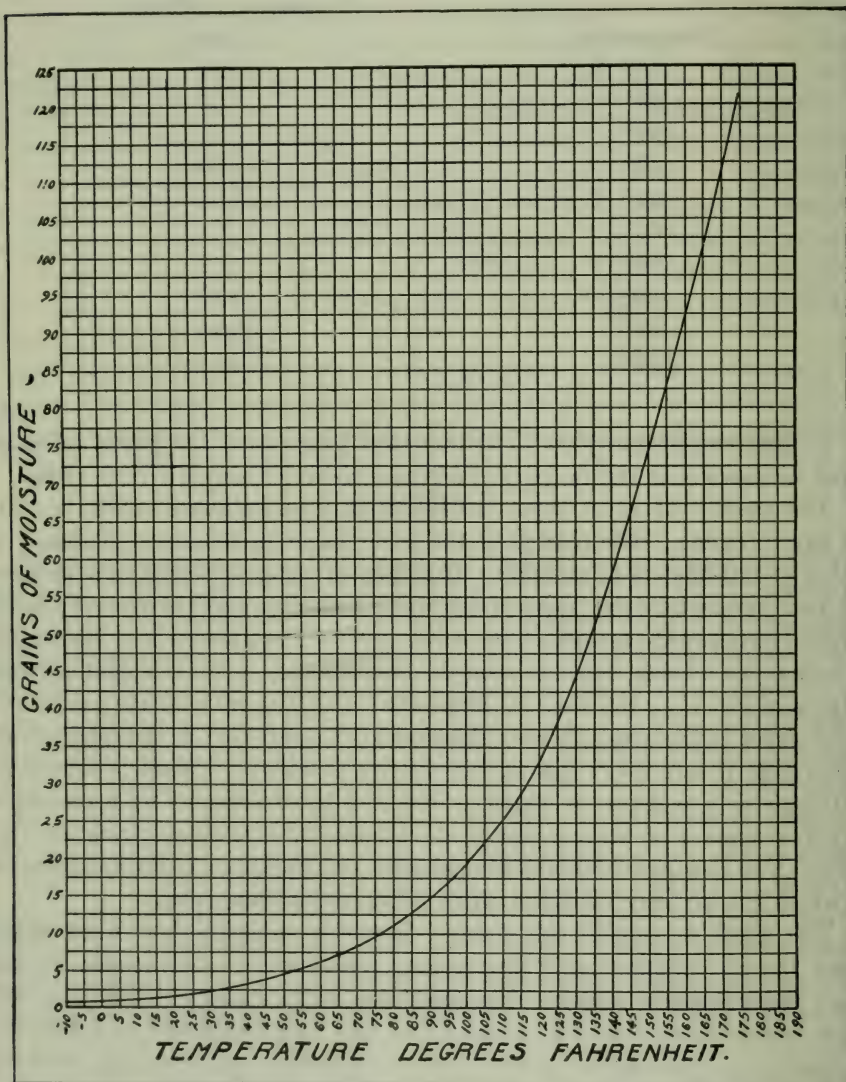


FIG. 64.—DIAGRAM SHOWING ABSOLUTE HUMIDITY IN GRAINS AT DIFFERENT TEMPERATURES.

locality, the relative humidity is usually stated; but it should be noticed that the relative humidity bears no constant relationship to the absolute humidity. As the relative humidity varies greatly throughout the day, and as the readings are not always taken at the same time of day in different localities, it at

once becomes evident that comparisons are not reliable. In fact, a moist or dry climate cannot be predicted from the relative humidity. Thus, the mean relative humidity of Davos is as high as 79 per cent, whereas it is generally known that the climate at Davos is dry. On the other hand, in Egypt the average relative humidity is very low, although this country is known to have a moist climate. This is for the reason that the humidity readings in Egypt are taken from 10 A.M. to 6 P.M., and vary from 30.5 per cent at Assouan to 51.7 per cent at Menahouse. As a matter of fact, the relative humidity in Egypt decreases from 100 per cent at dawn to 22 per cent at noon, and may be quickly altered to the extent of 50 per cent by a warm wind. The humidity, therefore, through the hot, sunny daytime is not a measure of the climate, so far as moisture and dryness are concerned.

In England the relative humidity averages 75 per cent. In California it drops from 100 per cent at dawn to 22 per cent at noon. A hot wind, by increasing the capacity of the air for moisture, may also lower the relative humidity very quickly. Thus, the Föhn wind when it reaches the Riviera lowers the relative humidity 50 to 60 per cent in an hour or two. The mean relative humidity of Denver for the year is only 42 per cent; at San Diego, on the coast, 72.9, at Los Angeles, a few miles inland, 66.6. In the heart of the Libyan desert the relative humidity may be as low as 9 per cent. At the seaside daily variations in humidity are less than inland (Macfie). The air in forests is 10 or 20 per cent more humid than air in the open. There may be a very great difference in the relative humidity of outside cool air and of air in a closed heated room, in that the latter may be very much drier.

So far as the effect of humidity upon health is concerned Huggard well states that the really essential point is not the amount of moisture, relative or absolute, that is present, but the amount that can still be taken up. This varies enormously with the same degree of relative humidity at different temperatures, as the following table from Renk will show:

Temperature, Centigrade	Relative Humidity, per Cent	Absolute Humidity, Gms. per Cubic Meter	Grams of Vapor That Can Still be Taken Up
-20°	60	0.638	0.426
-10°	60	1.380	0.920
0°	60	2.924	1.950
10°	60	5.623	3.749
20°	60	10.298	6.866
30°	60	18.083	12.056

We see by this table that the same expression, 60 per cent relative humidity, might be applied to air capable of taking up 0.426 gram or 12.056 grams of vapor, and thus the expression as a measure of the drying capacity of the air is obviously misleading.

Huggard gives a second very instructive table, the obverse of the above:

TEMPERATURE, CENTIGRADE	RELATIVE HUMIDITY, PER CENT	VAPOR, GRAMS PER CUBIC METER	
		Present	Capable of Still Being Taken Up
3°	0	0	6
10°	36	3.4	6
15°	53	6.8	6
20°	65	11.2	6
25°	73	16.9	6
30°	80	24.1	6

We see from this second table that air with relative humidities of 0, 36, 53, 65, 73, and 80 per cent, and containing quantities of water vapor varying between 0 and 24.1 grams per cubic meter, are all capable of further taking up exactly the same amount of vapor. Again the expression of relative humidity is misleading.

When the relative humidity reaches 80 to 85 per cent, moisture condenses and begins to show upon objects in rooms. This influences natural ventilation through porous building materials.

There may be a very marked difference between the humidity of indoor and outdoor air, owing in part to the condensation of moisture, especially in winter, upon the cold walls and windows.

The difference between external and internal humidities depends largely upon the temperature of the surfaces in the room. These surfaces, though apparently dry, may, in fact, hold moisture in large quantities; the walls and ceilings may contain more water than all the air in the room. Ordinarily there is a continual exchange of moisture between the air and the room surfaces. In this way the walls serve as a compensating reservoir to help maintain the humidity of the air approximately constant. Cold walls, cold windows, and cold surfaces generally condense the moisture from the air so rapidly that great difficulty is experienced in raising the relative humidity of the air of a room under these circumstances.

The humidity in the air is influenced by altitude. The higher we go the colder and rarer the air becomes; therefore, it contains less moisture. Its absolute humidity, therefore, decreases. Half of the total water vapor of the atmosphere is below 2,000 meters. On the other hand, the relative humidity shows no regular change with change of altitude. Clouds do not necessarily imply high relative or absolute humidity of the lower atmosphere. Rainfall also gives only a very general indication of the humidity of the atmosphere. A place with high rainfall may have low absolute and relative humidity, and *vice versa*; that is, a rainy district is not necessarily a damp district, so far as the atmosphere is concerned.

Dew also bears no constant relationship to the humidity of the atmos-

phere, for a clear sky and a dry atmosphere favor its formation. Air containing mist is obviously moist.

Normal Values.—The amount of moisture in the air conducive to health and well-being is often stated to be somewhere between 50 and 75 per cent relative humidity. These figures may be very misleading. There is no such thing as a normal humidity, for the amount of moisture in relation to health depends upon the temperature, clothing, motion of the air; also upon diet and muscular activity and other factors. Neither the relative humidity nor the absolute humidity nor the temperature of the air alone is a satisfactory guide as to its condition in relation to health. One factor alone gives the sanitarian scant information (see page 816).

Methods of Determining Humidity in the Air.—The amount of water vapor in the air may be determined either by (1) weighing, (2) psychrometers or hygrometers, (3) the dew-point.¹⁴

Weighing.—The amount of moisture in the air may be determined by passing a given volume of air through a tube or flask containing an hygroscopic substance, such as calcium chlorid or sulphuric acid. If sulphuric acid is used small flasks are filled with pieces of pumice which have been heated to a high temperature over a Bunsen burner, and dropped white hot in concentrated sulphuric acid; removed, and quickly drained.

The increase in weight represents the amount of moisture in the volume of air passed through the flasks, or the absolute humidity. Knowing the temperature of the air, it is then easy to determine the relative humidity by reference to tables of maximum water capacity for certain volumes of air at varying degrees of temperature. Engineers use graphic psychrometric charts (Fig. 65) which are reliable and convenient.

Psychrometers.—The most convenient of all methods for measuring atmospheric moisture is to observe the temperature of evaporation, that is, the difference between the temperatures indicated by wet and dry bulb thermometers. The United States Weather Bureau regards the sling psychrometer as the most reliable instrument for this purpose. In special cases aspiration psychrometers draw a given volume of air over the wet bulb in a given time, or rotary fans or other means may be employed to move the air rapidly over stationary thermometer bulbs.

The *sling psychrometer* consists of a pair of thermometers provided with a handle, which permits them to be whirled rapidly (see Fig. 66). The bulb of the lower of the two thermometers is covered with thin muslin, which is wet at the time an observation is made. This muslin covering should be kept in good condition and should be frequently renewed. It is also desirable to use pure water, because of salts and dirt which soil the muslin. The so-called wet bulb is thoroughly saturated by dipping it into distilled water. The thermometers are then whirled rapidly for fifteen or twenty seconds, stopped,

¹⁴ "The Measurement of Humidity in Closed Spaces," Food Investigation Bd., Spec. Rep. 8, April, 1925, H. M. Stationery Office.

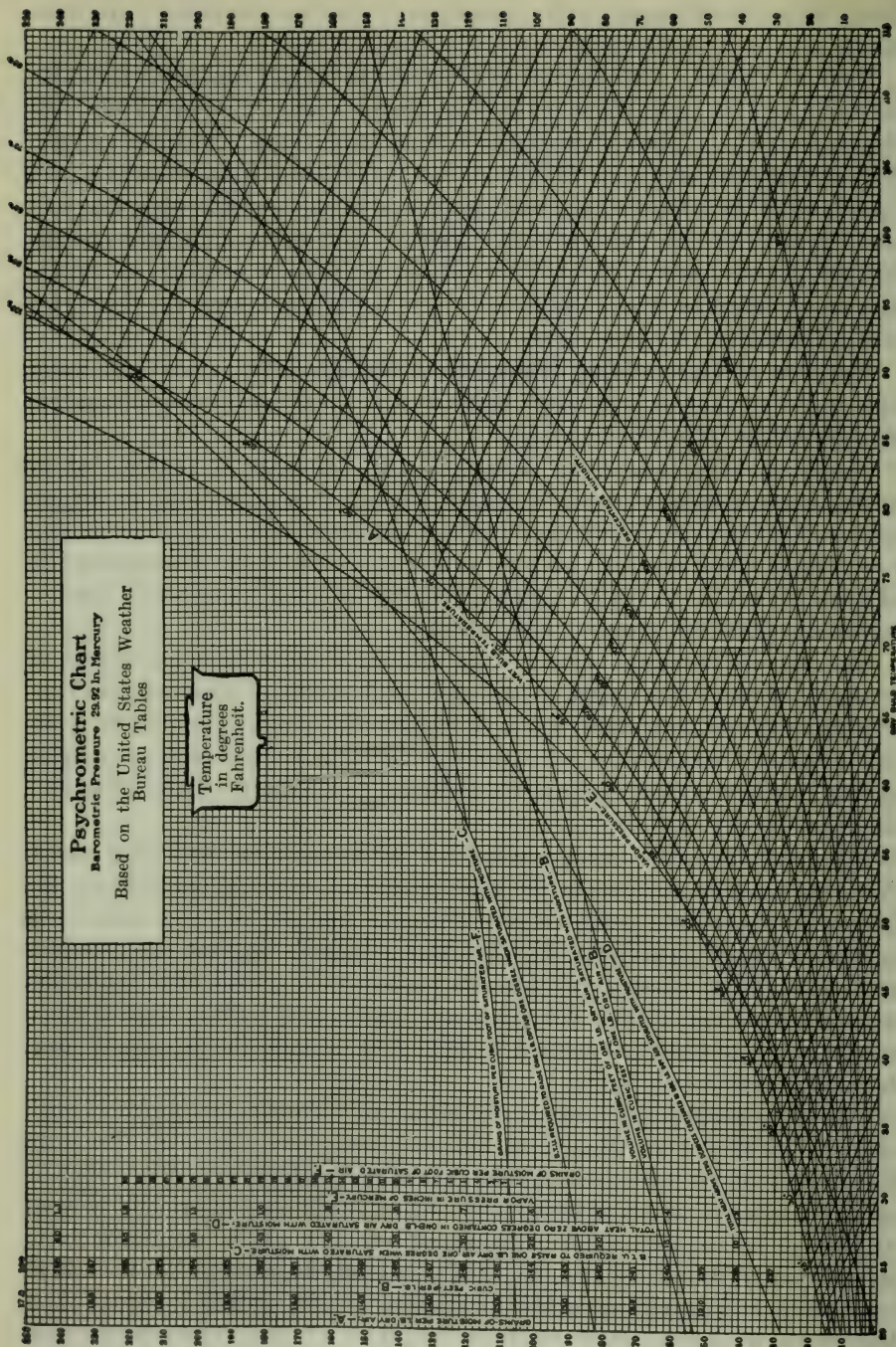


Fig. 65.—PSYCHROMETRIC CHART. (Courtesy Carrier Engineering Corporation.)

and quickly read, the wet bulb first. This reading is kept in mind, the psychrometers immediately whirled again and a second reading taken. This is repeated three or four times or more, if necessary, until at least two successive readings of the wet bulb are found to agree very closely, thereby showing that it has reached its lowest temperature. A minute or more is generally required to secure a correct reading. The psychrometer should not be whirled in the direct rays of the sun, and if used out of doors the observer should face the wind. It is a good plan, while whirling the instrument, to step back and forth a few steps farther to prevent the presence of the observer's body from giving rise to erroneous observations. Since the reading varies with the amount of air passed over the wet bulb, the speed and time it is whirled are important. The United States Weather Bureau recommends velocities of at least six hundred feet per minute. If this rate is exceeded no inaccuracies result, provided water is not blown off the wet bulb before it cools the mercury of the thermometer.

In correcting psychrometric observations the atmospheric pressure at the time must be obtained, and the results deduced from the tables based on a pressure nearest that observed. The difference in the temperature between the wet and dry bulb is computed to the nearest tenth of a degree. Having the temperature and the pressure of the air and the depression of the wet bulb, it is only necessary to read directly from the tables the dew-point, the vapor pressure, and the relative humidity. These tables will be found in *Bulletin Number 235* of the United States Weather Bureau. A condensed table is given in Figure 63.

The Hair Hygrometer.—This apparatus depends upon the expansion and contraction of a suitably prepared hair under the influence of moisture. It can be made a reasonably accurate instrument, and some types are arranged for continuous record. One of the principal difficulties with hair hygrometers is that a sufficient current of air does not always come in contact with them. They are sensitive but require frequent calibration.

The Dew-Point.—The dew-point can be determined accurately and quickly with a tin can and an ice mixture. Note the temperature at which fogging occurs as the thermometer drops, and check by adding warm water and going in the opposite direction.

The dew-point can also be determined from Regnault's apparatus. A polished silver cup contains ether which is

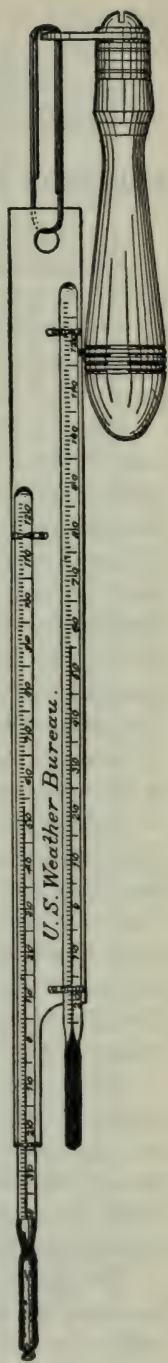


FIG. 66.—SLING PSYCHROMETER.

evaporated by a current of air. The evaporation causes cooling, and the temperature is noted at the point dew fogs the silver cup.

Direct determinations of the dew-point are ordinarily not made, for this can be estimated with accuracy from the wet and dry bulb readings.

RELATION OF TEMPERATURE, HUMIDITY AND MOVEMENT OF THE AIR TO HEALTH

Temperature, humidity and movement of the air each has its own particular and special effect upon health, but cannot be separated because always combined. Furthermore, the effect of each one of these three factors is influenced by the other two. It is therefore necessary to consider them in pairs or in combinations.

The physiological significance of moisture in the air varies with many factors, but especially with temperature. In a general way it may be said that moist air is depressing and enervating, while dry air is tonic and stimulating; also that cold air is invigorating, while warm air is enervating. Metabolism is slowed in warm¹⁵ air, quickened in cold air. The human body can adapt itself to wide variations in heat and humidity, and by means of suitable clothing and food the range may be greatly increased. Certain combinations of heat and humidity are trying or even hurtful; the most mischievous combinations are *cold damp* air and *warm moist* air, also an excessively dry air, especially when artificially warmed.

Excessive moisture makes hot air feel hotter and cold air colder—the first by hindering evaporation and the second by favoring conduction:

Humidity influences the output of heat from the body in two ways: (1) it increases the conductivity of atmosphere for heat—a cooling influence—hence cold moist air is chilling; (2) it interferes with evaporation of perspiration—a heating influence—hence warm moist air is enervating. Many climates in which people are reasonably healthy have a relatively high humidity, and some regions famed for their salubrity are notoriously dry and arid. Frequent changes of temperature, movement and humidity are beneficial and stimulating.

The vasomotor mechanism and the nervous control of perspiration are stimulated and made vigorous and efficient through moderately cool moving air, especially through changes in the temperature and motion of the air; through well-adapted clothing; cold baths within the limits of reaction; assisted by exercise, normal rest, proper diet, and other factors in personal hygiene that favor good nervous control.

The temperature and humidity of the air affect health mainly by influencing the heat-regulating mechanism of the body. More heat is produced within the body than is required, hence heat must be lost, else heat stagnation or heat stroke will result. Heat loss is influenced by the temperature, hu-

¹⁵ Metabolism is quickened as soon as the air becomes warm enough to raise the body temperature, but at this point symptoms supervene.

AIR TEMPERA- TURES.		DIFFERENCE BETWEEN THE DRY AND WET THERMOMETERS																														AIR TEMPERA- TURES.	
0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30			
30	100	89	78	67	57	47	36	26	17	7																					30		
35	100	91	82	73	65	54	45	37	28	19	12	3																			35		
40	100	92	84	76	68	60	53	45	38	30	22	16	8	1																	40		
45	100	92	85	78	71	64	58	51	44	38	32	25	19	13	7	1															45		
50	100	93	87	80	74	67	61	55	50	44	38	33	27	22	16	11	6	1													50		
55	100	94	88	82	76	70	65	59	54	49	43	39	34	29	24	19	16	10	6	1											55		
60	100	94	89	84	78	73	68	63	58	53	48	44	39	34	30	26	22	18	14	10	6	2									60		
65	100	95	90	85	80	75	70	65	61	56	52	48	44	39	35	31	28	24	20	17	13	10	6	3							65		
70	100	95	90	86	81	77	72	68	64	60	55	52	49	44	40	36	33	29	26	23	19	16	13	10	7	4	1				70		
75	100	95	91	87	82	78	74	70	66	62	58	55	51	47	44	40	37	34	31	27	24	21	19	16	13	10	7	5	2		75		
80	100	96	92	87	83	79	75	72	68	64	61	57	54	51	47	44	41	38	35	32	29	26	23	20	18	15	13	10	8	3	80		
85	100	96	92	88	84	80	77	73	70	66	63	60	56	53	50	47	44	41	38	36	33	30	28	25	22	20	17	15	13	11	85		
90	100	96	92	88	85	81	78	75	71	68	65	62	59	56	53	50	47	44	41	39	36	34	32	29	26	24	22	20	17	15	90		
95	100	96	93	89	86	82	79	76	72	69	66	63	60	58	55	52	49	47	44	42	39	37	35	32	30	28	25	23	21	19	95		
100	100	97	93	90	86	83	80	77	74	71	68	65	62	59	57	54	51	49	47	44	42	39	37	35	33	31	29	27	25	23	21	100	
105	100	97	93	90	87	84	81	78	75	72	69	66	64	61	58	56	53	51	49	46	44	42	40	38	35	33	31	30	28	26	24	105	
110	100	97	94	90	87	84	81	78	76	73	70	67	65	62	60	57	55	53	50	48	46	44	42	40	38	36	34	32	30	28	27	110	
115	100	97	94	91	88	85	82	79	76	74	71	69	66	64	61	59	57	54	52	50	48	46	44	42	40	38	36	34	33	31	29	115	
120	100	97	94	91	88	85	83	80	77	75	72	70	67	65	62	60	58	56	54	51	49	47	45	44	42	40	38	36	35	33	31	120	
125	100	97	94	91	88	86	83	80	78	75	73	70	68	66	64	62	59	57	55	53	51	49	47	45	43	42	40	38	37	35	33	125	
130	100	97	94	91	89	86	83	81	78	76	74	71	69	67	65	62	60	58	56	54	52	50	49	47	45	43	42	40	38	37	35	130	
135	100	97	94	92	89	86	84	81	79	77	74	72	70	68	65	63	61	59	57	55	53	51	50	48	46	45	43	41	40	38	37	135	
140	100	97	95	92	89	87	84	82	79	77	75	73	71	68	66	64	62	60	58	56	55	53	51	49	48	46	44	43	41	40	38	140	
0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30			

FIG. 67.—RELATIVE HUMIDITY TABLE.

midity and motion of the air. The first is of most importance when the temperature of the air is low, since the body heat is then removed actively by radiation, convection and conduction. The second, humidity, becomes predominant at high temperatures when the skin is covered with perspiration and most of the body heat is removed by evaporation. A stay of about three hours in an atmosphere at 40.4° C. (104.7° F.) and 95 per cent relative humidity may produce a rise of several degrees in the body temperature of an adult man. It will, therefore, be necessary briefly to review the mechanism by which the constant temperature of the body is maintained.

Heat Regulating Mechanism.—Under normal conditions the temperature of the body is maintained by an extraordinary mechanism which keeps a balance between heat production and heat loss. The former is brought about by chemical means—by the combustion of food material in the body; the latter by physical means—by radiation and convection, also by evaporation of sweat.

The chief source of the body comes from the food we eat.¹⁶ Approximately 80 per cent of the food we eat is used to furnish heat to maintain the body temperature, while only about 20 per cent furnishes energy in the form of motion. Heat is lost from the body chiefly in two ways: (1) by *heat transfer*, or loss by radiation, conduction, and convection, controlled almost entirely by changes in the dilatation and contraction of the blood-vessels of the skin; (2) by *evaporation*, chiefly by the evaporation of the water of perspiration, controlled by the varying activity of the sweat glands. Pettenkofer and Voit estimated the loss of water by the lungs at 286 grams, and from the skin at from 500 to 1,700 grams daily. This will give some idea of the magnitude of the effects here concerned. Pulmonary and cutaneous evaporation increases with the state of dryness of the atmosphere. It becomes almost double when there are five grams of water vapor instead of nine in one cubic meter of air. The benefit of drinking water freely when exposed to unusually high temperatures is very apparent. Hill¹⁷ estimates that a man loses 4.8 per cent of his body weight on a summer's day in twenty-four hours, and if he is working hard his loss is at the rate of 7.7 per cent. He warns against the loss of water to 10 per cent of the body weight. Barbour¹⁸ indicates that blood dilution occurs in response to external heat, whereas cold leads to a concentration of the circulating medium.

The loss by heat transfer diminishes as the temperature of the surrounding air rises. The temperature of the body would rise when the atmospheric temperature goes above 70° F. were not perspiration then secreted. As long as the perspiration can evaporate freely the heat production and heat loss are balanced. With a high humidity evaporation is lessened and the balance

¹⁶ An increase of heat production in a normal individual is due to the taking of food, the doing of muscular work, or exposure to cold. Fever, acidosis, and certain disturbances in internal secretions may also cause a rise.

¹⁷ *Recent Advances in Physiology and Biology*, Chaps. VII-XXI.

¹⁸ *Am. J. Physiol.*, 1924, 67: 366.

is maintained by rushing blood to the skin, which causes an elevation of the temperature of the surface, and thus the loss of heat by radiation, conduction, and convection is facilitated.

Air Motion.—Baetjer's experiments¹⁹ confirm our experience that some movement of the air is a factor of importance in practical ventilation, in that it contributes to our sense of well-being and comfort. It is not, however, entirely clear what physiologic reactions are concerned in producing this happy result. The play of a current of air upon the skin may directly stimulate the cutaneous sensory fibers (pressure, temperature) or may act indirectly upon metabolism or upon the vasomotor apparatus. It is well known that currents of air cause either local or general vasoconstriction or vasodilatation, resulting in changes in our cutaneous temperature sensations. Currents of air also have a physical effect upon the skin, influencing heat dissipation by convection and evaporation. This effect is influenced greatly by the temperature and humidity of the air. Rubner has shown that small or even imperceptible currents of air have a noticeable effect upon the loss of heat from the skin.

Another explanation why currents of air contribute to our comfort is that they act upon the sensory nerves of the skin, arousing cutaneous sensations of an agreeable kind. Baetjer indicates that currents as low as 0.03 meters per second give a perceptible stimulus to the sensory curves located in the skin around the eyes and mouth, and that currents as low as 0.15 meters per second give a perceptible stimulus to the sensory nerves of the cheek. The fact that the threshold value for velocity depends upon the temperature of the current of air leads us to believe that the temperature nerves are mainly concerned. The higher the temperature, the greater must be the velocity of the current to be perceived. Therefore, in ventilating a room the amount of air movement should be controlled by the temperature of the moving air.

A room at ordinary room temperature in which the currents of air are below the threshold limit, produces an uncomfortable feeling of closeness, while a room with currents much above the threshold values becomes unpleasant on account of drafts. Since an air current of threshold value is per-

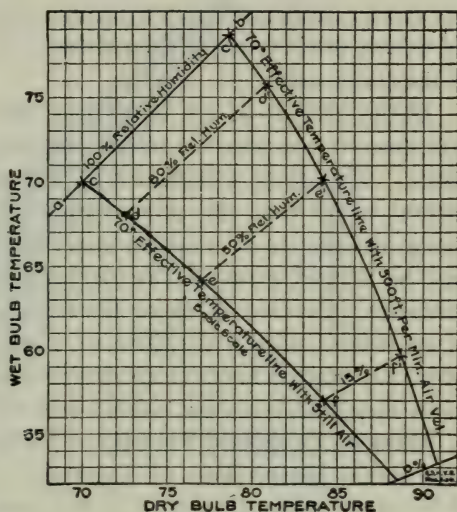


FIG. 68.—CHART SHOWING EQUIVALENT CONDITION OF TEMPERATURE, HUMIDITY AND AIR MOVEMENT IN STILL AIR AND IN AIR WHICH IS MOVING WITH A VELOCITY OF 300 FEET A MINUTE.

(From Yaglou, *J. Indust. Hyg.*, 1926, 8: 5.)

¹⁹ *Am. J. Hyg.*, 1924, 4: 650.

ceptible when it first strikes the skin, but ceases to be perceptible when continued for a few minutes, it is likely that variable air currents rising above and falling below the threshold velocity, and also varying in temperature, produce a series of mild cutaneous stimulations of agreeable character. The variation of temperature and movement probably explains the preference for window ventilation over the uniform monotony of mechanical systems. The New York Commission on Ventilation gives evidence that a window-ventilated room at 67° F. with fluctuating air currents, but without drafts, is fresher and more comfortable than a plenum-ventilated room at 69° C., in which the air flow is more uniform even though the cooling power of the air is greater in the latter case.

If the current of air is warmer than the surrounding air, it raises the skin temperature where it strikes the skin, due probably to local vascular dilatation. This gives a sensation of increased warmth. On the other hand, a current cooler than the surrounding air lowers the temperature of the skin, owing probably to vasoconstriction. In this case, through reflex action, there may be distant areas of vascular contraction with sensation of coolness or chilliness. In view of these facts, it is plain that in the case of crowded or overheated rooms, a system of ventilation which delivers the circulating air at a somewhat lower temperature than that of the room, and at or slightly above threshold velocities, should give the best results.

Value of Threshold Velocity.—Threshold velocity varies with the temperature but is independent of humidity. The threshold velocity for the skin of the cheek varies from 0.15 to 0.65 meters per second with current air temperatures varying from 12° to 30° C. When current air temperature equals normal room air temperature, the threshold velocity varies from 0.2 to 0.3 meters per second. Threshold currents with a temperature above that of the room by 1.1° to 7.8° C. cause a rise of temperature in the part of the skin acted upon, due presumably to vascular dilatation. Threshold currents with a temperature below that of the room by 1.2° to 7.0° C. cause a fall of skin temperature not confined to the part of the skin acted upon, due presumably to vascular constriction. The experiments indicate that the air movement in a well-ventilated room should not fall below the threshold value for the skin of the cheek.

There is a limit, for no beneficial effects are produced by moving air saturated with moisture at 98.6° F. to 100° F. even at high velocities; in fact, there is apparently some disadvantage.²⁰

Comfort Zone.—While the physical aspect of heat regulation is simple, its physiologic aspect is complex. Physiology demands that the heat interchange shall keep the body within a narrow temperature change to maintain a sense of comfort. If this becomes physically impossible, discomfort follows, and if continued, fever and collapse will ensue. However, large numbers of people remain comfortable and well in climates that would be trying if the

²⁰ R. R. Sayers, D. Harrington, *U. S. Pub. Health Rep.*, 1923, 38: 1616.

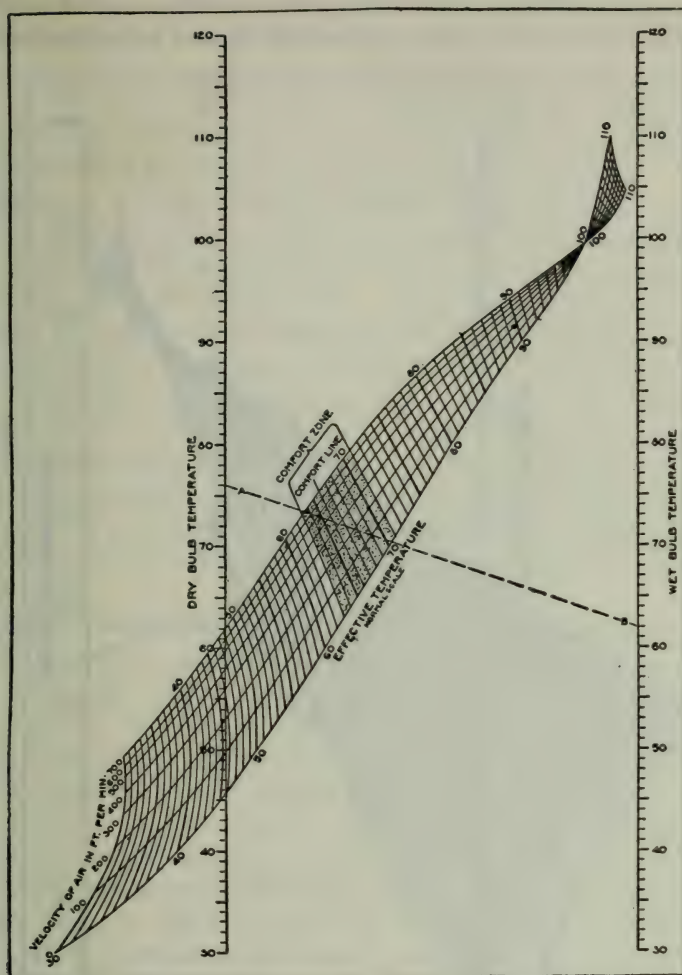


FIG. 69.—THERMOMETRIC CHART SHOWING NORMAL SCALE OF EFFECTIVE TEMPERATURE.
(MEN NORMALLY CLOTHED).

(From Yaglou, *J. Indust. Hyg.*, 1926, 8: 5.)

Example in Use of Chart

Given dry bulb 76° , wet bulb 62° , velocity of air 100 feet per minute, determine:
(1) effective temperature of the condition; (2) effective temperature with still air;
(3) cooling produced by the movement of the air; (4) velocity necessary to reduce the condition to 66° effective temperature.

1. Draw line AB through given dry and wet bulb temperatures. Its intersection with the 100-foot velocity curve gives 69° for the effective temperature of the condition.

2. Follow line AB to the right to its intersection with the 0 velocity line, and read 70.4° for the effective temperature with still air.

3. The cooling produced by the movement of the air is $70.4^{\circ} - 69^{\circ} = 1.4^{\circ}$ effective temperature.

4. Follow line AB to the left until it crosses the 66° effective temperature line. Interpolate velocity value of 340 feet per minute, to which the movement of the air must be increased for maximum comfort.

change were made suddenly. The body possesses great powers of adaptability in this regard within limits, which is one of the factors in acclimatization.

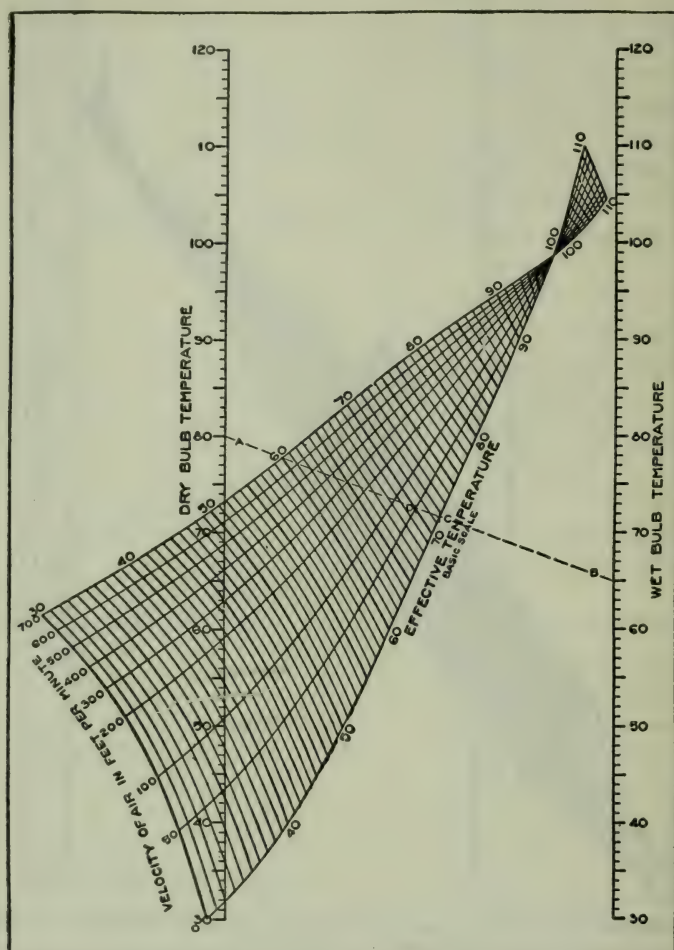


FIG. 70.—THERMOMETRIC CHART SHOWING BASIC SCALE OF EFFECTIVE TEMPERATURE. (MEN STRIPPED TO THE WAIST).

(From Yaglou, *J. Indust. Hyg.*, 1926, 8: 5.)

Example in Use of Chart

Given dry bulb 80°, wet bulb 65°, velocity of air 50 feet per minute.

1. Draw line AB. Its intersection with the 50-foot velocity curve at D gives 70° for the effective temperature of the condition.
2. Follow line AB to C and read 71.6° for the effective temperature with still air.
3. The cooling produced by the movement of the air is:
 $71.6^{\circ} - 70^{\circ} = 1.6^{\circ}$ effective temperature.

Heberden,²¹ one of the earliest students of the subject, realized the inadequacy of judging the comfort zone from the indications of any single instrument. Nearly one hundred years ago, he pointed out that the actual tem-

²¹ *Phil. Tr.*, Lond., 1826, Pt. II: 69.

perature of the air is only one factor in determining the effect of atmospheric conditions upon the temperature of the body. The humidity of the air, and particularly the movement of the air influence heat loss and body temperature. He conceived the idea of determining the combined effect of evaporation, convection and radiation by observing the rate of fall of an ordinary thermometer previously heated to a high temperature.

Haldane, in a series of articles between 1904 and 1907, concluded that at high temperatures the wet-bulb temperature was the more correct index. Bruce, in 1916, on the other hand, concluded that the dew-point rather than the wet bulb gave the truest index. These speculations led the Research Laboratory of the American Society of Heating and Ventilating Engineers, in coöperation with the United States Bureau of Mines and United States Public Health Service, to investigate the problem. This work included a study of pulse rate, rectal temperature, change in body weight, effect on systolic and diastolic pressure and work output. They find that *all three factors—dry bulb temperature, wet bulb temperature and air movement—*affect the human body in accordance with certain thermal laws which are called the effective temperature and which have been incorporated into an effective temperature scale.

Effective Temperature Index.—The effective temperature index is an index of atmospheric conditions developed by comparing the warmth sensed under a wide range of temperature, humidity and movement of the air. The comfort zone can therefore be determined by means of charts of the dry and wet bulb temperatures and the movements of the air (see Figs. 68, 69, 70). From Fig. 69 it will be seen that the comfort zone at rest includes conditions varying from 63° to 71° effective temperatures. A study of this chart discloses the fact that temperatures around 66° and 68° permit a wide range of humidity and motion compatible with comfort, although humidities in excess of 70 per cent and below 30 per cent are neither practical nor desirable. However, as we move above or below this temperature, a marked difference appears. In other words, the comfort zone is not a point. The conditions of humidity and motion vary with each degree on the scale.

The Comfort Zone placed at 66° to 70° by almost all early students of the subject agrees with the 66° effective temperature line and also with Huntington's optimum in his *Civilization and Climate*. Three separate groups of workers reach the same conclusion by different routes. This concurrence is interesting and significant. It is also in accord with everyday experience.

Effects of Clothing.—Rubner and his coworkers showed that the evaporation of water from the body cannot be regarded as being dependent merely on the percentage humidity of the atmosphere. The temperature of the layer of air in contact with the body is the factor of great importance. Thin clothes and still air, under certain conditions of external temperature, may favor evaporation, while nakedness and moving air favor conduction and radiation. The heat-losing mechanisms of the body are adjustable to varying conditions

within wide limitations, so that diminished loss by evaporation is compensated for by increased loss by conduction and radiation. The effect of clothing also varies with the fiber, the weave and the color.

According to Haldane,²² soldiers marching in uniform are liable to heat stroke at wet-bulb temperatures of under 21° C.; that, at 26.7° wet bulb, a marked rise of body temperature is noted with muscular exercise, and hard and continuous work is impracticable even when the subject is stripped to the waist; while at 31°-32° wet bulb, "in fairly still air the body temperature begins to rise, even in the case of persons stripped to the waist and doing no work; and when once started this rise continues until symptoms of heat stroke arise, unless the person leaves the warm air."

The effect of humidity with regard to heat output from the body is largely due to the increased conductivity of the clothing. At ordinary room temperatures and with people normally clothed, 1° F. of dry-bulb temperature is equivalent to about 2° F. wet bulb. In other words, an increase of 1° in the dry-bulb temperature of the air should be accompanied by a decrease of 2° in wet bulb, in order that the warmth of the atmosphere may remain unchanged. This is only true within the comfort zone, because around body temperature it was found that 5° F. dry bulb are equivalent to 1° F. wet bulb.

Effect on Temperature of the Body.—The extensive investigations of the New York State Commission on Ventilation have thrown some new light upon the general problem. These experiments dealt with the effect upon a large number of subjects of three atmospheric conditions: 20° C. with 50 per cent relative humidity (13° wet bulb); 24° C. with 50 per cent relative humidity (16° wet bulb); and 30° C. with 80 per cent relative humidity (27° wet bulb). At 24° wet bulb the average rectal body temperature of the subjects was 2 per cent higher and at 30° it was 5 per cent higher than at 20°, showing that the homiothermy of the human body is after all relative and not absolute, even within a moderate range of atmospheric temperature. A somewhat surprising observation was the close relation between the rectal body temperature at 9 A.M. and the mean air temperature for the twelve hours preceding. The curves were so perfectly parallel as to leave no reasonable doubt of the direct relation of cause and effect. There have been conflicting results reported by various observers who have compared body temperatures in the tropics and in the temperate zone; and it may be that after a prolonged sojourn in a warm climate a compensating mechanism is developed which maintains a lower body temperature with a given atmosphere outside. The diurnal variation in health rarely exceeds 1° C., despite extremes of external heat or cold or the irregularities in metabolism to which man may be subjected in the course of a day. Water is the central factor in the regulation of bodily heat. The effect of wide ranges of temperature, humidity and air movements on the body has recently been studied by Yaglou.²³

²² Eng. Dept. Com. on Humidity and Ven. in Cotton Weaving Sheds, 1910-1911.

²³ *Indust. Hyg.*, 1926. 8: 5.

Effect on Vasomotor Mechanism.—The New York State Commission on Ventilation found that in addition to a direct effect upon body temperature, atmospheric heat exerts a profound influence upon the general status of the vasomotor machinery as determined by the Crampton value. The Crampton value is an arbitrary index of the general tone of the vasomotor system, obtained from the relation between the changes in blood-pressure and heart rate on passing from a reclining to a standing posture.

The Katathermometer.—The katathermometer was devised by Hill in 1916.²⁴ The instrument is designed to measure the rate of heat loss from its surface at approximately body temperature. It consists of two specially constructed alcohol thermometers graduated from 95° to 100° F. One is used as a dry- and the other as a wet-bulb thermometer. The thermometers are heated in water to above the top gradation. The bare bulb is dried on a clean cloth, and the excess moisture is jerked off the silk covered bulb. The time taken to fall from 100° to 95° F. is noted best by the use of a stop watch.

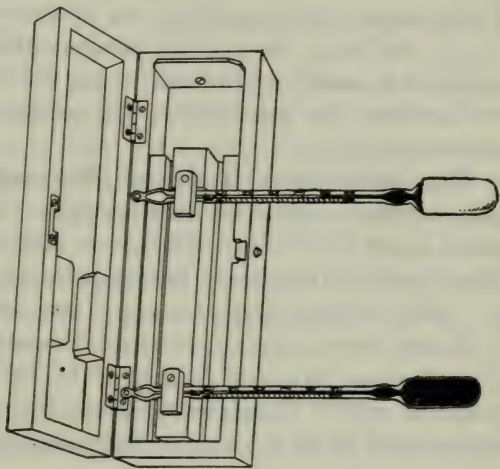


FIG. 71.—THE KATATHERMOMETER.

The rate of fall of both thermometers will obviously be affected by air movement and radiant heat as well as by air temperature, and that of the wet bulb by the humidity of the air as well. Hill believes that the combined influence of these factors will affect the katathermometers very much as it does the human body, and suggests a 45- to 60-second period for the wet bulb and a 150- to 180-second period for the dry bulb as limits for comfortable atmospheric conditions. Studies by Winslow²⁵ indicated that the lower of the limits set corresponds very closely to the average vote of a number of American observers as to bodily comfort. The lower limits may be shorter with advantage, but should not be exceeded.

Readings may also be taken with the thermometers (1) exposed to, or screened from, a source of radiant heat; (2) exposed to, or screened from, wind or draft; (3) with a thick knitted finger-stall placed over the bulb to imitate the effect of clothes. When this finger-stall is dampened, its efficiency is greatly diminished. Thus the cooling effect of damp clothes may be demonstrated. The effect of the color and texture of clothes may also be

²⁴ The science of ventilation and the open air treatment. Spec. Rep. Ser. 32 National Health Insurance, Medical Research Com., H. M. Stationery Office, 1919.

²⁵ *Science*, n. s., 1916, 42: 716.

demonstrated. A white finger-stall allows the instrument to cool, when exposed to sunlight, much quicker than a dark finger-stall. The latter absorbs the light rays and converts them into heat rays.

Hill, Griffith and Flack²⁶ presented, in 1916, a detailed study of the physical problems involved, in which the heat loss from the katathermometer is calculated in a more exact manner in millicalories per square centimeter per second. The wet katathermometer gives the rate of cooling by radiation, convection and evaporation. The dry katathermometer gives the rate of cooling by radiation and convection.

McConnell²⁷ studied the value and defects of the katathermometer. Yaglou also found inconsistencies in this instrument. He believes that although originally designed to measure the rate of cooling of the body, at present it is useful only for measuring air currents that have a velocity below two hundred feet per minute, and which cannot be determined by a vane anemometer.²⁸

The Effect upon Health of Wet and Dry Bulb Temperatures.—At temperatures around the freezing point, the dry-bulb temperature is the better index of comfort; whereas at high temperatures the wet bulb is the better index. At ordinary temperatures, the wet- and dry-bulb thermometers are about of equal importance for men stripped to the waist. If clothed, 1° F. dry bulb is equivalent to 2° F. wet bulb. During rest in still air a wet-bulb temperature of about 31.1° C. (88° F.) could be borne without any abnormal rise in rectal temperature, but above that the temperature rises, accompanied by an increase in pulse rate, profuse sweating and dyspnea, until finally exhaustion sets in.

Rubner states that an untrained man can be in comfort in a temperature of 75° F. and 80 per cent humidity (wet bulb about 70° F.) only when he is quiet. At 73.4° F. and 60 per cent humidity he found a resting man lost by evaporation 75 grams of water per hour, and at 84 per cent humidity (wet bulb 70° F.) only 19 grams. These figures show that three-quarters of the heat loss may be maintained by conduction and radiation when the wet bulb reaches 70° F.

Cadman concludes that at:

72° F. wet bulb, inconvenience is experienced, unless heavy clothing is removed and light clothing worn.

78° F. wet bulb, little inconvenience is felt if considerable bare body surface is exposed. Hard work is much facilitated if a perceptible current is passing over the body.

82° F. wet bulb, if clothes be removed, and maximum body surface exposed, work can be done providing current of air is available.

85° F. wet bulb, body temperature becomes affected, and only light work is possible.

²⁶ "The Measurement of the Rate of Heat Loss at Body Temperature by Convection, Radiation and Evaporation," *Phil. Tr., Lond., Series B*, 1916, 207: 183.

²⁷ *U. S. Pub. Health Rep.*, 1924, 39: 2293.

²⁸ *J. Indust. Hyg.*, 1926, 8: 5.

Boycott made the following significant observations upon himself:

"At rest and stripped I found that my body temperature rose rapidly if the wet bulb exceeded 88° or 90° F. with a dry bulb of about 100° F., though no rise occurred with a dry bulb of 110° F. and wet bulb of less than 85° F. I have on many occasions spent periods of about an hour in doing ordinary laboratory work in air with the dry bulb at 95° F. and the wet bulb at about 65° F. without any material discomfort. If, however, the wet bulb rises to 88° or 90° F., one's body temperature begins to go up, even when completely at rest, and one becomes exceedingly uncomfortable and on occasions feels very ill. These sensations can be, to some extent, remedied by local cooling of the skin (e. g., cold water on the head), but the rise of body temperature is progressive and must eventually end in heat-stroke."

A man is much less efficient in a warm moist atmosphere; hence it is an advantage to both employer and employee that work be performed at temperatures below 70° F. by the wet bulb. At the lower temperatures work is done faster, more efficiently, and with less fatigue, discomfort, and injury to health. To work in a warm moist atmosphere increases the temperature, pulse, and loss of moisture out of proportion to the work done. It is the master's pockets which suffer under such conditions, for the workers instinctively avoid the discomfort of overheating themselves through lessened exertion.

The New York State Commission on Ventilation clearly showed the effects of high atmospheric temperatures upon the working power, or more properly upon the actual performance of the working organism. Under a strong stimulus the power to do mental or physical work for a short time is not diminished even at 30° C. with 80 per cent relative humidity. "It is a matter of common experience that even a highly uncomfortable degree of heat is no hindrance to absorbing intellectual work and no bar to a good game of tennis" (Winslow). The disinclination to do active work at a moderately high atmospheric temperature may be interpreted as a conservative and protective process on the part of nature.

There is a fair range of adaptation or acclimatization to unusual temperature and humidity. Heat regulation, basal metabolism, circulatory and blood-making functions appear to remain unimpaired in the tropics under suitable conditions of living.

Effects of Warm Moist Air.—In a hot moist atmosphere an undue amount of blood is brought to the surface of the body, mental and physical activity is reduced, a feeling of depression is felt, and the resulting rise in temperature of the body influences the entire nervous and circulating systems. There is a disinclination to make a physical or mental effort; in other words, the effect is enervating. When air above 88° F. becomes saturated evaporation can no longer compensate for decrease in radiation, and the body temperature accordingly rises and heat-stroke may ensue. The injurious effects of the summer heat are practically always the result of combined heat and humidity.

According to Rubner and Lewaschew, when the air is very humid the heat loss by evaporation is very much lessened, and, accordingly, at 80 per cent

humidity and temperature of 24° C. (75.2° F.) becomes after a time insupportable to a man unaccustomed to it, and exposure to it is only possible with complete muscular rest. If, however, the air is very dry a temperature of 24° to 29° C. (75.2° to 84.2° F.) can be usually endured. These temperatures are often exceeded in the summer time in America. By practice a certain amount of accommodation to the effects of a hot moist climate may be acquired.

There is no known serious injury to health caused by working in a warm moist air, provided that a considerable rise of body temperature is avoided. The effects of heat and moisture may be diminished by light clothing, bare legs and arms, whereby the loss of heat from the skin is increased.

Working in moist, overheated rooms has the further disadvantage of wetting the clothes with perspiration, which causes discomfort, dirt, and untidiness, and liability to chilling the surface on going outdoors.

The effect on efficiency is not diminished power, but loss of desire to work. Winslow found that 37 per cent more work was done at 68° F. than at 86° F. This has been abundantly confirmed.

A poorly ventilated room in which the air becomes vitiated is usually a warm moist atmosphere, and the ill effects of a vitiated atmosphere are mainly caused by the heat and moisture. One of the most mischievous effects of a warm moist atmosphere is disinclination to mental and physical effort and loss of appetite.

Effects of Cold Damp Air.—Cold damp air causes a rapid loss of heat and chilling of the body. When such air is injurious the victim is usually underclad, improperly fed, or has been living an indoor life. In certain cases cold damp must always be injurious, as, for instance, where the vital forces are at a low ebb and where there is restricted capacity for making heat, such as infancy or old age; in cases of kidney disease, where hindrance of evaporation and increased metabolism means extra work for the kidneys; also in cases where there is a tendency to rheumatism or disorders of metabolism. The effects of cold damp air may be neutralized by proper clothing, by muscular activity, and, to a limited extent, by diet.

Just how cold damp air influences health is not well understood. It throws an added load upon the heat-producing mechanism to maintain the body temperature; the strain falls especially upon digestion, and metabolism, and also upon the circulation and the kidneys, and indirectly upon the nervous system. H. I. Bowditch, in 1862, formulated the law of soil moisture, and believed that tuberculosis was more common over moist soils than dry ones. According to our present conception, the relation between dampness or moist soil and tuberculosis is quite indirect; if there is any connection it is due merely to the fact that the combination of cold and dampness depresses vitality and thereby lowers resistance.

Some persons will shiver and their lips turn blue on a very cold, misty day, especially when facing the wind. This is doubtless a circulatory phenomenon, aggravated by the fact that moisture interferes with diffusion.

Under such conditions, the respiration is shallow, perhaps the result of a protective mechanism.

A healthy man may daily move in and breathe cold damp air without suffering in health to any appreciable extent; however, it is generally believed that a cold damp air predisposes to affections of the respiratory passages, to rheumatism, and neuralgias.

Effects of Warm Dry Air.—A relatively dry air feels better than moist air at most temperatures. The stimulating and pleasant effects of a dry climate can only be appreciated by one who has visited an arid region—such as our southwestern plateau. However, when air is abnormally dry, especially if warm, the evaporation from the body is greatly increased. Thus, Rubner and Lewaschew found that a man weighing fifty-eight kilograms gave off the following amounts of carbon dioxide and moisture in one hour at different temperatures in dry and moist air:

TEMPERATURE, CENTI- GRADE	DRY AIR			MOIST AIR		
	Relative Humidity of Air, per Cent	CO ₂ , Grams	H ₂ O, Grams	Relative Humidity of Air, per Cent	CO ₂ , Grams	H ₂ O, Grams
15°	8	32.2	36.3	89	34.9	9.0
20°	5	30.0	54.1	82	28.3	15.3
25°	6	31.7	75.4	81	31.4	23.9
29°	6	32.4	103.3

Air that is warm and at the same time abnormally dry, such as that produced by furnace heat,²⁹ causes an excessive loss of moisture and concentration of the fluids in the tissues and organs of the body. Man consists of 58.5 per cent of water. A very small percentage of loss may be serious; when the percentage reaches 21 per cent death results. The warmed and dried atmosphere of our overheated houses gives a sense of chilliness, owing to excessive evaporation, and favors irritation and infection of the respiratory mucous membranes.

Drinker³⁰ points out that during the months of central heating, increasing the humidity of the air permits the maintenance of lower temperatures, decreases dustiness, prevents the deterioration of plants and furniture, and improves the atmosphere from the standpoint of health and comfort. Moisture is lost initially through absorption by the contents of the residence and continually through air leakage and by condensation of moisture on cold surfaces, such as corridors. Warm, dry air is wasteful in that it increases fuel consumption.

The problem of constructing buildings in such a way as to keep the interior up to a fair degree of humidity is a large one. So far engineers have made

²⁹ See page 902.

³⁰ *Am. J. Pub. Health*, 1925, 15: 689.

little practical progress toward its solution. Satisfactory devices may be had to improve the moisture in large public buildings, but these devices have so far proved too expensive for private dwellings, offices, or schoolrooms.

The humidity in living rooms may be improved by setting about growing plants and porous dishes, such as flower pots full of water. If such receptacles or wet towels or wicks are set near electric fans evaporation is facilitated. Pans or pots of water may also be placed upon the radiator.

A *cool, dry air* is bracing. All the body functions are more active, breathing is deeper and more frequent, the circulation of the blood is increased; digestion, assimilation, and metabolism are stimulated.

CHAPTER III

MISCELLANEOUS

ODORS

People have always believed, and still naturally cling to the notion, that anything that smells bad must be detrimental to health. Science has demonstrated that our sense of smell is a poor sanitary guide. Smell is not primarily a protective sense. Its chief function among animals is to find food and mates. While disagreeable odors may not be harmful, they should be eliminated for æsthetic and psychological reasons, as well as for decency and cleanliness.

The viruses of most diseases have no odor whatever. Some diseases, as smallpox, are associated with a disagreeable odor. In no case is the odor diagnostic. Infections in water, milk or food cannot be sensed by smell.

Odors in a living room come mostly from human sources. The sources of these odors are: foul breath, decaying teeth, unclean mouths, nasal catarrh, sudoriferous glands, especially those of the pubes, feet, and axillæ, also gases from the stomach and bowels. The decomposition of matter on the skin and also in the clothes adds a very disagreeable odor, accentuated in a warm moist atmosphere. The peculiar odor in some rooms, especially sick rooms, seems to be none of these; just what constitutes the somewhat characteristic man-smell is not known.

While odors may be very unpleasant, they are not known seriously to influence health; contrary to common opinion, they are not by any means a reliable sign of danger. The presence of bacteria or dust in the atmosphere has no special relation to odors. Some poisonous gases, such as carbon monoxid, are practically inodorous.

The air of inhabited rooms ordinarily must be quite full of various scents which we do not appreciate, either because our sense of smell is not keen enough, or because we have become so accustomed to them that they are not noticed. An atmosphere that does not appear to be unpleasant while remaining in a room may seem intolerable upon returning to it after a period in the fresh outdoor air. Man's sense of smell is not keen when compared to that of some of the lower animals; nevertheless it is extremely sensitive to certain odors. Thus, it can determine 0.000,000,5 milligram of tincture of musk. The acuteness of the sense of smell varies markedly in different individuals.

Odorous molecules detach themselves from the surface of solids and liquids by simple evaporation, by oxidation and by hydrolytic decomposition. Cur-

rents of air will carry odors as they carry dust along with them to quite a distance. All odorous substances are freely soluble in oil.

It is well-known that we can perceive odors much more readily when the air is moist than when it is dry. It is also known that the mucous membrane of the nose must be moist in order that an odor be perceived. Moist rooms and people with moist clothes are sure to be smelly. Most fishes have a well-developed sense of smell.

Passy determined the least amounts of odorous matter that can be perceived by us. He gives the following figures per liter of air in which certain substances are dissolved and can be perceived.

<i>Substance</i>	<i>Milligram per Liter of Air</i>	<i>Substance</i>	<i>Milligram per Liter of Air</i>
Camphor	0.005	Sulphurated hydrogen	0.0005
Ether	0.004 to 0.005	Mercaptan	0.000,000,04

The olfactory nerves soon tire of most odors, and after a certain time, fail to respond. While in this condition they can at once perceive the sudden appearance of other odors. For example: Aronson found that persons having become insensible to the odor of iodine from continuous use found their perception to the odor of ether at once perfect. Ordinary ventilation does not remove the vapors which are held on solid surfaces by absorption. A jet of compressed air frequently played against the interior surfaces of buildings and against the clothing will remove this source of odors. Hence, one of the advantages of opening windows and thoroughly flushing out rooms from time to time.

When a room smells stuffy and close it may be taken as a fairly reliable index that the air is vitiated; this is especially true in a clean room not complicated with odors from clothing and sources other than man. In fact, the odors observed upon entering a room from the outside fresh air often furnish better evidence of imperfect ventilation or lack of cleanliness than laboratory tests.

De Chaumont made accurate observations and found that when the carbon dioxid amounts to six parts per ten thousand in an inhabited room, the atmosphere begins to smell close and stuffy. Pettenkofer found air containing 7.5 parts of carbon dioxid per 10,000 from the expired breath to have a marked odor, and ten parts a very unpleasant odor. With a little practice various grades of vitiated air can be detected up to ten or twelve parts of carbon dioxid per ten thousand.

The odors from marshes and from decomposing organic matter are not apparently hurtful. One of the most famous stench that has been recorded, if not the most famous, was that which arose in 1858 and 1859 from the Thames, which at that time was grossly polluted with the sewage of London (Sedgwick). Budd insisted that no very serious results followed. After giving

his proof Budd¹ states: "Before these inexorable figures the illusion of half a century vanished in a moment." We now know that odors in the air bear no reference to contagion or infection and, however unpleasant, need not be feared as such. Sewer "gas" is discussed on page 873.

The effect of odors upon health is not well understood. When we sense a pleasant smell we involuntarily take deeper breaths; on the other hand, unpleasant odors diminish the respiratory exchange. The latter are accordingly harmful to that extent and the former stimulating. Odors influence the nervous system in various ways; some stimulate, others depress psychic activity; some odors have a well-known influence upon sexuality. Occasionally odors are so disagreeable that they induce nausea, even vomiting. It is remarkable how quickly we may become accustomed to odors, but because our sense of smell has been dulled is no guarantee that the cause of the odors may not continue to produce its effects. Leonard Hill thinks that it is very doubtful if the unpleasant smelling exhalations of the bodies of men have any ill effects on men accustomed to them, and not of æsthetic temperament. Winslow and Palmer² found that "odors" of vitiated air have an unfavorable influence upon appetite.

Odors in a confined space are not removed by washing the air through a spray of water. The water absorbs some of the odors so that the wash water smells like a stuffy room. The odors may be neutralized with ozone or formaldehyd; or concealed by other smells. Organic odors from garbage incinerators and other forms of public nuisance may be cured with ozone or chlorin. Odors are an index of cleanliness. Clean rooms, clean stables, clean toilets do not smell.

LIGHT AND ITS EFFECT UPON HEALTH³

All the rays of the sun pass through the atmosphere before they reach the earth.⁴ The air acts as a differential filter, holding back many rays, especially those of shorter wave length; that is, the ultraviolet end of the spectrum. These rays have marked chemical and photodynamic action. We have already seen that some of the heat rays are also absorbed by the atmosphere. "More heat and we might be roasted, more light and we might be blinded, more chemical energy and we might be slain like the microbes."

The air as a filter of the sun's rays bears a very important but little understood relation to life. It is now well known that some of the sun's rays have intense chemical and "vital" power. We know something about the chemical

¹William Budd, *Typhoid Fever: Its Nature, Mode of Spreading, and Prevention*, pp. 148-151, London, 1873. This is a remarkable contribution which the student is advised to read.

²*Proc. Soc. Exper. Biol. & Med.*, 1915, 12: 141.

³The effect of light upon the eye is discussed in the chapter on the Conservation of Vision, page 451. The germicidal property of light will be found on page 1336 and 838; the illumination of schoolrooms page 1288.

⁴The waves of light are not waves of the atmosphere, but of the ether; however, they are absorbed, reflected or refracted by the dust and moisture contained in the air. It is convenient to consider light, as well as radio-activity, at this point.

rays, the luminous rays, and the calorific rays, but there are doubtless vibrations of which we know nothing. The cosmic rays of Milliken have just been disclosed. Macfie speculates that, "even, indeed, as the crops of the northern zone outstrip the crops in the south of France, so at certain times may the activity of nations be stimulated or depressed by atmospheric variations affecting the composition of solar radiation."

Life on the earth depends upon the radiant energy from the sun. Heat, sunshine, and other forms of radiation, such as x-rays, are physically of the same general nature, and differ from each other in their frequency which causes the variation in their action. Light induces or hastens many chemical reactions just as heat does. The greater number of the constituents of living cells are colorless; that is, they do not absorb rays of the wave-length of visible light. Many of them, however, absorb ultraviolet light so that radiation of this kind has a powerful effect on living cells. The effects of radium and x-rays on living tissue are destructive as are those of intense ultraviolet light.

Ultraviolet light and other rays of short wave-lengths have chemical and photodynamic powers which must have an important relation to health. They are potent and important rays from the sun and other sources of light. These rays act upon photographic negatives; hasten the hatching of flies' eggs and frogs' eggs; they sunburn the skin and produce freckles; they kill many bacteria, including the tubercle bacilli; they cause heliotropism; they combine chlorin and hydrogen into hydrochloric acid; they cause the oxidation of oxalic acid and other chemical reactions; they blacken silver salts.

Light of the visible spectrum is beneficial, even necessary to chlorophyll-bearing plants and many higher animals. On the other hand, ultraviolet light is deadly to some of the lower forms of life. Some functions are stimulated by light; thus, the sunflower and most leaves turn to the sun. Other functions of life thrive only in the dark, as the roots of most plants. Some small animals, such as tiny crustacea found in fresh water, flee from places "illuminated" by ultraviolet light. In sufficient strength and of appropriate wave-length, it kills many microorganisms.

The bactericidal action of sunlight is due entirely to the ultraviolet rays. These rays of short wave-length are further considered throughout this section, and also on pages 629, 633, 1037, and 1336.

Light may act in more than one way; it may inhibit pathologic processes or destroy pathogenic organisms, or it may conceivably promote some of the bodily functions that are perhaps dependent on a stimulus from without. In the case of rickets, light rays seem to supply something analogous to certain food factors. The knowledge of the curative action of ultraviolet light on rickets dates back only to 1919, when Hultschinsky reported favorable results with the mercury vapor quartz lamp; while Hess and Unger first demonstrated that successful treatment could also be secured with sunlight alone. Light ionizes the air, and the breathing of such air may have an influence upon health. Light probably affects health in other direct and indirect ways concerning which we are in blissful ignorance.

Physiologic Action.—The physiologic action of light is just beginning to receive the serious attention it deserves. We are all familiar with the calming effect of the dim religious light of churches and the stimulating effect of the glare of the "Great White Way." The intense light of the tropics and of high altitudes is believed in some way to bring on nervous disorders, but the relation is but vaguely understood. Some of the ill effects attributed to bad air and poor ventilation are due in part to the overstimulations of excessive illumination.⁵

The first systematic effort to study light and its therapeutic uses was made by Finsen when he founded his Light Institute in Copenhagen in 1896. Much valuable work, both theoretical and practical, has been done there since, with especial success on the therapeutic side, in the treatment of lupus, but the fundamental problem of the mode of action of light on the living cell remains unsolved. Recently, the rapidly accumulating clinical results of light treatment in tuberculosis, rickets, tetany, malaria, etc., as well as the results of x-ray and radium treatment, continually emphasize the biological importance of radiant energy and increase its mystery. It is at first disappointing to find that there is, apparently, in the animal kingdom no effect analogous to the action of light on the chlorophyll system of the green plant, by means of which light energy is stored and oxygen restored to the atmosphere. Although there is a universal conviction that sunlight is healthful, it is certain that man and other animals can live a long time in darkness without any noticeably bad results. Blessing, who acted as physician to Nansen during his expedition in the Fram, published a report showing that members of the party exhibited no evidence of anemia after the long Arctic night. More recently, Grober and Sempell examined horses that had worked for years in coal mines and found no anemia in any case where a satisfactory condition of nutrition existed. Cramer and Drew⁶ found that animals kept in a dark room since birth do not show any obvious signs of ill health. Nevertheless, they do differ from the normal; there is an abnormally low number of blood platelets. But, though the physiological effect of sunlight seems at first sight indefinite and of dubious importance, the action of far ultraviolet light on normal tissue, and the action of near ultraviolet and visible light under certain pathological conditions, have been investigated enough to show that there are well-defined effects due to light. These results are gradually assuming considerable importance and present theoretically an interesting but elusive problem in physiology. The subject is brought up to date in an article by Janet Clark⁷ which contains a good bibliography.

Desert climates may cause a slight though unmistakable increase in the red corpuscles. The most tenable hypothesis thus far advanced charges a

⁵ An illuminating discussion of the physiologic action of light will be found in W. M. Bayliss' *Principles of General Physiology*, London, 1915, a book which should be studied by all students.

⁶ *Brit. J. Path.*, 1923, 4: 271.

⁷ *Physiol. Rev.*, 1922, 2: 277.

stimulation of the blood-forming organs to peculiar and intensive light conditions that prevail in the desert. Thus it has also been shown experimentally that exposure to the light of the mercury arc may produce increases in erythrocytes in animals,⁸ and is being used in the treatment of pernicious anemia. If this proves to be correct, a new aspect of climatology will have been emphasized in the influence of sunlight upon blood-making organs.

Freer,⁹ Chamberlain¹⁰ and others have carefully studied the effect of a tropical climate on soldiers in the Philippines, especially in reference to the effect on blonds. They found that all soldiers, irrespective of their coloring, reacted in the same way to life in the tropics. They showed loss in weight, higher pulse rate and rate of respiration, and a lower blood-pressure. They had a high red count and low color index, and the differential white count showed a high percentage of lymphocytes and a low percentage of polynuclears.¹¹ The same changes in pulse, respiration and blood-pressure can be brought about by moist heat,¹² and it is quite possible that the heat may also be responsible for the blood changes. On the whole, the work in the Philippines showed that the deleterious effects of the tropics are due to heat and not to light.

The effect of light on the blood, however, does not strikingly change the erythrocyte count. There is a temporary decrease in the red count and the percentage of hemoglobin in the dark and an increase in the light after long exposures. On the contrary, the white blood-cells, especially the lymphocytes, respond to short exposures of any radiation (sunlight, ultraviolet light, x-ray, heat). Ultraviolet light stimulates a lymphocytosis in man and animals. The production of lymphocytosis, as well as the formation of pigment by radiations in the luminous part of the spectrum and by ultraviolet light, may well be an important factor in heliotherapy.

Light exerts some effect on body metabolism, for there is a change in the amount of carbon dioxid expired, a change in rate and depth of respiration, and an increased rate of growth in the light as compared to the dark. Recent experimental work on rickets and tetany has given evidence that light is concerned in the phosphorus and calcium metabolism of the body. In other words, it acts like vitamin D. Light, however, does not take the place of vitamin A.

Sunlight.—Sunlight has an important bearing upon health which is highly beneficial. Excessive exposures to sunlight may be harmful; on the other hand, we have snow-blindness in the arctic zone, and sunburn in any climate. The radiant energy of the sun in outdoor life has a most important influence on the surface temperature of the skin and our feeling of comfort. Sunlight is only one of the numerous environmental factors to which we are exposed. Each portion of the spectrum produces special effects: thus, the action of

⁸ Bickel and Tasawa, *Charité-Ann.* 37, 1913.

⁹ *Phil. J. Sc.*, B., 1910, 5: 1.

¹⁰ *Ibid.*, 1911, 6: 427, 467, 483.

¹¹ W. A. Wickline, *Mil. Surg.*, 1908, 23: 282.

¹² J. M. Phalen, *Phil. J. Sc.*, B., 1909, 4: 273.

chlorophyl depends upon the visible spectrum; other effective rays in sunlight seem to be between 290 $\mu\mu$ and 330 $\mu\mu$. Ordinary window glass is practically opaque and absorbs these rays, while quartz is transparent and permits them to pass through.

Aschenheim¹³ found that after an hour's exposure of the body to the direct action of sunlight there was a general leukocytosis in the peripheral blood with a relative increase in the lymphocytes and a decrease in polymorphonuclear leukocytes in 80 per cent of the cases. Lymphocytes are regarded as one of the body's defenses against tuberculosis, and Aschenheim suggests that this may account for the favorable effect of sunlight on tuberculosis.

The health blessings derived from radiant energy in the sun are obvious, but excessive exposure to sunlight, especially in those unaccustomed to it, may be harmful. The injury is believed to be due almost entirely to the ultra-violet portion of the sunlight. Exposure to sunlight causes burning, tanning or freckling. Tanning is beneficial, burning is injurious. Prolonged exposure to bright sunlight in those not accustomed to its rays may even be dangerous. The damage is apt to be more than the sunburn resulting. Headache and symptoms of severe meningitis may develop. In two of Romer's cases¹⁴ symptoms of serous meningitis (meningismus) developed. If the temperature and humidity are high, sunstroke (insolation) is one of the consequences. All bacteriologists know the germicidal power of sunlight on bacteria, which is due to the rays of short wave-length beyond the violet end of the spectrum. Sellards¹⁵ states that bilirubin was found to exert a pronounced effect upon the clotting of blood in vitro in the presence of sunlight. A small amount of bilirubin in oxalated plasma exposed to sunlight completely prevented clotting when serum was added. Controls kept in the dark clotted readily.

Bovie¹⁶ speculates that the red color of the blood protects our bodies from the actinic solar rays quite as efficiently as we protect our photographic plates by using a ruby light. The pigments of the skin also serve a protective purpose. Blonds are more susceptible to sunburn than brunettes, who are also believed to stand tropical climates better than the former.

The benefits of sunshine are obvious, although not fully understood.

Snow-Blindness.—The dazzling reflection of the rays from snow and ice fields is often a source of great annoyance and even danger to mountaineers and arctic explorers. The damage is due to the excess of ultraviolet rays rather than to the intensity and direction of the light. The excessive amount of ultraviolet rays burns the conjunctiva and even the cornea, and may cause serious inflammation. The same effects occur in electric arc welding which even affects the skin if exposed. Hence, helmets and gloves are worn. The best protection for snow-blindness is afforded by mussel-shaped, smoked tinted, or special glasses. See page 472.

¹³ *Ztschr. f. Kinderh.*, 1913, 9: 2.

¹⁴ *Deutsch. med. Wchnschr.*, July 8, 1915, 832.

¹⁵ *J. Med. Research*, 1918, 38: 3.

¹⁶ *Ibid.*

Effect of Light on Microörganisms.—Downes and Blunt¹⁷ showed for the first time that sunlight retards the growth of bacteria, and proved that this was not due to heat since the same result was obtained with tubes cooled in ice. In 1893, Marshall Ward¹⁸ used colored filters and demonstrated that the effect was due to the violet end of the spectrum. In 1905, Hertel¹⁹ showed conclusively that the shorter the wave length, the greater the lethal factor on bacteria and paramecia. This work seemed to show that sunlight contains few rays short enough to affect bacteria. It is now known that light less than 300 $\mu\mu$ is not met with under usual conditions, and produces in all living cells strong and even very harmful reactions. The effect is doubtless due to some photochemical reaction produced when light energy is absorbed. In large organisms, the effect is a surface one, owing to the small penetration of ultraviolet light. We do not know the exact nature of the photochemical reactions produced in protoplasm by ultraviolet light.

By means of its lethal action on bacteria and also on protozoa, the sun is of undoubted hygienic value to mankind, but it is probably beneficial in a less direct way as well, as there is growing evidence to show that light may have a direct action on higher animals.

Light and Nutrition.—Radiant energy is necessary to obtain the utilization of calcium and phosphorus supplied in the food. Without supplying either phosphorus or calcium, radiant energy or the potent component of cod-liver oil, causes the organism to operate normally in this respect. Both radiant energy and cod-liver oil supply something that makes metabolism more efficient; that is, they cause the organism to operate with increased economy. Steenbock and Daniels have shown that light can impart anti-rachitic properties to certain otherwise inert compounds. This transformation has been noted with meat, milk, cereals and various other common foods that ordinarily are not antirachitic in potency. Even cholesterol, the long known lipid component of animal cells, is readily activated in this way with ultraviolet rays. Steenbock and Daniels assert that it thus becomes immaterial whether active compounds are ingested or whether they are activated after they are absorbed. The end result is the same.

Light and Immunity.—Sunlight and light from other sources, especially containing rays of short wave-length, affect our resistance and susceptibility to a number of different conditions. This has already been noted in the case of rickets, tetany, tuberculosis, and various skin manifestations. The nature and degree of these processes has been little studied and are therefore poorly understood. In vitro, light has an injurious effect upon most toxins and antitoxins and other antibodies. From the evidence at hand it seems that light, depending upon its intensity and nature, affects immunity favorably and under other conditions unfavorably.

¹⁷ *Proc. Roy. Soc.*, 1877, 26: 488.

¹⁸ *Proc. Roy. Soc.*, 1893, 52: 393; 23: 54; 472.

¹⁹ *Ztschr. f. allg. Physiol.*, 1905, 5: 95.

Hektoen²⁰ found that exposure of dogs and rabbits to Roentgen ray at about the same time as antigen is introduced may restrain partially or completely the production of antibodies. When antibody production is at or near its height, exposure appears to have but little if any effect on the antibodies in the blood, and there appears to be at this time an increased resistance to the ray.

Photodynamic Action.—Familiar examples of photodynamic reactions are the chlorophyll system, retinal processes, the action of ultraviolet light, and photographic methods. The phenomenon is also considered under the term photochemical reaction, sometimes photosensitization.

Sunlight in the presence of certain fluorescent substances²¹ may become surprisingly active, approaching in its deleterious effects on living cells, the order of magnitude of the effects of x-rays and similar short radiations.

The most important of all photochemical reactions is that by means of which the chlorophyll of green plants stores up light energy, and in the process gives oxygen to the atmosphere in return for carbon dioxide absorbed. Through a process of photosynthesis it manufactures sugar and starch. Chlorophyll absorbs chiefly the red end of the spectrum.

The combined action of light and certain fluorescent substances may cause skin rashes and serious disorders terminating in death. Thus, buckwheat when fed to sheep and swine for the most part produces no untoward effect, especially in the dark days of winter, when the animals are kept under cover in stalls. But if the animals are white, or have white spots exposed to bright light, then serious symptoms frequently develop (fagopyrismus). The symptoms, if not too far advanced, subside when the animals are returned to darkened buildings, or even if the hair is artificially colored. Under the same conditions of light and food, animals that are dark in color remain well.

The phenomenon of photodynamic sensitization was discovered accidentally by Raab,²² working under the direction of von Tappeiner. He found that acridin is lethal to paramecia only in the light. With a strength of 1:20,000, paramecia are killed in six minutes in direct sunlight, one hour in diffuse daylight, and are unharmed in the dark. This led von Tappeiner, Jodlbauer and their coworkers²³ to study the subject extensively and they found many substances to act as sensitizers, fluorescein and its derivatives being especially potent. In vitro a surprising number of interesting results were obtained. Thus, it was found possible to sensitize to the action of visible light, bacteria, protozoa, red blood-corpuscles, enzymes, ferments, various immune bodies and certain well-defined chemical substances, such as the combination of mercuric chlorid and ammonium oxalate. Von Tappeiner laid great stress on the fact that most of the substances which act as sensitizers are fluorescent. Fluores-

²⁰ *J. Infect. Dis.*, 1918, 22: 28.

²¹ Phosphorescence and fluorescence represent photochemical reactions with storage of light energy. H. von Tappeiner und A. Jodlbauer; *Die sensibilisierende Wirkung fluoreszierender Substanzen*, Leipzig, 1907.

²² *Ztschr. f. Biol.*, 1900, 39: 524; 1903, 44: 16.

²³ *Ergebn. d. Physiol.*, 1909, 8: 726.

cence, in fact, is a more general property than is usually supposed. The sensitized reactions are specific. Although eosin seems to be a sensitizer for all cells and chemical substances, this is not invariably true of other dyes. Although a great many substances sensitize in vitro, only eosin, chlorophyll and certain derivatives of hemoglobin have so far been found effective in vivo, and the only markedly effective sensitizer for higher animals is hematoporphyrin.

Hausmann²⁴ injected white mice with hematoporphyrin. Those animals which were kept in the dark developed no symptoms, while those exposed to direct sunlight developed lesions resembling sunburn at the most exposed parts, such as the tips of the ears, nose and tail. Death subsequently resulted. Exposure to bright diffused light produced marked symptoms.

Meyer-Betz, with more devotion to science than to his own welfare, injected 200 milligrams of hematoporphyrin into his own blood. Subsequent exposure to light produced most distressing symptoms similar to those seen in the mouse, and he remained light-sensitive for a long time. In hydroa vacciniformis, a light-sensitive condition in children which recurs each summer, hematoporphyrin is found in the urine, and the symptoms bear some resemblance to Meyer-Betz's reactions.

The eruptions on the exposed surfaces in pellagra and other diseases have been explained upon the principles of photosensitization. Duke²⁵ found that urticaria may be caused by light.

The action of light in the presence of fluorescing substances upon red blood-corpuscles and immune bodies has been studied by Sellards, Bovie and Brooks.²⁶ The agglutinating action of ricin, the hemolytic action of croton, the poisonous action of diphtheria and tetanus toxins, and the protective effects of tetanus antitoxin are injured or destroyed by photodynamic action. Many enzymes are susceptible to light alone, but this effect is usually increased by the presence of a fluorescent body.

HELIO THERAPY

The pioneer work of Finsen in the treatment of lupus vulgaris emphasized the importance of considering a diversity of forms of radiant energy in skin affections. The treatment of lupus vulgaris gives strikingly good results. Urticaria and acne are sometimes favorably influenced by light, and birth-marks may be improved, but the same results can be obtained with x-rays. In tuberculosis, especially surgical tuberculosis, heliotherapy also produces good results. Most remarkable are the results in bone and joint tuberculosis. Light of short wave-length, which is known to have marked bactericidal effects, may not be without salutary influence in the treatment of wounds.

²⁴ *Wein. klin. Wochenschrft.*, 1910, 23: 963, 1820.

²⁵ *J. Am. M. Ass.*, 1923, 8: 1835; 1924, 83: 3.

²⁶ This article also contains a good discussion and bibliography of the subject. *J. Med. Research*, 1918, 38: 3.

Hess and Unger and also Howland and his associates showed that the calcium concentration of the serum of children with rickets can be raised to the normal level by exposing the children to rays from the mercury vapor quartz lamp (see page 635). Infantile tetany is favorably influenced by light. In 1920, Huldshinsky²⁷ treated six children suffering with tetany by means of ultraviolet rays. The symptoms disappeared in from four days to four weeks. It is known that in cases of active infantile tetany, the calcium concentration of the serum is uniformly low. The good results in tetany following exposure to the rays are associated with calcium concentration of the serum, increased approximately to normal. Similar results can be obtained in tetany by the administration of large doses of calcium chlorid, but this does not increase the phosphorus in the serum, whereas exposure to light increases both the calcium and the phosphorus. This may help explain the way in which light favorably influences the healing of tuberculous lesions, and acts as a preventive in this infection.

Heliotherapy appears to be equally effective when the exposure is to direct sunlight or to alpine lamps and other sources of light which contain the necessary rays of short wave-length. The object is to produce a tan without burning. Rollier insists that the beneficial effects seem to be directly proportional to the degree of tanning. The entire surface of the body is used and the exposure gradually increased within the tolerance of the individual.

RADIO-ACTIVITY

Radio-activity is a property resulting from radiations of exceedingly high frequency (short wave-lengths) or x-rays and are assumed to be produced by a discharge of electrons.

Soon after the discovery of radium by the Curies it was proved, chiefly through the investigations of Elster and Geitel, that the air and soil and certain mineral springs contained radio-active substances. Newly fallen rain and snow are also radio-active. Air drawn from the soil by means of a pipe, or air shut up in underground cellars and caverns, is especially radio-active, as is also the air on mountain tops. The air in clear weather has greater radio-activity than in dull weather.

Certainly radio-active substances have important physiological, physical, and chemical effects. They ionize the air, rendering it a conductor of electricity; they cause a fluorescence of certain chemical substances; they produce a sensation of light if they strike the eye; applied to the body in sufficient "dosage," radium, α , β , γ (x-rays) cause destruction of tissue, changes in the blood, and general constitutional symptoms. Local destruction of tissue may be reflective, even specific, as in certain cancers and for lymphoid structures, as in Hodgkin's disease.

²⁷ *Ztschr. f. Kinderh.*, 1920, 26: 207.

SMOKE

Smoke is a product of combustion and consists of a mixture of gases containing solid particles.²⁸ Ordinary smoke consists largely of unburned carbon particles, hydrocarbons, and other pyroligneous products; gases, some of them poisonous, such as carbon monoxid; also mineral acids, etc. Angus Smith gives the following analysis of smoke from a common house fire:

	Carbon Dioxid	Carbon Monoxid	Oxygen	Nitrogen
Gas from chimney 4 feet above the fireplace {	0.35 1.65 0.38	16.93 19.29	80.02 78.68
Gas from the middle of a good fire. A great mass of coal over the fire, the gas taken from below the glowing mass..... {	19.46 20.90 17.50 17.44	0.09 0.10 0.60 0.39	80.45 79.00 80.04 82.17
A heap of glowing coal, gas taken close to spot where carbonic oxid was burning... {	15.43 18.17	3.49 2.48	0.96	80.12 79.35
Gas from clear fire below..... {	16.10	4.95	78.95
Gas from the same fire at upper part, 1 inch below the surface..... {	17.21 18.20 0.99	4.25	78.54 78.21

Cohen of the Manchester Air Analysis Committee gives the following analysis of soot collected from the roofs of glass houses in Kew and Chelsea:

Constituents	Chelsea, per Cent	Kew, per Cent
Carbon	39.0	42.5
Hydrocarbons	12.3	4.8
Organic bases (pyridins, etc.)	2.0
Sulphuric acid.....	4.3	4.0
Hydrochloric acid.....	1.4	0.8
Ammonia	1.4	1.1
Metallic iron and magnetic oxid of iron.....	2.6
Mineral matter (chiefly silica and ferric oxid)	31.2	41.5
Water not determined (say difference)	5.8	5.3

Large manufacturing chimneys are the chief offenders. There are two main causes of smoky chimneys: (1) insufficient boiler capacity, and (2) improper stoking. The cure of the smoke nuisance consists in the installation of boilers of sufficient power so that they need not be forced, and the use of mechanical stokers. The electrification of railroads and the more general use of electric power generated from water pressure help materially to lessen the amount of smoke in cities.

²⁸ Smoke consists largely of solid particles suspended in the air; fog, of liquid particles.

The London County Council permits black smoke for five minutes after the lighting of furnaces. Other towns allow as much as fifteen minutes. Most laws distinguish between black smoke and white smoke, although the one is about as pernicious as the other.

In Boston the density of the smoke is graded into four classes, in accordance with Ringelmann's chart. This is a rather complicated system, depending upon the character of the stack, the density of the smoke, and the time, as shown in Fig. 72.²⁹

The amount of smoke in some manufacturing centers is almost incredible. W. N. Shaw estimates that London gives to the atmosphere every day about 7,000,000 tons of smoky air containing over four hundred tons of soot, and he calculates that smoke deprives London of about one-sixth its possible sunlight and daylight in summer and about one-half its possible sunlight and daylight in winter.

Besson³⁰ finds that at ten kilometers from Paris, with the wind blowing from or toward the city, there is a diminution of 15 to 25 per cent in illumination due to this cause.

The injurious effect of smoke on health has perhaps been overestimated. It acts directly and indirectly. Directly it irritates the mucous membranes of the upper respiratory passages, and Asher and also Rubner believe that it increases the mortality from acute pulmonary diseases. They state that smoke and soot predispose to acute pulmonary tuberculosis. Indirectly smoke is a source of dirt and general nuisance and leads to depression of the spirits. It shuts out the light, soils with soot, and deters the opening of windows in order to let in fresh air. The presence of mineral acids in the air has a corrosive influence upon inorganic substances, and doubtless acts injuriously upon plant and animal life. The economic losses from the soiling action of soot are enormous. Even if it were not injurious to health, smoke is so evident a nuisance that communities are justified in every effort to check and prevent this growing abomination.

Klotz³¹ characterizes pulmonary anthracosis as a community disease. He found as much as 1.2 to 5.3 grams of carbon in the lungs of persons living in Pittsburgh, while only 0.14 and 0.4 gram in the lungs of two residents in Ann Arbor, Michigan. Anthracosis, then, affects city dwellers in proportion to the amount of smoke in the air. The amount of carbon in the lungs is dependent upon the amount inhaled. Carbon in the lungs causes a loss of elasticity of the tissue; structural changes, especially fibrosis, about the anthracotic deposits; the air spaces are encroached upon, resulting in compensatory emphysema. When anthracosis is well marked it seriously impairs the function of the lungs. Pleural adhesions do not develop as a result of the deposit (page 1265).

²⁹ For construction of Ringelmann's smoke chart and methods of taking smoke readings, *Smoke Investigation*, Bull. No. 8, p. 191, Mellon Institute.

³⁰ *Compt. rend. Acad. d. sc.*, 1923, 176: 180.

³¹ *Am. J. Pub. Health*, 1914, 4: 887.

TABLE SHOWING THE DENSITY OF SMOKE, IN ACCORDANCE WITH THE RINGELMANN CHART, WHICH MAY BE EMITTED FROM THE VARIOUS CLASSES OF STACKS IN BOSTON, MASS., AND THE DURATION OF SUCH EMISSION.

YEAR	CLASS 1		CLASS 2		CLASS 3		CLASS 4		CLASS 5		CLASS 6		LOCOMOTIVES MOVING TRAINS OF SIX CARS OR MORE	
	Chart Number	Minutes	Chart Number	Minutes	Chart Number	Minutes	Chart Number	Minutes	Chart Number	Minutes	Chart Number	Seconds in 5-Minute Periods	Chart Number	Seconds in 5-Minute Periods
1910	3	6	4	5	4	10	4	9	4	12	3	40	3	50
1911	3	4	3	10	3	20 including 5	3	12	3	15	3	30	3	40
1912	2	8	3	6	2 including 3	30 including 10	3	7	3	9	3	20	3	30
1913	2	6	3	3	2 including 3	25 including 5	3	3	3	5	3	20	3	30

Reduced Copy of Ringelmann Chart

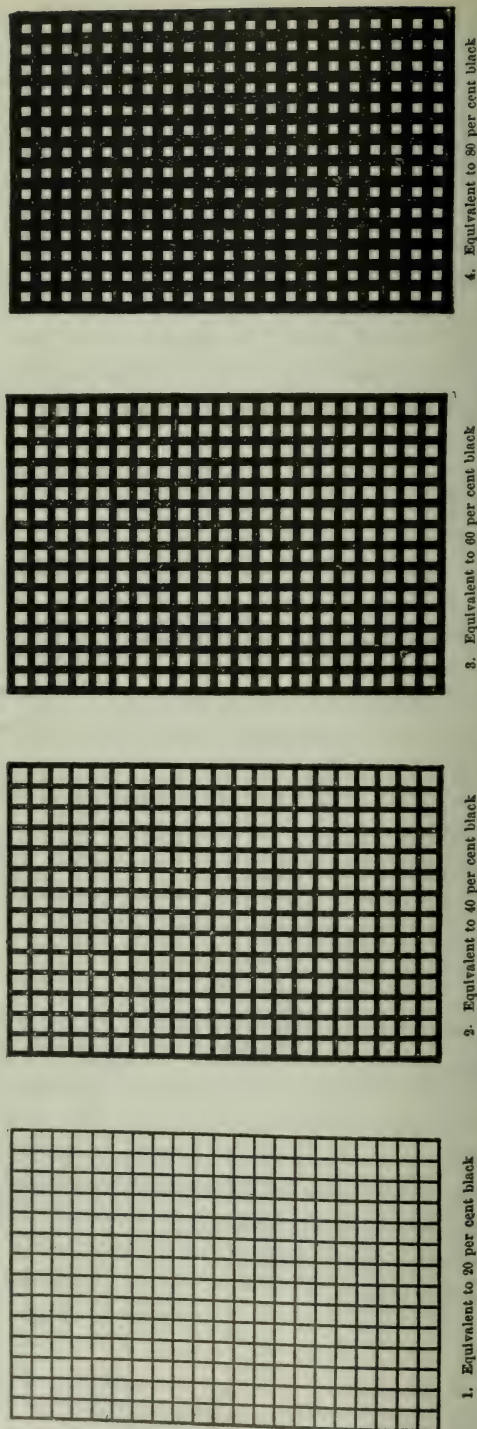


FIG. 72.—INSTRUCTIONS FOR USING THE RINGELMANN CHART.

Hang smoke chart on a level with the eye, about 50 feet from observer, as nearly as possible in line with chimney. Glance from smoke to chart and note corresponding number, recording same and time of observation. Repeat observations at one-fourth or one-half minute intervals. From these records the average density may be determined for each hour or for each day. No smoke is recorded as No. 0. 100 per cent black smoke is recorded as No. 5. Experienced observers often record in half chart numbers.

Smoke polluted with poisonous chemical vapors may be quite serious. Thus, hydrogen sulphid, found in large quantities in the smoke generated in sulphate of ammonia and tar works and from alkali wastes, is a poisonous gas. The arsenical vapors given off chiefly from lead and copper smelters kill vegetation for wide areas around.

FOG

Fogs are caused by the condensation of water vapor on particles of dust and ocean fog can form on NaCl crystals. Dust particles have a varying capacity for condensing and attracting moisture, depending upon their power of radiating heat and on their affinity for water. Carbon dust is non-hygroscopic but none the less promotes fog formation; in fact, any small particles will do this. The ammonia and sulphuric acid in smoky air also occasion and aggravate fog. The air of manufacturing cities, therefore, possesses all the elements to form a fine persistent fog which forms a "chemical pall" between the city and the sky.

The more carbon a fog contains the blacker it is. The general result of a fog is to shut out sunlight and fresh air and to "partially suffocate unfortunate citizens in clouds of noxious chemicals." It filters out much of the ultra-violet light and promotes rickets. Fog contains all the irritating properties of smoke in a concentrated form, and it also in a measure prevents the escape of the city-made carbon dioxid. The carbon dioxid in the city air during a fog may rise to ten parts per ten thousand. If smoke is bad, fog is ten times worse. It has been shown that during city fogs sickness increases and the death rate rises. From the economic standpoint fog causes greater financial losses than smoke. Russell calculates the annual loss to the people of London from fog to total about \$9,000,000 a year. The main items in this loss consist in extra washing, including extra soap, the damage to dresses, curtains, carpets, and textile fabrics, the replacing of wall papers, and the painting of houses, the restoring of gilt and metal work, the slow destruction of granite, marble, and stonework of buildings, the extra cost of artificial illumination, etc. This estimate does not include the losses resulting from its action on health.

For further studies on fog and smoke the student is referred to the admirable series of annual reports of the British Atmospheric Pollution Committee, H. M. Stationery Office, London.

DUST

Dust is not only a nuisance, but under certain conditions is known to be prejudicial to health. Dust is in reality a normal and very important constituent of the air; it exists everywhere in the atmosphere and profoundly affects some of the physical conditions of our environment. One of the most important functions of dust is to limit the humidity of the air by causing the precipitation of moisture in the form of rain, and to help control temperature by the formation of clouds, mists, and fogs. Without dust there would be no

rain, no clouds, no mist, for the water vapor which condenses upon each particle of dust forms the nucleus of a raindrop.

Dust disperses the light and decreases the transparency of the atmosphere, especially if the atmosphere be also humid. What is known as haze is really dust carrying a minute amount of moisture.

Although dust particles are universally present in the known atmosphere, they are very irregularly distributed. Organic dust exists only in the lower strata, while inorganic particles are found wherever the air has been examined. Ordinarily there is more dust indoors than in outdoor air. The size of the dust particles varies enormously, from gross masses to microscopic and ultra-microscopic particles. The vast numbers and universal presence of these particles may be realized by examining a sunbeam—the Tyndall effect. Air free of dust is an artificial product obtained only with special care in small amounts in the laboratory.

Both the size and the specific gravity of the particles are of great importance. Thus, lead particles 0.2 to 2 μ in diameter may float about almost as readily as silica which is six to seven times lighter. Fine particles settle out, in still air, directly as the specific gravity and as the square of the diameter (Stokes' law). Of the two the size is therefore the more important. Drinker and Thomson³² give the following classification: (1) *dusts* made up of particles ranging from 150 to 1 micron; (2) *fumes*, particles from 0.2 to 1 micron; (3) *smokes*, particles from 0.3 micron and less.

Most of the dust is torn from the earth by the winds; much of it comes from the carbon and other particles in smoke; considerable amounts consist of minute grains of salt derived from sea spray; and great quantities are added by volcanoes. Finally, the air contains interplanetary particles which fall through it in a constant shower.

The spectrum shows the bands of sodium everywhere in the atmosphere. This is lifted into the air by the wind from the sea spray. The water evaporates, leaving the salt particles to float about at the will of the wind.

Organic dust consists of the dry and disintegrated particles which are blown into the air from the animal and plant kingdoms. They consist of epithelial scales, seed, spores, bacteria, pollen, plant cells, fluff of various kinds, bits of insects, starch, pus cells, algæ, rotifers, fragments of hair, feathers, and bits of tissue, fibers of cotton, etc.

The inorganic dust, which is derived mostly from the soil, from the sea, and from interplanetary space, consists chiefly of silica, aluminium silicate, calcium carbonate, calcium phosphate, magnesia, iron oxid, sodium chlorid, etc.

Modern cities are dust producers. Whipple found the number of dust particles visible with a magnification of one hundred diameters, at the air inlets of some of the commercial buildings in Boston, as determined by microscopical counts, to range from 100,000 to nearly 1,000,000 per cubic foot.

The number of particles vary from one per cubic centimeter on mountain

³² *J. Indust. Hyg.*, 1925, 7: 261.

tops on exceptionally clear days to millions per cubic centimeter in ordinary indoor air. Considering only particles of hygienic importance, namely, those having a diameter of 1 micron or over, recent analyses show 900 particles per liter, or 0.11 gram per million liters of country air on a clear day. At the other extreme, 760 grams per liter or 24,000,000 particles per liter, have been found in the air of an abrasive factory.

The number of dust particles in a room is inversely proportioned to the amount of fresh air supplied. Ordinary air washers remove from 20 per cent to 70 per cent of the dust.

Dust particles may be carried enormous distances by the winds. Ehrenberg detected organisms belonging to Africa in the air of Berlin; and fragments of infusoria belonging to the plains of America in the air of Portugal. The smoke of the burning of Chicago reached to the Pacific coast. The volcanic dust of Krakatoa, consisting chiefly of glassy pumice, was found for years in our atmosphere, and it is assumed that some of it may have traveled several times around the world. Macfie has seen in the Canary Islands clouds of dust sufficient to obscure the sun, though the dust had come all the way from the African mainland. All of us living on the Atlantic seaboard have seen the yellow days caused by forest fires several thousands of miles away.

Dust and Disease.—"Normal" atmospheric dust, free from bacteria, causes no appreciable irritation to the healthy respiratory mucous membranes. Dust becomes injurious when excessive in amount or when irritating in character, or when it contains injurious microorganisms; the injury also depends upon the constancy of its presence and somewhat upon the susceptibility of the individual.

Dust may act indirectly as a predisposing cause of many infections, as well as directly irritating and inflaming the respiratory passages. The statement is often made that dust opens the door to tuberculosis and other infections of the air passages, such as common colds, influenza, pneumonia, etc.

The effect of dust is fully discussed in *Dusty Trades*, in the chapter on Industrial Hygiene, and is therefore not repeated here. The question of bacteria in the air is discussed in the next section.

In general, there is a relation between dust and the number of bacteria in the air.

Winslow and Kligler³³ report an average of 49,200,000 microbes per gram in New York street dust, and between 3,000,000 and 5,000,000 per gram in indoor dust. Street dust includes the colon bacilli and acid-forming streptococci.

House dust is more harmful than outside dust, not only because there is more of it, especially in badly ventilated and ill-kept rooms, but because it is more apt to contain living pathogenic bacteria. House dust may be kept down by cleanliness and avoidance of dry dusting and sweeping; by the use of vacuum cleaning; and by free ventilation. Much house dust is blown in

³³ *Am. J. Pub. Health*, 1912, 2: 663.

from the outside, and some of it comes in on dirty shoes. In buildings ventilated with a mechanical system the air may be filtered or passed through a water curtain, which will eliminate much dust. Oiling floors with a wax or paraffin mixture helps to keep down indoor dust. Carpets tacked down are sanitary abominations and should be replaced with rugs that permit outdoor cleaning and sunning.

Street dust contains coal dust, metallic dust from the operation of trolley cars, material swept from houses and from shaking rugs from windows, the grinding up of roadbeds by vehicles, ashes, and other materials blown from barrels and teams; the bacteria are derived from dried fecal matter from horses and other animals, dried sputum, the soil, and a variety of other sources. Street dust may contain pathogenic organisms, such as the tubercle bacillus, many varieties of cocci, the colon bacillus, *Bacillus aërogenes capsulatus*, and possibly, under special conditions, tetanus, malignant edema, and occasionally other pathogenic microorganisms. Street dust, therefore, becomes more than a nuisance, for it is not only irritating, but may be a source of infection.

To keep down street dust requires, first of all, a well-constructed road with a good surface, oiled or properly cared for; the control of animals; the covering of ash barrels and carts hauling dusty loads; the use of automobile vacuum cleaners to replace the old or the present-day methods of dry sweeping. Attention must also be given to spitting on sidewalks and streets, the enforcement of smoke ordinances, the more extensive flushing of streets, and general attention to cleanliness.

When dust is violently stirred up by dry sweeping or beating carpets, or still more by a March wind in a dry, dirty street, the quantity inhaled with attached microorganisms has a real sanitary significance.

The pollen of certain plants flying in the air as dust leads to hay fever in susceptible individuals.³⁴

Methods for Examining Dust.—The methods for examining dust in the air may be grouped as follows:

1. *Settling.*—Settling depends on the falling of particles on to a known area in a given period. Haphazard devices give only crude results. Owens' methods³⁵ of recording are useful in meteorological determination; they also have hygienic significance in cities like London, Leeds, Manchester, Pittsburgh, etc.

2. *Filtration.*—Filtration depends upon drawing a given volume of air through a filtering substance, and weighing before and after. Filtering sub-

³⁴ For data in regard to the influence of dust upon disease, see B. A. Gohoe, *The Relation of Atmospheric Smoke and Health*; Bull. No. 9, Smoke Investigation, Mellon Inst. of Indus. Research and Sch. of Spec. Ind., Pittsburgh, 1914, p. 7; S. R. Haythorn, "Some Histological Evidences of the Disease Importance of Pulmonary Anthracosis," *J. Med. Research*, 1913, 29: 259; F. L. Hoffman, *The Mortality from Consumption in Dusty Trades*, Bull. No. 79, U. S. Bureau of Labor, Nov., 1909, p. 633; O. Klotz, "Pulmonary Anthracosis, a Community Disease," *Am. J. Pub. Health*, 1914, 4: 887; W. C. White and P. Shuey, "The Influence of Smoke on Acute and Chronic Lung Infections," *Tr. Am. Climatol. Ass.*, 1913; also Bull. No. 9, Smoke Investigation, Mellon Inst. of Indus. Research and Sch. of Spec. Ind., Pittsburgh, 1913, p. 164.

³⁵ *Brit. Atmosph. Pollution Rep.*, 1921-25.

stances commonly used are cotton, sugar, extraction thimbles, resorcinol, fine mesh wire, cloth, filter paper, collodion, wool, cheesecloth, canton flannel, etc. Filtration methods are convenient but uncertain.

3. *Water Spray*.—Air may be bubbled through water in a device such as the Palmer apparatus, which is compact, easily transported, and samples a large volume of air in a short time. It is best applied to dusts of high concentration, such as 0.5 gram per cubic meter, or 50 particles per cubic centimeter. The disadvantages are that it requires electric power to pump the air and is not suitable for soluble dusts or those attacked chemically by water. The efficiency of water as a dust-catcher is low.

4. *Impaction or Impinging*.—The air is forcibly blown against surfaces specially prepared with sticky substances. Usually glass slides or cover slips prepared with vaselin are used. The impinging method is a reliable method for smokes and fumes. Owens' "jet dust counter" ³⁶ and also Greenberg and Smith's impinger ³⁷ are accurate, giving results about equal to electrostatic precipitation.

5. *Electrostatic Precipitation*.—The electrostatic precipitation of dust particles depends upon the ionization of finely divided substances in an electric field. This method is based upon the laws of electrically charged bodies, and is applied commercially to the recovery of valuable dusts. Its use for the sanitary analysis of air was studied in my laboratory by Bill ³⁸ and also by Drinker, Thomson and Fitchet.³⁹ Electrostatic precipitation is applicable to a great variety of dusts, and all sizes of dusts, fumes and smoke. It is accurate and sensitive.

6. *Condensation*.—Condensation methods depend upon the condensation of water vapor about dust particles and even on ions in a rarefied atmosphere.

The koniscope, invented by John Aitken, consists of two brass tubes connected at right angles and suitably fitted with stopcocks and a small air pump. By exhausting the air from one of the tubes, allowing the space to become saturated with water vapor by evaporation from wet blotting paper within, and then allowing this moisture to condense upon the dusty atmosphere under examination, clouds of different degrees of density will form inside the tube. The approximate density of the clouds can be measured by looking through the tubes; windows being provided for this purpose. A table is supplied with the instrument to give the approximate number of dust particles corresponding to clouds of different degrees of density. This method has limited usefulness, because it makes apparent exceedingly minute particles that have little sanitary significance.

7. *Photometric Methods*.—Photometric methods enable rapidity in reading, and give reliable indices of the size and dispersion of the particles.

³⁶ *J. Indust. Hyg.*, 1922-23, 4: 522; also Drinker and Thomson, *J. Am. Soc. Heat. & Ventl. Engin.*, Nov. 1924.

³⁷ *Pub. Health Bull.*, No. 144, 1925.

³⁸ *J. Indust. Hyg.*, 1919, 1: 323.

³⁹ *Ib. d.*, 1923-24, 5: 162.

Drinker and Thomson ⁴⁰ made a careful study of these methods and found that they are applicable to true colloids like acacia in water, or tobacco smoke in air, as well as to coarse unstable suspensions, like silica dust in water or in air. The principle depends upon a comparison of the standard illumination with that of the unknown. This is done by matching the brightness of the light through the telescope of the instrument and reading the foot candles. Thomson ⁴¹ has devised a method for sampling dust in the alveolar air by the use of dust counting instruments, in conjunction with Haldane's alveolar air sampler.

Many of the methods for examining dust in the air are faulty in one or more respects; excepting electrostatic precipitation and Owen's jet dust counter, all the practical methods lack extreme sensitivity. A comparison of methods, with advantages and disadvantages, is given in *Public Health Bulletin* 144, 1925; also *United States Public Health Reports*, 1925, 40: 765.

⁴⁰ *J. Indust. Hyg.*, 1925, 7: 567.

⁴¹ *Ibid.*, 385.

CHAPTER IV

BACTERIA AND POISONOUS GASES IN THE AIR

BACTERIA IN THE AIR

The number of bacteria in the air ordinarily has a direct relation to the amount of dust; in fact, many of the bacteria in the air are attached to dust particles. Bacteria in the air are commonly considered as one kind of dust, but on account of their significance they are given separate consideration.

Bacteria are not found everywhere in the air; uninhabited places are quite free; and the number diminishes as we ascend.

Bacteria do not multiply in the air; in fact, most of them soon die, especially when exposed in dry air to sunshine. For the most part, the bacteria in the air belong to the harmless varieties, although the number and kind vary greatly with circumstances. They come chiefly from the soil and are carried into the air by the wind and traffic movements; that is, bacteria in the air are derived from practically the same sources as dust. The dangerous bacteria in the air, however, come directly or indirectly from man and some of the lower animals.

The number of bacteria differs greatly with the local conditions. There are more in the air of towns than in the open country; few in high mountains, desert places, or at sea; more in windy weather than calm air; more indoors than in outside air; more in dry air than in moist air; more before than after rain. The air of badly ventilated rooms, especially if not kept clean, contains very many bacteria, and more when occupied, as the movements of the occupants stir up the dust.

Miquel of the Observatory of Montsouris studied the number of bacteria in the air of various localities. He found about 150 per cubic foot in the air of Paris, but only six after rain; on the top of the Pantheon he found $1\frac{1}{2}$; in the streets about twelve per cubic foot; in a neglected hospital 3,170; in a gram of laboratory dust 75,000 and in a gram of house dust 2,100,000. Flügge considers that on the average there are about one hundred microorganisms to a cubic meter of city air—an average evidently below that of Paris. Jean Binot did not find a single bacterium in one hundred liters of outside air taken at the summit of Mont Blanc; and he found a progressive decrease in the number as the height increased. Thus, he found the following number of bacteria per liter:

At Montanvert	49
At the Mer de Glace	23

At the Place de l'Aiguille	14
At the Grand Malet	8
At the Grand Plateau	6
On the summit	0

Again, Graham-Smith found at the top of the Clock Tower of the Houses of Parliament in London only one-third of the number at ground level. Whipple found 1,330 bacteria per cubic feet in the air at the street level, while at the tenth story of the John Hancock building in Boston the air contained 330. Speaking broadly, from two to three hundred times as many particles of dust as bacteria are found in the outside air of cities. Haldane found 256 bacteria per cubic foot of air in an unventilated room compared to practically none in a ventilated room.

Pasteur, in experiments that will ever remain classic, exposed organic infusions in flasks to the air of various places, and used the results thus obtained to prove the presence or absence of bacteria in the air and to dispel the illusion of spontaneous generation. Of twenty such flasks exposed to the air of the Mer de Glace nineteen showed no contamination. About the same time (1875) Tyndall exposed twenty-seven flasks containing an infusion to the air of the Aletsch glacier (8,000 feet); none showed putrefaction, while 90 per cent of the flasks opened in a hayloft were "smitten."

It is estimated that a person living in London breathes about 300,000 microbes in the inspired air each day. Winslow and Brown¹ examined 1,037 samples of air, both indoors and outdoors, with counts varying from two to 5,200 per cubic foot.

The expired air, during normal respirations, is practically bacteria-free, no matter how many may be contained in the inspired air. The moist mucous membranes of the upper respiratory passages act as a bacterial trap. When the expired air contains bacteria it is only as a result of coughing, sneezing, talking, or other forced expiratory efforts (see Droplet Infection).

Stillman² states that when mice are exposed to an atmosphere containing certain bacteria in the form of a fine mist, the bacteria may be recovered from the deeper respiratory passages. Pneumococci which have reached the lungs of normal mice as a result of this procedure usually disappear within a few hours. Hemolytic streptococci and *Bacillus influenzae* generally persist in the lungs for about twenty-four hours.

The number of bacteria in the air has no special sanitary significance, but their kind may be important. Ordinarily, there is little if any danger from air bacteria except under special circumstances, such as droplet infection and close personal contact, or dairy conditions. The harmful bacteria in the air and the danger of contracting disease through air-borne infection are considered below.

¹ *Month. Weather Rev.*, 1914, 42: 452.

² *J. Exper. M.*, 1924, 40: 353.

Method for Determining Bacteria in the Air.—A rough idea of the bacterial population of the air may be obtained by exposing suitable culture media in Petri plates for various periods of time, and counting the colonies which develop from the germs falling upon them.

A large number of different devices have been described for a more accurate determination of the number of bacteria in the air. These are all adaptations of the three general methods: (1) filtration of air; (2) bubbling air through some liquid medium; (3) precipitating the bacteria from a given volume of air. Each of these methods can be made to give fairly satisfactory results in the hands of competent workers, but the Committee of the American Public Health Association recommends the following method of Petri on account of its simplicity and general applicability:³

Filtration Method of Petri.—The filter tubes are glass tubes $1\frac{1}{2}$ centimeters in diameter and 10 centimeters long. In the end of each is placed a perforated cork stopper, through which a glass tube 6 millimeters in diameter is passed. The filtering material consists of sand which has been passed through a 100-mesh sieve. The sand in the filter tube is 1 centimeter deep and supported by a layer of bolting cloth covering the cork. Two filter tubes are connected in tandem, and a measured volume of air, ten liters or more, is drawn through at a constant rate by suction. The suction is applied by means of an aspirator of known volume, preferably one of the double or continuous type. Either the Magnus aspirator or the double aspirator are suitable for this purpose. Before using a pair of filter tubes a test for possible leakage is made by placing the thumb over the cotton stopper and applying the aspirator; if the suction is weak or absent the corks must be tightened or the tubes discarded. All corks should be tightened and connections wired and the apparatus sterilized before using the filters. The collection of the sample should take from one to two minutes per liter.

After filtering a definite volume through the tubes the sand is poured into ten cubic centimeters of sterile water, thoroughly shaken, and aliquot portions plated in ordinary nutrient agar, all plates being made in duplicate. The plates are incubated at room temperature for five days, when final counts are made.

Rettger's Method.—An improved method of enumerating air bacteria has been described by Rettger,⁴ which commends itself as the best method yet devised. The method consists of bubbling a given quantity of air through salt solution. The bacteria in the air are trapped in the salt solution, and may then be planted in the usual way and the number of colonies counted.

Air and Infection.—The air was long regarded as the vehicle and even the source of the communicable diseases. Theories, such as noxious effluvia, poisonous emanations, and infectious miasms, gave way with the advent of bacteriology. When the early classical researches of Pasteur, Tyndall, and

³ See also "Relative Methods of Enumerating Bacteria in Air," *U. S. Pub. Health Rep.*, 1925, 40: 2167.

⁴ *J. Med. Research*, 1910, 22: 461.

others showed that bacteria exist in the air almost everywhere in inhabited places the conclusion was jumped at that the air must be particularly dangerous. Within recent years, however, we have learned that the air is not very much to be feared on account of the bacteria it may carry, except under occasional circumstances. This change in our views during recent times is nowhere better illustrated than in the relation of the air to surgery. During the early days of antiseptic surgery so much fear was entertained for the bacteria in the air that Lister attempted to neutralize the danger with carbolic sprays and other means; now the surgeon pays little heed to the air of a well-kept operating room. Instead he ties several layers of sterile gauze over his mouth and nostrils and over his head to guard against particles falling from these sources.

It was one of the great surprises when bacteriologists demonstrated that the expired breath under normal conditions of respiration is practically sterile.

At one time many, if not most, of the contagious diseases were believed to be air-borne; many observations are on record purporting to prove that contagium may be carried long distances through the air. With the increase of our knowledge concerning the modes of transmission of infection the list of air-borne diseases has steadily dwindled. The theory is reluctantly given up, for it is the easiest method of explaining the spread of the readily communicable diseases. There are only two diseases of man, viz., smallpox and measles, which may possibly be air-borne, in the sense that this term is generally used. Both these diseases are so readily communicable that the virus seems to be "volatile"; it is assumed that the active principle is contained in the expired breath; however, there is no proof of this assumption, and some evidence to the contrary. Further, it is noteworthy that we are still ignorant of the causes and the precise mode of entrance of the contagium in both measles and smallpox. Even in these two diseases the radius of danger is much more limited than was once supposed to be the case.

The more the transmission of the communicable diseases is studied the less the air is implicated. The fact that malaria (bad air), yellow fever, and other diseases are conveyed by mosquitoes has robbed the air itself of false accusations, and given a death blow to miasms, effluvia, and intangible theories. Pettenkofer insisted that the air became contaminated with poisons that were generated in a polluted soil, and he believed that these emanations were responsible in part for typhoid fever and cholera. Some association between soil, air, and disease still persists in both medical and lay minds, but with a more precise knowledge of the causes and modes of transmission of infections, such as typhoid fever and cholera, the air becomes a negligible factor. Out-of-door air contains relatively few bacteria; further, the dilution is enormous. Most microorganisms pathogenic for man soon die when dried or when exposed to sunlight. Whatever danger, then, resides in the air, so far as living principles of disease are concerned, is found rather in indoor air, and especially in the air of badly ventilated, dusty, and crowded places. Here the danger may be either from the bacteria-laden dust or from droplet infection.

In a crowded and stuffy street car, in a poorly ventilated office, or in a closed, close sick room it would be very easy for the microorganisms of diphtheria, scarlet fever, whooping-cough, measles, pneumonia, influenza, common colds, tuberculosis, pneumonic form of plague, and other infections contained in the secretions from the nose and mouth to be carried in droplets so that exposed persons may contract the disease. This probably occurs more frequently than we are at present inclined to admit.

The radius of danger through droplet infection is quite limited. It is difficult to conceive that infection may be carried long distances in the air and still be dangerous. My own experience indicates that there is practically no hazard in establishing a hospital for contagious diseases upon the high road or even in a thickly inhabited part of the city. In fact, the communicable diseases are not conveyed in the air from ward to ward or even from bed to bed in well-managed hospitals.

Hutchinson found that prodigious bacilli in sputum droplets may be carried almost 2,000 feet when the temperature is low, but this is exceptional. It therefore seems probable that diphtheria bacilli would persist longer and carry farther in droplet infection in cold weather than in warm weather. This hypothesis has been given to account for the seasonal prevalence of plague, diphtheria, etc., but without substantiation. The two chief ways in which bacteria are transferred through the air are (1) by droplet infection and (2) by dust. Mouth spray is really a form of contact infection.

Chapin states that many contagious hospitals have been maintained for years with no increase of the disease in the vicinity, as, for instance, at Boston and Providence, Rhode Island. At the Kingston Avenue Hospital in Brooklyn various diseases, as smallpox, measles, scarlet fever, and diphtheria, are treated in wards only a few feet apart, with no evidence of aerial transference. At North Brother's Island the tuberculosis ward is only about twenty-five feet from the diphtheria ward, but the tuberculous patients do not contract diphtheria. A number of hospitals for communicable diseases have recently been built with entire disregard of aerial infection. At the hospital of the Pasteur Institute, Paris, the patients are cared for each in a separate ward opening into a common hall. The same nurses go from case to case. In two and one-half years after it was opened in 1900 there were treated 2,000 persons, of whom 524 had smallpox, 443 diphtheria, 126 measles, 163 erysipelas, 92 scarlet fever, and 166 non-diphtheritic sore throat. The only evidence of the transfer of infection was the development of four cases of smallpox and two of erysipelas. In the Hôpital des Enfants Malades in Paris the beds, instead of being in separate rooms, are separated by partitions. Of 5,017 cases there were only seven cross-infections, six of measles and one of diphtheria. These were attributed to lapses in aseptic precautions. Moizard thinks that this experience proves that even measles is not air-borne. Grancher, in another Paris hospital, has two wards in which there are no partitions, but only wire screens around the beds, simply as a reminder for the nurses. He also insists that measles is probably not an air-borne disease, and that

adjacent patients do not necessarily infect one another. At various English hospitals similar methods have been tried with success.⁵ The recent investigations of Thompson⁶ and also Harries⁷ in England certify to the efficiency of bed isolation when the nursing technic is efficient. This has become the common experience in hospitals for contagious diseases.

While the air plays a minor rôle in the spread of the infections, bad air plays an important part in reducing vitality and predisposing to disease. This will be discussed presently.

POISONOUS GASES IN THE AIR

Some of the poisonous gases of the air come from natural sources, as marshes, mines, or decomposing organic matter, but those that concern the sanitarian particularly are the gases which arise from the works of man. These gases are carbon monoxid, ammoniacal vapors, hydrochloric acid, carbon disulphid, carbureted hydrogen, hydrogen sulphid, arseniureted hydrogen, etc.

Carbon Monoxid.—Carbon monoxid (CO) is a frequent and serious cause of acute poisoning, often with disastrous after-effects. It has become one of the commonest forms of gas poisoning. Deaths from carbon monoxid in large cities now exceed those from any other poison.⁸ It is usually found associated with other gases, especially from the incomplete combustion of coal or wood, which takes place when there is insufficient oxygen, that is, carbon monoxid is formed instead of carbon dioxid and water. It is also one of the ingredients of illuminating gas especially of "water gas," and is one of the constituents of the gases of coal mines. Burning charcoal gives CO in great abundance, and it is also given off from red hot cast-iron stoves; further, it is found about lime kilns and where open coke fires and braziers are used in confined spaces; also from iron and copper furnaces, the exhaust of gas engines and from many manufacturing processes.

The gases from stoves or furnaces contain 79.7 per cent nitrogen, 10 to 13 per cent oxygen, 0.6 per cent carbon dioxid and 0.3 to 0.5 per cent carbon monoxid when formed by the incomplete combustion of wood or coal in closed spaces; that is, with the damper closed. Illuminating gas from coal contains from 6 to 10 per cent carbon monoxid; from wood 62 per cent; water gas, 30 per cent and more. Gases in coal mines contain from 4 to 10 per cent carbon monoxid, from 0.6 to 1 per cent hydrogen sulphid, and 53 per cent carbon dioxid. Gases from smokeless powder and gun cotton contain high quantities of carbon monoxid and are quite dangerous.

For public health purposes the chief sources of carbon monoxid are leaky

⁵ Chapin, *J. Am. M. Ass.*, 1908, 51: 2048.

⁶ *Lancet*, June 9, 1923.

⁷ *Ibid.*, March 8, 1924.

⁸ The total number of cases of gas poisoning in Cook County, Illinois, for 1916 was 501,—nearly 8 per cent of the entire number of coroner's cases. (*J. Am. M. Ass.*, 1918, 69: 257.) See also reports of the Commission on Resuscitation from Carbon Monoxid Asphyxia, *J. Ind. Hyg.*, 1922-3, 4: 463; 1923-24, 5: 109-125; *J. Am. M. Ass.*, 1922, 79: 1137.

gas fixtures, open coal fires, the premature closing of dampers of stoves and furnaces, or defects in apparatus burning coal, spent gases from automobiles and explosives in technical pursuits.

Air containing 0.4 per cent of carbon monoxid may, in one hour, prove fatal. In higher concentration a person may be overcome at once and death soon ensues. Kinnicut⁹ states that breathing an atmosphere containing 0.3 per cent of carbon monoxid, for any considerable period, is fatal, and the presence of 0.2 per cent is capable of destroying life (Haldane). Gréhan found that inhalation of an atmosphere containing 1 part of carbon monoxid to 275 parts of air was fatal to a dog, and that 1 in 70 killed a rabbit. Less than a gram of carbon monoxid may kill a man. Breathing an atmosphere containing 0.05 per cent of carbon monoxid may cause unpleasant, even serious symptoms (Oliver). Even as little as 0.07 to 0.12 per cent for half an hour will render one quarter of the red corpuscles incapable of uniting with the oxygen. When the amount in the air is not more than one part per thousand, a man at rest can breathe it for two and one-half hours before the blood takes up as much as 50 per cent; with two parts per thousand, utter powerlessness and unconsciousness come on; and three parts mean death unless rescue is fairly prompt. Two parts per ten thousand in air may be breathed for a long time without perceptible symptoms; four parts do not cause any appreciable harm for a short time; ten parts cause headache and symptoms; and fifteen to twenty parts become dangerous.¹⁰ The effects of carbon monoxid in the air breathed depend upon the rapidity of breathing, the presence of other gases, the age of the individual, muscular exertion, temperature, humidity and other effects. Within limits the quantity of carbon monoxid present in the air is somewhat more important than the length of exposure to it. Because of the increased disabling effect of muscular exertion, it has happened in mine accidents that the rescuers have suffered more severely than those they came to save. Hence, the importance of avoiding unnecessary exertion when exposed.

A child having relatively large respiratory exchange for its size is overcome more quickly than an adult. On this account, small animals, such as birds and mice, are overcome more quickly than men. Canaries are always carried by the rescue crews of the Bureau of Mines when carbon monoxid is suspected.

With increasing concentrations of carbon monoxid, the time required for a given amount of hemoglobin to combine with carbon monoxid would decrease very rapidly, until with 1.0 per cent it may require only time enough to take a few breaths to produce a saturation of 60 to 80 per cent, which is usually

⁹ *J. Am. Chem. Soc.*, 1900, 22: 14.

¹⁰ These figures correspond to the studies of Henderson and Haggard (*Rep. State Tunnel Com.*, 1921) in connection with the Holland vehicular tunnel under the Hudson River. The great problem in such enterprises is one of ventilation, with especial reference to removal of exhaust gases containing carbon monoxid. Henderson advised that if tunnels were so constructed that persons passing through would be exposed to not more than four parts of carbon monoxid in 10,000 parts of air (0.04 per cent) for not longer than forty-five minutes, they would experience no ill effects.

fatal. Roughly, for a person at rest, it can be assumed that 80 per cent of the equilibrium values is attained after the following periods of time:

Percentage Concentration of Carbon Monoxid in Air (Inclusive)	Percentage Blood Saturation (80% of Approximate Equilibrium Values)	Time
0.02—0.03	23—30	5 — 6 hours
0.04—0.06	36—44	4 — 5 hours
0.07—0.10	47—53	3 — 4 hours
0.11—0.15	55—60	1½ — 3 hours
0.16—0.20	61—64	1 — 1½ hours
0.20—0.30	64—68	½ — ¾ hours
0.30—0.50	68—73	20 —30 minutes
0.50—1.00	73—76	2 —15 minutes

Carbon monoxid or carbonic oxid is a colorless, tasteless and practically odorless gas; it burns with a pale blue flame. Its poisonous action depends upon the fact that it combines with the hemoglobin of the red blood-corpuscles to form carbon-monoxid-hemoglobin. This is a stable compound which, therefore, prevents the hemoglobin giving up its oxygen to the tissues. When present in only small amounts and for long periods of time, the effects of carbon monoxid may be compensated for by a polycythemia—an increased number of corpuscles taking the place of those disabled. It is commonly stated that carbon monoxid has a direct destructive action upon the cells of the central nervous system, causing paralysis in acute cases and psychoneuroses in chronic cases. Yandell Henderson, however, denies any direct action because tissue cultures will grow in vitro in a concentrated atmosphere of carbon monoxid.

There is no satisfactory evidence that carbon monoxid damages the tissues other than through oxygen starvation, and chronic poisoning is denied. The question is still in dispute. The carbon monoxid taken in is always sooner or later discharged in pure air, and a person is therefore never continuously in air containing carbon monoxid. Thus, it does not act as a cumulative poison. There may be, however, a cumulative effect of all the injuries done to the blood and indirectly to the tissues. The damage is due mainly to a disturbance of nutrition from anoxemia, but may also result indirectly from toxic products which may be formed. Persons much exposed to carbon monoxid have a compensatory increase in the number of red blood-cells and are in very much the same situation as those who live in high altitudes.

Symptoms.—The symptoms of carbon monoxid poisoning may be divided into two stages, the first covering the period beginning with normal and ending in syncope, and the second a depression of the central nervous system beginning in syncope, extending through coma, and ending in apnea. The symptoms have a direct relationship to the amount of carbon monoxid.

The following symptoms are caused by various percentages of carbon monoxid in the blood:

*Percentage of Blood**Saturation**Symptoms*

0-10	No symptoms.
10-20	Tightness across forehead; possibly slight headache, dilatation of cutaneous blood-vessels.
20-30	Headache; throbbing in temples.
30-40	Severe headache, weakness, dizziness, dimness of vision, nausea and vomiting, collapse.
40-50	Same as previous item with more possibility of collapse and syncope, increased respiration and pulse.
50-60	Syncope, increased respiration and pulse; coma with intermittent convulsions; Cheyne-Stokes's respiration.
60-70	Coma with intermittent convulsions, depressed heart action and respiration, possibly death.
70-80	Weak pulse and slowed respiration; respiratory failure and death.

The individual feels dizzy and complains of headache, noises in the ears, throbbing in the temples, and the feeling of sleepiness and sense of fatigue. There may be vomiting and a sense of oppression at the chest, palpitation, and an inability to stand or walk straight. Convulsions may or may not come on; pupils are dilated and react slowly to light; the face is red; consciousness is gradually lost, but owing to the great loss of motor power the individual, though aware of the danger, is often unable to escape from it. The onset may be as sudden as a stroke of apoplexy. In animals the heart beat at first is slow, while the blood-pressure is high; in man the action of the heart is frequently violent even during the stupor. When a man has recovered from the acute effects of carbon monoxid his life is still imperiled for some days to come. He runs the risk of dying as late as eight days after the accident and then he has still to face the risk of glycosuria, or other serious sequelæ. There is no known poison producing effects so varied and so widespread as carbon monoxid.

When a person is removed from the poisonous atmosphere there is slow return to consciousness, but headache, nausea and weakness persist for a long time. In case of continued inhalation of the poison there is a marked dilatation of the peripheral vessels, causing extensive red spots on the skin.

Death occurs from paralysis of the respiratory apparatus. If a case does not terminate fatally, there may be serious sequelæ, such as apoplexy followed by softening of the brain, or blisters, decubitus, or paralysis may develop, also chorea, idiocy, or minor grades of psychoneurosis.

The postmortem appearance, following acute intoxication with carbon monoxid, shows the following: features placid, face and skin bright ruddy color, both arterial and venous blood bright cherry red, and showing the

spectrum of carbon-monoxid-hemoglobin. The muscles, brain, and all organs are more pink than usual. Lungs may be emphysematous, and red patches may be observed on the surface of the abdominal viscera; occasional submucous hemorrhages in the stomach and intestines.

Tests.—The presence of carbon monoxid in air may be determined with considerable accuracy with a solution of blood. A few cubic centimeters of normal blood solution are shaken to saturation with the sample of air. A dilute blood solution is yellow; it becomes pink when treated with traces of carbon monoxid. By comparing the color with carmin this method will serve for quantitative purposes.¹¹ More accurate determinations may be made by the iodine pentoxid method of Seidell,¹² or more rapidly by the pyrotannic acid method.¹³

The diagnosis of carbon monoxid poisoning is made by examination of the blood for carbon-monoxid-hemoglobin. Sayers and Yant¹⁴ developed the pyrotannic acid method for this test. By this, a small amount of blood, 0.1 cubic centimeter, which can be procured by a prick of the finger, is enough to make a quantitative determination in a few minutes.

Illuminating Gas.—Illuminating gas may be harmful either from the products of its combustion or, more so, when the unconsumed gas escapes in the household. The two principal illuminating gases used are coal gas and water gas. The poisonous effects of both are due mainly to the carbon monoxid which they contain.

Coal gas is made by the destructive distillation of coal. It contains hydrogen, marsh gas, and carbon monoxid, occasionally also ethylene, acetylene, and carbon dioxid. A cubic foot of coal gas completely burned gives to the atmosphere about one-half a cubic foot of carbon dioxid and about 1.34 cubic feet of water vapor. An ordinary gas jet burns about six cubic feet of gas per hour, and thus produces about three cubic feet of carbon dioxid.

Water gas is made by blowing a current of steam through incandescent coke or coal. The water is decomposed into hydrogen and oxygen. The hydrogen passes on and the oxygen unites with the carbon to form carbon monoxid. Water gas so produced burns only with a pale blue flame. It is, therefore, enriched in a carburetor with vaporized petroleum; this furnishes the hydrocarbons necessary to give a luminous flame. Water gas contains about 30 per cent of carbon monoxid.

One of the most common sources of carbon monoxid in the household is from illuminating gas. Illuminating gas may pass from a broken gas main

¹¹ For methods for determining carbon monoxid and other gases in the air, see: J. S. Haldane, *Methods of Air Analysis*, J. P. Lippincott, 1912. Dennis, *Gas Analysis*, New York, 1913; A. Melzel, "Ueber den Nachweis des Kohlenoxydhämoglobins," *Verhandlungen der Physikal.-Med. Gesellschaft zu Würzburg*, n. f., 23: 47; F. Müller, "Biologische Gasanalyse," *Handbuch der Biochemischen Arbeitsmethoden*, 3: 2, Berlin, 1910.

¹² *J. Indust. & Engin. Chem.*, 6: 321; also Teague, *ibid.*, 1920, 12: 967.

¹³ *U. S. Pub. Health Rep.*, 1923, 38: 2311.

¹⁴ U. S. Bureau of Mines, Tech. Paper 373, 1925; *U. S. Pub. Health Rep.*, 1923, 38: 2053.

through the soil into the cellar and thence permeate a dwelling; this is aided by the suction and pumping action of the furnace in the cellar. In passing through the soil illuminating gas may be robbed of its characteristic odor, thus rendering it so much more dangerous because not perceived. The danger from this source is further increased in the winter time and in cities with asphaltum and concrete pavements, because under these circumstances the escape of gas into the air is hindered and the chance of more of it reaching the house through the cellar is favored. An occasional source of carbon monoxid in the air of houses is through hot-water heaters, using illuminating gas as fuel. The soot gradually collects in these devices and may become incandescent, thus furnishing ideal conditions for the production of carbon monoxid. In the arts carbon monoxid is formed by passing water vapor over incandescent carbon. I know of one case in Washington where carbon monoxid from a water heater collected in a kitchenette in such concentration that three persons were overcome upon entering the room and died.

In an ordinary sized bathroom, an instantaneous heater may generate enough carbon monoxid in fifteen minutes to asphyxiate the bather.

The flueless gas heating stove is now responsible for much of the poisoning in this country, and the radiant heater has been found the most dangerous. During the winter 1922-23 in the state of Ohio there were sixty-four deaths and 113 cases of partial asphyxiation. Of these, forty-one deaths and forty-six asphyxiations were due to heaters of the radiant type. In the next year, 1923-24, there were sixty-one deaths, forty-two of which were due to gas heaters.

It is not generally understood that carbon monoxid poisoning often occurs when gas heaters, without flues, are burned in the middle of a room. The same hazard comes from gas heaters in a "fireplace" without a chimney. Under these conditions, carbon dioxid is reduced to carbon monoxid. The latter poisonous gas, which is almost without odor, is not necessarily due to incomplete combustion, but to catalytic decomposition from the iron of the heaters which furnish the radiating surfaces. Natural gas, in the Middle West contains no carbon monoxid, but this gas is formed on burning and under the above circumstances poisoning is by no means uncommon.¹⁵

Most coal contains sulphur, which appears in coal gas as sulphuric acid, which is irritating and poisonous. Most of the sulphur compounds in coal gas are removed by processes of purification during manufacture, but, owing to the difficulty of complete removal, 20 grains of sulphur in every hundred cubic feet are generally allowed by law. The sulphur restrictions have recently, but unwisely, been removed in England. In Massachusetts the legal limit has been raised to 30 grains per hundred cubic feet. These changes were brought about by the claims of gas companies that it is much more difficult than formerly to procure coals low in sulphur, so that the processes for the removal of the sulphur have become costly and burdensome.

¹⁵ Yant and Jones, *J. Am. Soc. Heat. and Ven. Eng.*, 1923, 29: 669.

Illuminating gas is required by law, in Massachusetts and in many other places, to be free from ammonia as well as sulphurated hydrogen, but this is more because of injury to fixtures than because of danger to health.

The effect of these carbonaceous illuminants is to elevate the temperature and increase the moisture of a room. They also add carbon monoxid, carbon dioxid, nitric and nitrous acids, compounds of ammonia and sulphur, marsh gas, carbon particles (soot), acids of the fatty group in small but variable amounts. The following instructive table gives the comparative candle power and also the gases and heat produced by the usual forms of illuminants:

Form of Illuminant	Quantity Consumed	Candle Power	Oxygen Re- moved, Cubic Feet	Carbon Dioxid Pro- duced, Cubic Feet	Mois- ture Pro- duced, Cubic Feet	Heat Calories Pro- duced	Vitia- tion Equal to Adults
Tallow candles.....	2,200 grains	16	10.7	7.3	8.2	1,400	12.0
Sperm candles.....	1,740 grains	16	9.6	6.5	6.5	1,137	11.0
Paraffin oil lamp....	992 grains	16	6.2	4.5	3.5	1,030	7.5
Kerosene oil lamp...	909 grains	16	5.9	4.1	3.3	1,030	7.0
Coal gas, No. 5 Bats- wing burner.....	5.5 cubic feet	16	6.5	2.8	7.3	1,194	5.0
Coal gas, Argand burner	4.8 cubic feet	16	5.8	2.6	6.4	1,240	4.3
Coal gas, regenera- tive burner.....	3.2 cubic feet	32	3.6	1.7	4.2	760	2.8
Coal gas, Welsbach incandescent	3.5 cubic feet	50	4.1	1.8	4.7	763	3.0
Electric incandescent light	0.3 lb. coal	16	0.0	0.0	0.0	37	0.0

Water gas is cheaper than coal gas, and is, therefore, preferred by gas companies. Usually a mixture of the two gases is supplied. Experience shows that if water gas is properly diluted with coal gas the danger is greatly lessened. Illuminating gas containing 6 per cent of carbon monoxid is not hazardous. Most cities limit the amount to 10 per cent. In 1890, the 10 per cent statute was repealed in Massachusetts, and it is since then that the marked increase in illuminating gas poisoning has occurred. There were 1,231 deaths caused by illuminating gas in Massachusetts during the years 1886 to 1909. About one-half of these deaths were suicidal. This only represents the fatalities.

Sedgwick and Schneider¹⁶ stated in 1911 that the death rate from poisoning by illuminating gas in Massachusetts and Rhode Island had become nearly equal to that of scarlet fever or measles.

The employees from gas companies who suffer most from gas are the testers of meters and the men doing extension work; that is, making connections for new lines and working in ditches, trenches and manholes. According to Hamilton,¹⁷ officials of gas companies expect the men on extension work to

¹⁶ *J. Infect. Dis.*, 1911, 9: 3.

¹⁷ *Industrial Poisons in the United States*, Macmillan, N. Y., 1925, p. 371.

get gassed, and state that the only thing to do is to have someone watch at the top of the ditch, and when the man staggers and falls pull him out and send another in. The Committee on Accident Prevention of the American Gas Institute reported some 11,000 accidents in industry between 1915 and 1919. There were thirty deaths, only four of which were caused by gas. The records for 1919 cover about 1,000 accidents, occurring in seventy-seven plants. Thirty of these were men overcome with gas, but none of them died. The opinion held by this Committee is that practically all cases of gassing recover within forty-eight hours with no permanent effects.

Gas pipes in a dwelling should be tested from time to time with a pressure gauge, and minor leaks from faulty stopcocks, from "rubber" tubing used for droplights, etc., should be carefully searched for and corrected. A flaring gas burner is not only wasteful, since it implies the escape of unburned gas, but is also harmful to health. A gas jet should burn steadily without jumping and flaring. The incandescent mantles of the Welsbach type are more efficient illuminants and contaminate the air less than the naked gas flames.

It is a hygienic paradox that in the United States we have strict laws concerning plumbing, but any old gas fixture will do.

Hydrogen Sulphid.—Hydrogen sulphid is a colorless, transparent poisonous gas possessing the smell of rotten eggs. It is a product of the putrefaction of organic substances containing sulphur, and therefore found where vegetable or animal matter is undergoing decay. It is also generated by the decomposition of organic matter by anaërobic bacteria in deep lakes and ponds, tainting the water, in which it is soluble, or coming to the surface and tainting the atmosphere where its presence is indicated by the discoloration caused to neighboring dwellings painted with white lead. Small sluggish streams receiving the sewage of towns become defiled with this gas from which source it may be discharged in noticeable quantities as it is carried by the wind in different directions. Hydrogen sulphid is formed spontaneously whenever a soluble sulphate remains in contact with decaying organic matter with deficiency of air. It is also formed directly by the union of sulphur and hydrogen, and indirectly by the action of acids on sulphids; it is found in the gases contained in some ground waters; further, in some mining, smelting, and other industrial processes; and in illuminating gas, which contains traces. Its intense odor enables it to be recognized when present in minute quantities, 1 part in 10,000 being easily noted. It is slightly heavier than air—specific gravity, 1.1912.

Toxic Action.—Hydrogen sulphid is very poisonous. Six to ten parts in 10,000 parts of air; that is, 0.06 to 0.1 per cent is sufficient to cause serious symptoms within a few minutes. As a toxic agent, hydrogen sulphid stands between hydrocyanic acid and carbon monoxid. The comparative lethal doses are: hydrocyanic acid, 0.12 per thousand parts of air; hydrogen sulphid, 0.5; and carbon monoxid, 1.5. The exact method of hydrogen sulphid poisoning is still unknown. Sulphmethemoglobin is formed and the gas may be directly poisonous to delicate nervous structures. The susceptibility of man to this

gas varies. Its dangerous nature is fully recognized in all chemical laboratories. The effects of small amounts are not well understood; Thackrah could find no bad effects. On the other hand, Hirt believed it produced chronic poisoning, the symptoms being chiefly weakness, depression, anorexia, slow pulse, furred tongue, and marked pallor.

According to Lehman an atmosphere which contains 0.7 to 0.8 of hydrogen sulphid per thousand liters of air is dangerous to human life, while air containing 1 to 1.5 per thousand destroys life rapidly. Vivian Lewes states that man is killed in one and one-half minutes after breathing air containing 0.2 per cent of hydrogen sulphid. A concentration of 0.005 will cause poisoning. Death may come on like a stroke of lightning. The sudden death of men when working in sewers is sometimes supposed to be due to sulphurated hydrogen.

The symptoms caused by exposure to considerable amounts of hydrogen sulphid are redness and pain of the eyes, nasal catarrh, and irritation of the mucous membrane and bronchi, dyspnea, cough, rapid beating of the heart, dizziness, headache, numbness, and cold perspiration. Sudden exposure to large volumes of the gas causes death with striking rapidity; respiration stops before the heart. Death results from complete paralysis of the central nervous system, and even though persons are rescued, they may subsequently succumb from bronchopneumonia, caused by the irritating nature of the gas.

Autopsies performed immediately after rapid death disclose no changes, not even in the blood. In case of slow death edema of the lungs or pneumonia will be present, and the body has the characteristic odor of hydrogen sulphid. If death comes more slowly asphyxia is added to the nervous symptoms; the blood is dark and its hemoglobin may be altered, while the urine may contain albumin or sugar.

Hydrogen Sulphid in Sewers.—Workmen in excavations are sometimes overcome by hydrogen sulphid, when a spring containing this gas is tapped. Workmen are also occasionally overcome in the dead ends of sewers, in gate chambers or manholes, and in these cases hydrogen sulphid is sometimes said to be the cause of the accident.

Hydrogen sulphid is formed from sewage by the breaking down of protein and also by bacterial action upon inorganic sulphates.

In America, and, so far as known, in Europe, there are no data indicating that this gas ordinarily is present in measurable quantities in sewers. At Worcester it is stated that careful examination of large volumes of sewer air failed to show the presence of either hydrogen sulphid or carbon monoxid. At Lawrence it is also stated that hydrogen sulphid has never been detected in measureable amounts in the gas of any of the septic tanks.

On the other hand, it is known that hydrogen sulphid makes its presence known around a number of septic tanks by the discoloration of lead paint, and even from the odor of the gas, as well as its disintegrating effects upon masonry. W. Thwaites¹⁸ records from 0.2 to 1.1 per cent of free hydrogen

¹⁸ *Tr. Am. Soc. Civil Eng.*, 54: Part E, 214-230.

sulphid per volume and 0.2 to 0.9 per cent of combined hydrogen sulphid in the sewerage system of Melbourne, Australia. The sewage of this city flows for a distance of about eighteen to twenty-five miles and is applied to sewage farms. Hydrogen sulphid combines readily with basic constituents of sewage and thus differs from methane, nitrogen, hydrogen, and other gases arising from decomposition.

The spent liquors from tannery wastes sometimes contain calcium sulphid, which is used to remove the hair from the hides, and also sulphuric acid which is used in one of the processes of tanning. When the acid meets the calcium sulphid, hydrogen sulphid is evolved, endangering those in the sewers or along the trunk lines. I investigated two fatalities attributed to this cause occurring in Stoneham, Massachusetts.

The amount of hydrogen sulphid ordinarily found in "sewer gas" which may escape into houses as a result of defective plumbing is so small and so dilute as to produce no known symptoms. Hydrogen sulphid is by no means the only malodorous product of the decomposition of sewage; indol, skatol, cadaverin, mercaptan and other ill-defined products are even more offensive than hydrogen sulphid.

Methane (CH_4), also called "marsh gas," "fire damp" or "light carburated hydrogen," is found in nature as "natural gas" in and about coal and oil regions. Methane is very light compared to air (specific gravity 0.5596), and forms an explosive mixture as soon as it amounts to 1/18 of the volume of the air. Fortunately, the mixture does not ignite readily, but is nevertheless the cause of many accidents in mines. The gas has no odor, is slightly soluble in water, burns with a pale smokeless flame, yielding watery vapor and carbon dioxid—"after damp." Methane forms a large proportion of illuminating gas. It is usually regarded as an indifferent gas, but it probably has slight toxic properties. Haldane found that 5.5 per cent of methane had no effect on man; 45 per cent causes slower and deeper breathing, and 70 per cent endangers life. With 70 per cent the oxygen is reduced to only 6.3 per cent and the nitrogen to 23.7 per cent.

The table shows the percentage composition of illuminating gases, with the gases of an Imhoff tank, for comparison.

Constituents	Imhoff Tank, per Cent	Natural Gas, per Cent	Water Gas, per Cent	Coal Gas, per Cent
Hydrogen	8.6	1.31	30	47.49
Methane	84.1	87.75	24	38.67
Carbon dioxid	4.6	6.6	1.04
Nitrogen	3.1	4.34	2.5	.85
Carbon monoxid	29	6.74
Oxygen42
Hydrogen sulphid	1.5
Ethylene	12.5	5.21

Methane is also given off in large quantities from decomposing matter in swamps, sewers and septic tanks. Methane may constitute 70 to 80 per cent

of the gases found in a septic tank. The gases from an Imhoff tank may be used for illumination and heating.

Sulphur Dioxid.—Sulphur dioxid is extremely irritating and causes bronchitis. Those exposed to the fumes in the bleaching of cotton and worsted goods are frequently sallow and anemic.

Other Gases in the Air.—*Ammoniacal Vapors.*—Ammoniacal vapors irritate the conjunctiva, but have no other evident effect on health in the amounts ordinarily found in the air.

Hydrochloric Acid Vapors.—Hydrochloric acid vapors in large quantities are very irritating to the conjunctiva and respiratory mucous membranes. In the alkali manufactures they are sometimes poured into the air in sufficient quantity to destroy vegetation. When in sufficient concentration they may induce bronchitis, pneumonia, and even destruction of lung tissue, as well as inflammation of the eyes.

Carbon Disulphid.—Carbon Disulphid is given off in the vulcanizing of India rubber. It produces headache, vertigo, pains in the limbs, formication, sleeplessness, nervous depression, and loss of appetite; sometimes deafness, dyspnea, cough, febrile attacks, and even paraplegia. The effects seem due to a direct anesthetic action on the nervous tissue.

Other poisonous gases are discussed in the chapter on Industrial Hygiene, page 1247.

RESUSCITATION

Artificial Respiration.—As soon as the patient is clear of the gas quickly feel with your finger in his mouth and throat and remove any foreign body (tobacco, false teeth, etc). If the mouth is tight shut, pay no more attention to it until later. Do not stop to loosen the patient's clothing, but immediately begin actual resuscitation. Every moment of delay is serious. Proceed as follows:

1. Lay the patient on his belly, one arm extended directly overhead, the other bent at elbow and with face to one side, resting on the hand or forearm, so that the nose and mouth are free for breathing (see Fig. 73).
2. Kneel straddling the patient's hips with knees just below the patient's hip bones or opening of pants pockets. Place the palms of your hands on the small of the back with the fingers over the ribs, the little finger just touching the lowest rib, the thumb alongside of the fingers; the tips of the fingers just out of sight, as in Fig. 73.
3. While counting one, two, and with arms held straight, swing forward slowly so that the weight of your body is gradually, but not violently, brought to bear upon the patient (see Fig. 74). This act should take from two to three seconds.
4. While counting three, swing backward so as to remove the pressure, thus returning to the position shown in Fig. 75.
5. While counting four, five, *rest*.

6. Repeat these operations deliberately, swinging forward and backward twelve to fifteen times a minute—a complete respiration in four or five seconds. Keep time with your own breathing.



FIG. 73.—POSITION IN WHICH PATIENT SHOULD ALWAYS BE PLACED AND KEPT UNTIL CONSCIOUS; ALSO FIRST POSITION FOR OPERATOR STARTING ARTIFICIAL RESPIRATION. (American Gas Association, 1923.)

7. As soon as this artificial respiration has been started, and while it is being continued, an assistant should loosen any tight clothing about the patient's neck, chest, or waist. Keep the patient warm.

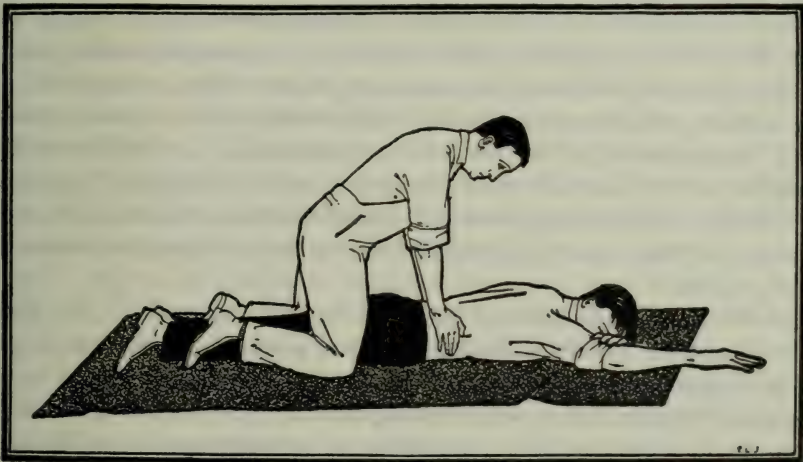


FIG. 74.—SECOND POSITION OF OPERATOR GIVING ARTIFICIAL RESPIRATION. (American Gas Association, 1923.)

8. Continue artificial respiration without interruption until natural breathing is restored, if necessary four hours or longer, until a physician de-

clares rigor mortis (stiffening of the body) has set in. If natural breathing stops after being restored, use resuscitation again.

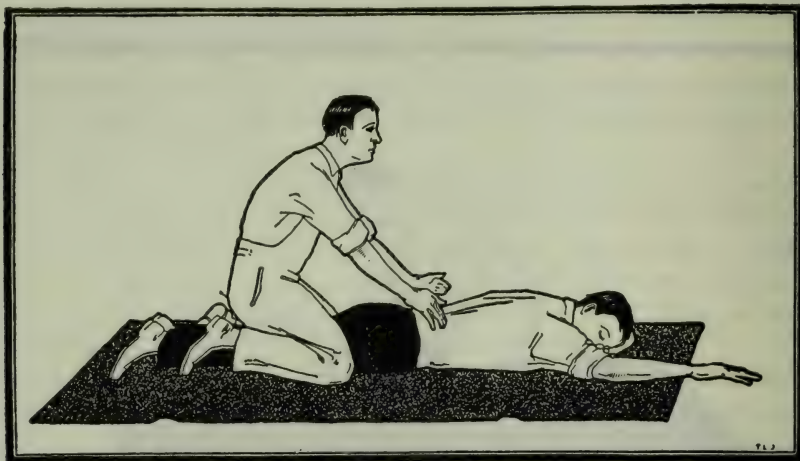


FIG. 75.—THIRD POSITION OF OPERATOR GIVING ARTIFICIAL RESPIRATION.
(American Gas Association, 1923.)

Inhalation.—It has been suggested by Henderson and Haggard¹⁹ that a mixture of 95 per cent oxygen and 5 per cent carbon dioxid be used for resuscitation from carbon monoxid poisoning. In these proportions the mixture affords the oxygen necessary to displace carbon monoxid from the combination of hemoglobin and carbon monoxid in the blood while the 5 per cent carbon dioxid stimulates respiration; one or two shallow breaths of this mixture cause a gassed subject, who may be barely breathing, to take a deeper breath which in turn causes further breathing. In a normal resting man this mixture causes the total lung ventilation to increase three to fourfold.

A suitable portable apparatus for administering this mixture is made by several companies while the method is recommended by the Resuscitation Committee of the American Gas Association and by Haldane.²⁰ Drinker²¹ has published an interesting account of actual results in the use of the method particularly by the rescue squads of the various metropolitan gas companies in our large cities.

The victim should be removed to fresh air as soon as possible. The Resuscitation Committee recommends that the first emergency measure in gassed cases is to apply artificial respiration by the Schäffer prone pressure method. If the gassed subject has ceased breathing he cannot be revived by the oxygen-carbon dioxid alone: artificial respiration is necessary and must be applied at once. If the oxygen-carbon dioxid treatment can be administered at the same time as artificial respiration is applied it has been found in actual rescue

¹⁹ *J. Am. Ass.*, 1922, 79: 1137.

²⁰ *J. Indust. Hyg.*, 1926, 8: 50.

²¹ *Ibid.*, 1925, 7: 539.

work to be extremely helpful. The committee further makes it clear that the great danger in relying on devices like the pulmotor is that nothing is done to start respiration until the pulmotor arrives; in the interim the subject may die. Artificial respiration can be applied by anyone with a few demonstrations and lessons. Circulation should be aided by rubbing the limbs and keeping the body warm with blankets, hot-water bottles, hot bricks or other devices, care being taken that these are wrapped or do not come in contact with the body and produce burns. This aids in tiding the body over a period of low vitality. Other stimulants, such as hypodermics of caffein-sodio-benzoate, or camphor in oil, should not be administered except by a doctor after he has considered the possibility of overstimulation and consequent collapse. The fact that the pulmotor and similar devices are decidedly unphysiologic in their action is not perhaps so much an objection as causing otherwise competent helpers to stand by idly at the very time when help by the Schäffer prone pressure method would do the most good. The patient should be kept at rest, lying down in order to avoid any strain on the heart. Later, he should be treated as a convalescent and given plenty of time to rest and recuperate.

GAS MASKS, HOSE MASKS AND OXYGEN BREATHING APPARATUS ²²

Gas Masks.—As a result of the development of gas masks in the World War industrial gas masks are now available for protection against low concentrations of literally all the common industrial gases such as carbon monoxid, hydrogen sulphid, ammonia, hydrocyanic acid gas, vapors of benzene, carbon tetrachlorid, tetrachlormethane, etc. The headpiece is adapted from the United States Army 1919 Model ²³ and to it is attached a short length of non-collapseable tubing connecting to the canister. A rubber flap valve, through which the expired air passes, is attached to the face piece. Canisters can be obtained for use against low concentrations of all gases ²⁴ or against low concentrations of any particular gas. In addition to the gas-absorbing chemicals within the canisters are filters which serve to remove fine particles of mists, smokes, and fumes. Some felt-like substance is now generally used to filter out particles, as in smokes or fumes. An activated carbon made from cocoanut or other nut charcoal is used to absorb gases. Certain gases need special chemicals, such as soda lime, to neutralize them. Nothing will absorb carbon monoxid. For this gas, the canister contains a catalytic oxidizer which converts it into carbon dioxid. For moisture, calcium chlorid or other hygroscopic substance is used. The activity of any of the chemicals used is soon exhausted and they must therefore be renewed.

²² An excellent series of bulletins have been published by the United States Bureau of Mines on gas masks, hose masks, and oxygen breathing apparatus.

²³ E. B. Vedder, *The Medical Aspects of Chemical Warfare*, Williams & Wilkins, 1925.

²⁴ Bureau of Mines, Ser. No. 2719, Nov. 1905.

In general the life of the modern canister is but a few hours. It affords no protection whatever in oxygen deficiency. Where gas concentrations over 2 to 3 per cent are encountered, most masks are not safe. All of the best masks are now equipped with an ingenious timing device which indicates how long the mask can be worn with the assurance of the perfect protection it should afford.

Hose Masks.—A hose mask consists of the regular gas mask face piece and expiration valve, but in place of the canister pure compressed air is supplied at a pressure automatically controlled by inspiration. Either motor or hand driven compressors serve to pump air to the subject. Obviously a device of this kind will protect against any irrespirable atmosphere just so long as pure air is supplied through the hose which may be of any desired length. Hose masks find abundant industrial application in work such as cleaning out benzol tanks, lead burning in the holds of ships, and other work where gas masks are unsuitable and ventilation impractical.

Oxygen Breathing Apparatus.—In cases where men must enter uncertain atmospheres and hose masks cannot be used, oxygen breathing apparatus is essential. Here the subject carries his own oxygen supply and the life of the apparatus depends entirely on how much actual weight he can carry and still perform his work—usually vigorous rescue work. The oxygen is carried in a small tank on the man's back and passes through a special reducing valve to the face piece. Expired air passes through a "regenerator," which removes carbon dioxid, is then replenished automatically with whatever additional oxygen is needed. The chief application of this apparatus is for rescue work from fires or explosions in mines, buildings, etc., and in high altitude flying.

SEWER GAS

Sewer gas, once a hygienic bugaboo, is now not seriously regarded by sanitarians. Sewer gas became the residual legatee of Murchison's pythogenic theory, namely, that typhoid fever was "produced by emanations from decaying organic matter." People naturally cling to the notion that anything that smells bad must be detrimental to health; sanitarians know, however, that our sense of smell is a very poor sanitary guide.

Sewer gas is nothing more or less than air containing the volatile products of organic decay coming from sewers and drains. Sewer gas is a variable mixture both as to composition and concentration. Some of these gases are more or less poisonous, but not in the great dilution ordinarily found in sewer air. As a matter of fact, the air of sewers is ordinarily freer of dust and bacteria than the corresponding outside air, although it may be a little higher in carbon dioxid—ten to thirty volumes per 10,000. It is absurd to regard sewer gas as the cause of diphtheria, typhoid fever, scarlet fever, and other communicable diseases. So far as unpleasant odors are concerned, they are more apt to come from defective drains or unclean and unventilated house plumbing than from a well-constructed sewer. The subject of putrid odors

from sewers is a highly complicated one. Odors are due, in most part, to decomposition products, such as hydrogen sulphid, ammonia, indol, skatol, phosphin, mercaptan, phenol, and various acids, such as acetic, butyric, valerianic and other compounds. Workmen employed in sewers and about sewage ordinarily remain hale and healthy. Sewer gas as a rule is no more hurtful than the gases and odors a farmer subjects himself to on the manure pile.

Winslow and Greenberg²⁵ exposed guinea-pigs to strong odors and gases from putrefying feces, and found that there was a reduction in the rate of growth during the first week, but the animals soon became accustomed to the odor and the unnatural situation and attained after two weeks a normal growth.

Delepine²⁶ could not detect any influence of sewer air either upon the growth curve of cats, rabbits, and guinea-pigs, or upon their susceptibility to a spontaneous epidemic due to infected food.

Bacteria in Sewer Air.—When it was found that there are no dangerous volatile poisons in sewer air attention was focused upon the bacteria; however, Nägeli as long ago as 1877 showed that putrescent liquids kept in the same sealed vessel for over two years did not infect each other. Sir Edward Frankland then showed that lithium carbonate in solution did not contaminate the air, but that when effervescence was produced the breaking of the bubbles on the surface of the liquid carried the lithium a distance of twenty-one feet up a vertical tube. The inference was that sewage through fermentation or splashing may send bacteria into the air. Pumpelly, in 1881, and others since have shown that bacteria are not given off from a liquid if the surface remains unbroken, even though the air may blow over it. In 1893, Miquel began a monumental work upon bacteria of the air. He made routine observations at the Montsouris Observatory, and for four years compared the bacteria in the air of a Paris street with the air of sewers. He found sewer air relatively pure from a bacteriological standpoint. Carnelly and Haldane, in 1877, found fewer bacteria in the sewers under the House of Parliament and other places than in the air of adjacent streets. The number of bacteria was largest in the best-ventilated sewers, because these brought the street bacteria along with them. Abbott, in 1894, showed that cultures of *Bacillus prodigiosus* are not carried over in bubbles produced by natural fermentation (yeast in a carbohydrate medium), but may be carried a short distance by blowing air at considerable velocity through the culture. He concluded that the danger of bacteria being transmitted from sewage into the air under ordinary circumstances is practically negligible. In 1907, Horrocks revived this question by placing *B. prodigiosus* in the water-closets of a large military hospital in Gibraltar, and recovering them on plates suspended on top of the soil pipes and in manhole openings. His work gave countenance to the views of a number of English sanitarians, who maintain the reality of the danger

²⁵ *Proc. Soc. Exper. Biol. & Med.*, May 15, 1918.

²⁶ *Report of Sewer Ventilation Committee upon the Effects on Health of the Air of the High Street Sewer in Manchester*, 1909.

from this source. Winslow repeated Horrocks' experiments in 1909, using the ordinary sewage of Boston, and by using quantitative methods threw a different light upon Horrocks' conclusions. He found that a vigorous foaming produced very slight bacterial infection of the air—only five prodigious colonies in thirty liters of air. Further, the infection always remained localized. Generally he found the air of house drains singularly free from bacteria. It, therefore, seems theoretically possible, but very improbable, that infection may take place in this way. Practically the question seems to have little importance. Thus, out of a series of examinations of plumbing systems in actual use, Winslow found intestinal bacteria only four times in two hundred liters of air, and these directly at the point of local splashing.

If there is any danger of sewage bacteria coming into our houses, it is rather that they are dragged in by rats, roaches, water bugs, and other vermin that use sewers and drains as highways.

Accidents in Sewers.—Workmen who enter parts of a sewerage system are sometimes overcome. Practical sewermen know that the danger is found in the dead ends, gate chambers, manholes, and similar places where the gases can accumulate; also in suddenly relieving an obstruction in a sewer, thus permitting a blast of gases. There are no such dangers in a well-ventilated sewer. Stagnant sewage and sludge, combined with lack of ventilation, form conditions resembling a septic tank, and it is this combination that menaces the workers. A free flow of sewage promotes ventilation and diminishes the chances of gases accumulating.

The principal gases given off from sewers are methane, carbon dioxid, hydrogen, ammonia, and sometimes hydrogen sulphid. None of these gases is particularly poisonous, excepting the last. When carbon monoxid is found in sewer air it does not come from the decomposition of the sewage, but from illuminating gas which leaks in. Many analyses of sewer air in many different places show that hydrogen sulphid is usually absent.

The following table gives the approximate percentage of gases found in settling tanks in various cities of the United States:

City	Methane	Nitrogen	Carbon Dioxid	Oxygen	Hydrogen
Atlanta	84.1	3.1	4.6	.4	8.6
Chicago	84.9	6.2	8.6
Urbana	81	6.1	12.3	.13	...
Columbus	83.5	9.3	8.1
Worcester	57.7	32.5	8.2	1.2	...
Lawrence	78.9	16.3	3.4	.5	...
Manchester	73	16	6	...	5

The danger in entering manholes, gate chambers, or dead ends of sewers is from: (1) illuminating gas which has leaked in; (2) asphyxia on account of an accumulation of carbon dioxid and methane; (3) trade wastes containing volatile substances, such as petroleum products from garages, dry cleaning

establishments, oil work, or gases caused by spent liquors from tanneries, chemical works, etc.; (4) poisonous gases, as hydrogen sulphid, which are said sometimes to accumulate in appreciable amounts.

Explosions in sewers are due to illuminating gas from leaks; benzol, naphtha, and gasolene from garages, dyeing and cleaning works, and also from lithographic works; hydrocarbon oils used by railroads to prevent freezing of switches, and other inflammable and explosive substances. The gases may be fired by open lights carried by workmen, by the sparks from trolley cars or fire engines passing over manholes, or by lightning. The prevention is constant ventilation; traps for catch basins, and furnishing workmen with electric lights or safety lamps to replace ordinary lanterns.

Illustrative Cases.—*Case No. 1.*—The main sewer of the town of Revere, Massachusetts, discharges into the sea near the southerly end of Revere Beach. In order that the sewage might not discharge on the incoming tide, a covered tank was built near the seashore into which the sewage collected, and the tank was constructed of such capacity that the sewage would not rise as fast as the tide. On the other hand, the bottom of the tank was somewhat above low tide. In consequence, the flow of the sewage was shut off by a tide gate when the tide was high for several hours and subsequently discharged automatically as the tide went down and the sea had fallen below half tide. Two men were sent to clean the tank, which was done by stirring up the sediment with a stream from a 2½-inch fire hose at about the time the tank would discharge. It appears that trouble had been previously experienced by gases in the tank chamber at such times, and the man in charge was warned of this, but he neglected to warn his helper whom he sent down into the tank; the helper was overcome and drowned. In the lawsuit the main question raised was whether the town agents exercised due care in the matter, and the jury decided that they did not, and awarded full damages to the plaintiff. No evidence was presented to show the character of the gas which doubtless caused the accident except that the evidence showed that the gas was odorless. It was generally believed by those interested in the case that the gas was carbon dioxid.

Case No. 2.—On August 6th Henry G. Parker,²⁷ an able and valued member of the staff of the Engineering Department of Los Angeles, lost his life while inspecting a weir chamber of the outfall sewer connecting the Los Angeles sewer system with the Pacific Ocean. Parker and Derby entered the chamber to test the working of the gate, which was raised and lowered by means of a hand-wheel and screw. In attempting to raise the gate they were overcome with the foul air and were required to retreat to the surface three separate times. The third time Parker suddenly fell a distance of twenty feet, and the body slipped from the sluiceway and was sucked into the sewer before rescue could be accomplished. Death was doubtless due in large part to the effect of the gases and the fall of twenty feet, and not to drowning, as

²⁷ *Engin. Rec.*, 1909, 60: 252.

very little water was found in the lungs. Just what gases were present is not stated.

Case No. 3.—During the pumping out of a manhole in Charleston, South Carolina, in May, 1911, a small boy was sent down to remove some chips which threatened to obstruct the suction pipe. He succumbed almost immediately. One of the workmen went down to rescue him; he also became unconscious. A third person, with a rope tied around him, went to the rescue of the two. When taken from the manhole, the small boy was dead, and the second man was unconscious. The symptoms reported by Jager and Jervey were those of hydrogen sulphid poisoning. A qualitative analysis of the air of the manhole made at a later time showed the presence of hydrogen sulphid and no appreciable quantities of carbon monoxid. It is assumed in this case that the hydrogen sulphid in the manhole probably accumulated and became concentrated in the bottom of the manhole, although it was recognized that other gases may have contributed to the gravity of the condition.

Case No. 4.—A laborer went into an intercepting sewer on Market Street in Lynn, Massachusetts, to inspect it, and after he had proceeded into the sewer about 250 feet his lantern exploded, covering him with blazing oil. The cause was assumed to be illuminating gas that had leaked into the sewer from some unknown place. The man had his hair singed and his face badly blistered, and was partly overcome. The sewer was not ventilated, nearby manholes having been closed.

Prevention of Accidents in Sewers.—To prevent accidents in manholes, and other parts of the system where sewer gases may collect, it is advisable for men always to work in pairs, and to be ready with rope, tackle and tripod in case of emergency. Accidents may be prevented by clearing out cul-de-sacs where sewer gases are likely to collect, before the men enter such places. This may be done by an air blast, but as this is usually not at hand the same purpose may be accomplished by a water spray having a good head and volume. This stirs up the air and ventilates the pocket. Under special circumstances, gas masks may be used.

Other precautions to determine the presence of dangerous or irrespirable gases consist in lowering a candle, a safety lamp, or small animals (canary or mouse) in a cage.

Ventilation of Sewers.—Sewers cannot be constructed airtight on account of the numerous openings into them. The tension of the air in sewers is generally not very different from that of the atmosphere outside. The movement of the air is generally in the direction of the flow of the current. The simplest plan of ventilation is by means of a shaft from the top of the sewer to the surface of the street or road above, where the opening of the shaft should be covered by an iron grating. These openings are usually placed at intervals of 100 yards or so. This system, which is in common use, has been much criticized, mainly on account of the fact that the objectionable gases are discharged more or less immediately under the noses of passers-by. To meet this objection it has been proposed, and actually come about in some places,

to locate tall iron shafts at suitable intervals to permit the discharge of air and gases at a level well above the roofs of houses. As a matter of fact, if sewers are well constructed, have sufficient fall and flow of water, there will be no accumulation of foul gases. One of the main causes of decomposition is due to dead ends. These should not be tolerated by the engineer in charge of the sewage department. Recently an agitation has been started to solve this question of sewage ventilation by advocating the abolition of the intercepting traps on the house drains between the sewer and the house, thus converting every house drain and every soil pipe into so many sewer ventilators. There are many objections to this plan, as it would destroy the drain isolation between the houses, which is now possible from the sewer, and from the neighboring houses of the district.

CHAPTER V

FRESH AND VITIATED AIR

The Benefits of Fresh Air.—Fresh air is nature's tonic. It stimulates digestion, promotes assimilation, improves metabolism, strengthens the nervous system, and increases our resistance against some diseases. It is a common experience that fresh air gives us a general feeling of well-being. Much of the benefit of an outdoor life comes also from the exercise, diversion, sunshine, and other factors. The stimulating effect of outdoor air varies considerably with the temperature and movements of the air. The ceaseless variations in the rate of cooling, evaporation and absorption of radiant energy in outdoor conditions, relieve us of monotony and stimulate tone and metabolism. Cold air is especially stimulating, and much of the good of sleeping out of doors is perhaps secondarily due to the tonic action of cold. Sleeping out of doors or with open windows atones for much bad air during the daytime. However, the good results of fresh air may be neutralized by undue exposure to cold, especially in the young, the aged, and the feeble—or even in robust individuals not properly protected. Extremes in this as in all matters hygienic are to be avoided.

The Effects of Vitiating Air.—The effects produced by an atmosphere vitiated by the breath and other exhalations from human beings may be divided into acute and chronic. The acute effects are usually lassitude, headache, vertigo, nausea, vomiting, and even collapse. In extreme cases death may ensue. The chronic effects, so far as is known, include anemia, debility, and disturbances of digestion. Prolonged exposure to vitiated atmospheres also influences nutrition and metabolism, depresses vitality, and lowers the resistance to certain infections, especially to the pyogenic cocci, the tubercle bacillus, the pneumococcus, and to the microorganisms causing common colds. It is often difficult, especially in the poorer classes, to know how much is due to bad air and how much to crowding, poor food, overwork, loss of sleep and rest, worry and other afflictions of poverty. There is plenty of evidence to show that men living in insufficiently ventilated barracks and other habitations have a high death rate. The lower animals under like conditions in crowded and poorly ventilated stables also have a high mortality. The statistical evidence of the English Barracks and Hospital Commission, published as long ago as 1861, shows that men living a considerable portion of their time in badly ventilated rooms have a higher death rate than those having well-ventilated rooms, other conditions being about the same.

The high morbidity and mortality in crowded places are due, in part at

least, to the favorable conditions for the spread of the communicable diseases, and must not be laid entirely to the effects of vitiated atmospheres. Crowding, rather than bad air, may be the dominant factor.

Some Extreme Cases.—The acute and fatal effects caused by breathing a seriously vitiated atmosphere, under unusually severe conditions, are well illustrated by the three following instances:

After the battle of Austerlitz three hundred Austrian prisoners were shut into a prison in a small cellar, and 260 were killed by the impure air in a few hours.

In the tragedy known as the Black Hole of Calcutta, the military prison of Fort William, January 18, 1756, 146 adults were shut into a room only eighteen feet square and with but two small windows on one side to ventilate it. They were shut in at 8 P.M., and within an hour some were dead, and when the door was opened at 6.20 next morning only twenty-three were found to be alive. One of the survivors gives the following description of the horrors of the night: "At this period so strong a flavor came from the prison that I was not able to turn my head that way for more than a few seconds at a time. Everybody except those at the windows now grew outrageous and many delirious. By eleven o'clock greater numbers were dead or dying, and those living were in an outrageous delirium and others quite ungovernable. A steam now arose from the living and the dead, which most awfully affected those who were still alive. At six o'clock next morning it came to the ears of the Indian governor the havoc death had made in this fearful place, and he ordered their release. At 6.20 there came out of this living grave twenty-three *half-dead creatures*, being all that remained of the 146 souls who had entered the Black Hole prison, and these were in such a condition that it seemed very doubtful whether they would see the morning of another day. Many of the survivors developed putrid fever and boils. The remaining twenty-three were poisoned by exhalations from their own lungs and bodies."

An almost equally terrible tragedy took place on the steamer *Londonderry*, going between Sligo and Liverpool. The episode is thus described by G. Henry Lewes (*Physiology of Common Life*):

"On Friday, December 2, 1848, she left for Liverpool with two hundred passengers on board, mostly emigrants. Stormy weather came on, and the captain ordered every one below. The cabin for the steerage passengers was only eighteen feet long, eleven feet wide, and seven feet high. Into this small space the passengers were crowded; they would only have suffered inconveniences if the hatches had been left open; but the captain ordered these to be closed, and—for some reason not explained—he ordered a tarpaulin to be thrown over the entrance to the cabin and fastened down. The wretched passengers were now condemned to breathe over and over again the same air. This soon became intolerable. Then occurred a horrible scene of frenzy and violence, amid the groans of the expiring and the curses of the more robust; this was stopped only by one of the men contriving to force his way on deck, and to alarm the mate, who was called to a fearful spectacle: seventy-two were al-

ready dead, and many were dying; their bodies were convulsed, the blood starting from their eyes, nostrils, and ears."

The foregoing instances are exceptional, and for practical purposes may be regarded simply as the results of suffocation, accentuated by heat and humidity and muscular struggle. The usual conditions never approach such extremes, but are, nevertheless, important, for they may be serious. We must first consider the question why an atmosphere vitiated by the presence of human beings produces ill effects.

Causes of Ill Effects Due to Vitiating Air.—Three explanations have been offered: (1) increase of carbon dioxide and diminution of oxygen; (2) poisons in the expired breath; (3) physical changes of the air. Each of these explanations is considered separately:

Increased Carbon Dioxide and Diminished Oxygen.—According to the older theories, the sensations of discomfort, arising in inclosed places, had their origin either in an excess of carbon dioxide or an insufficiency of oxygen. Thus, in the early experiments of Claude Bernard (1857) animals were confined in atmospheric air and in mixtures both richer and poorer in oxygen than atmospheric air. He explained the poisonous effects of carbonic acid when respired to be due to the fact that it deprived the animal of oxygen. Similar results were reported by Valentin and Bert. Richardson in 1860-61 found that a temperature much higher or lower than 20° C. had the effect of shortening very considerably the lives of animals confined in an unventilated jar. Pettenkofer in 1860 to 1863 cast the first serious doubt on the correctness of these theories. He believed that the symptoms observed in crowded, ill-ventilated places were not produced by the excess of carbonic acid nor by a decrease in the proportion of oxygen in the air. He further did not believe that the impure air of dwellings was directly capable of originating specific diseases, or that it was really a poison in the ordinary sense of the term, but that it diminished the resistance on the part of those continually breathing such air.

Hermans¹ showed that an atmosphere containing only 15 per cent of oxygen and as much as 2 to 4 per cent of carbon dioxide may not be harmful. On removing the carbon dioxide there was no great discomfort, even when the oxygen was reduced to 10 per cent. The air of certain breweries examined by Lehmann² contained 1.5 to 2.5 per cent of carbon dioxide, and men worked continuously in this for years without any ill effects. The carbon dioxide occasionally rose to 6 per cent and more, but this amount produces panting and distress. It is now generally admitted, upon the testimony of numerous experimenters, that an atmosphere containing as much as 3 per cent of carbon dioxide and as little as 15 per cent of oxygen has no toxic effects and produces no disturbing symptoms. In the most poorly ventilated rooms the carbon dioxide never reaches this amount, especially when produced by respiration alone. It is unusual to find 0.5 per cent. In the most crowded rooms the oxygen rarely reaches as low as 20 per cent. In mines the oxygen is often

¹ *Arch. f. Hyg.*, 1883, 1: 1.

² *Arch. f. Hyg.*, 1899, 34: 535.

deliberately kept down to 17 per cent with the object of avoiding dust explosions. It is, therefore, plain that an increase of carbon dioxid and a decrease of oxygen in the vitiated air of crowded rooms is not the cause of the trouble, and we must therefore look to other causes for the effects of vitiated air (see also pages 789 and 793).

Poisons in the Expired Breath.—In 1863, Hammond believed he demonstrated the presence of organic matter, because when vitiated air is passed through potassium permanganate, it decolorizes that strong oxidizing agent. Hammond confined a mouse under a jar in which the carbon dioxid was taken up by baryta water as fast as it was formed and the moisture absorbed by calcium chlorid. Nevertheless, the mouse died in forty minutes. The observation was repeated a number of times, and death ensued invariably in less than one hour. Brown-Séquard and D'Arsonval in 1888-9 claimed to be the first to demonstrate poisonous bodies in the expired breath. They condensed the moisture in the exhaled breath, which was injected into the veins of rabbits. Death usually took place in a few days, sometimes in a few weeks. They believed from this that they had discovered a volatile organic poison of the nature of an alkaloid, similar to Brieger's ptomains. These experiments were repeated with variable results, but in 1889 they reported ingenious experiments in which they obtained additional evidence in support of their former statements. Rabbits were confined in a series of jars connected with rubber tubing, permitting a constant current of air to be passed. The animal in the last jar received the air from the lungs of the animals in the other jars. This animal died after an interval of some hours, and the animal in the next jar was the next to die. The first and second animals usually remained alive. When absorption tubes containing concentrated sulphuric acid were placed between the last two jars, the animal in the last jar remained alive while the one in the jar just before was the first to die. These results confirmed their belief in the existence of a volatile poison absorbed by the sulphuric acid. Haldane and Smith repeated the experiments of Brown-Séquard and D'Arsonval, using five bottled mice. They continued the exposure for fifty-three hours without ill effects to the mice. Beu in 1893 also repeated these experiments, and came to the conclusions that acute poisoning through the organic matters contained in the expired air was not possible, and that the death of the animals was due to changes of temperature and accumulation of moisture in the jars. Rauer in 1893, also Lübbert and Peters, concluded from similar experiments that there are no organic poisons in the expired air. In fact, Merkel stood almost the only sponsor for the correctness of the conclusions of Brown-Séquard and D'Arsonval, and with some slight changes of technic he was unable to get uniform results.

Lehmann and Jessen, in 1890, collected from fifteen to twenty cubic centimeters of condensed fluid per hour from the breath of a person exhaling through a glass spiral laid in ice. This fluid was always clear, odorless, neutral in reaction, and contained slight traces of ammonia with good teeth; more with poor teeth. Inoculation of this condensed fluid into animals gave nega-

tive results. Many other experimenters, including von Hoffman-Wellenhof, Lehmann and Jessen, Haldane and Smith, Billings, Weir Mitchell, Bergey, and Rosenau, have shown that the fluid condensed from the breath is no more toxic than distilled water, when injected into animals. This has strengthened the general belief that poisonous bodies are not present.

In 1894, Brown-Séquard and Davis reported further experiments in which they inoculated over one hundred animals with the condensed fluid of respiration, and not only confirmed their former statements, but were unable to understand the failure of other experimenters, and emphatically reaffirmed that the breath contains a volatile poison and that the death of animals under experimental conditions is not due to an excess of carbon dioxide nor a deficiency of oxygen. These experiments were repeated by Billings, Mitchell, and Bergey,³ in 1895, who came to the conclusion that the ill effects of vitiated atmosphere depend almost entirely upon increased temperature and moisture, and not on an excess of carbon dioxide or bacteria or dust of any kind. They admit that the cause of the musty odor in unventilated rooms is unknown.

In addition to reducing potassium permanganate, it has been shown that the breath contains traces of ammonia and traces of hydrochloric acid. These have their origin in decaying teeth and decomposing particles of food or other putrefactive or pathological changes occurring in the upper respiratory passages. The ammonia and hydrochloric acid exist in such small quantities that they have no practical bearing upon the question under consideration.

Weichardt⁴ calls attention to the fact that putrefactive processes go on in the excretory products of the respiratory tract, especially in older persons. He claimed that the bronchial mucus of corpses contains a poison resembling kenotoxin (the toxin of fatigue). When injected into laboratory animals it produced a lowering of temperature, a slowing of respiration, and death. According to Weichardt, fluids condensed from the expired air and then concentrated, when injected into mice, produce like results. This investigator also evaporated some of the condensed moisture from the expired breath and obtained a weighable residue (9 mgms. from 10 c.c.). This he regards as partly organic matter. Also by means of the epiphanin reaction Weichardt considers that he has demonstrated protein-split products in the vitiated air of a room. He concludes that substances having such important biological power should not be longer overlooked. These results lack confirmation, and the methods are open to criticism.

Rosenau and Amoss⁵ demonstrated the presence of minute traces of non-poisonous protein matter in the expired breath through the reaction of anaphylaxis. The first injection into guinea-pigs of the fluid, obtained by condensing the moisture of expiration, is harmless, but the animals become sensitized, so that they react to an injection of human blood-serum after an interval of

³ Published by The Smithsonian Institute, 1895. Contains a summary of the literature to date, with references to authorities.

⁴ *Arch. f. Hyg.*, 1911, 74: 5.

⁵ *J. Med. Research*, 1911, 25: 35.

several weeks. These results lack confirmation; in fact, Weisman and also Winslow obtained negative results. The interesting work of Sewall⁶ bears on this problem, for his experiments indicated that sensitizing substances exist in the expired breath.

There is no evidence whatever that the expired breath contains a poisonous substance. By general consent, the symptoms experienced in a crowded room with vitiated air do not find their explanation by this hypothesis.

Physical Changes in the Air.—Owing to the failure of chemistry to demonstrate the cause of the ill effects produced by a vitiated atmosphere, attention has recently been focused upon the physical changes, such as the increase in temperature, increase in humidity, and the stillness of the air in a poorly ventilated room. Hermann, in 1883, first pointed out that heat and moisture were probably the factors that produced the harmful effects of bad air rather than its chemical composition. Important experiments were carried out in the Institute of Hygiene in Breslau by Heymann, Paul, and Erclentz. Flügge,⁷ who was then the director of the institute, has admirably summarized and interpreted the results as follows:

Paul placed healthy individuals in a cabinet of three cubic meters' capacity, where they were kept for a variable time up to four hours, and until the carbon dioxide had risen to 100 or 150 parts in 10,000—an accumulation of gaseous excretion practically never developed under ordinary conditions. In these experiments no symptoms of illness or discomfort developed so long as the temperature and moisture were kept low. Tests of the psychic fatigue of these individuals by means of the esthesiometer and ergograph, or by means of computations, gave negative results throughout, under similar conditions of temperature and moisture. Tests in a crowded schoolroom were similarly negative. Erclentz made the same observations on diseased persons. Those suffering from emphysema, heart diseases, kidney diseases, etc., with the exception of a few peculiarly susceptible anemic and scrofulous school children, bore the highly vitiated air for hours without any evidence of bodily or mental depression.

The results were very different, however, when the temperature and moisture of the air of the cabinet were allowed to increase. At 80° F. with moderate humidity, or at from 70° to 73.5° F. with high humidity, practically all persons began to show depression, headache, dizziness, or a tendency to nausea. The susceptibility was not alike for all. School children reacted slightly and

⁶ *Am. J. Hyg.*, 1923, 3: 300.

⁷ Flügge, *Ztschr. f. Hyg.*, 1905, 49: 363. Crowder, *Arch. Int. Med.*, 1911, 7: 85. Contains an admirable summary and references to the literature upon the subject. More recent references will be found in Crowder's article, *Arch. Int. Med.*, Oct., 1913, 420. C.-E. A. Winslow and G. T. Palmer, "The Effect upon Appetite of the Chemical Constituents of the Air of Occupied Rooms," *Proc. Soc. Exper. Biol. & Med.*, 1915, 12: 141. L. Hill, M. Flack, J. McIntosh, R. A. Rowlands, and H. B. Walker, "The Influence of the Atmosphere on Our Health and Comfort in Confined and Crowded Spaces," *Smithsonian Miscel. Col.*, 1913, No. 23, 60. Y. Henderson, "The Unknown Factors in the Ill Effects of Bad Ventilation," *Tr. 15th Inter. Cong. on Hyg. and Demog.*, Washington, 1913, 2: 622.

emphysematics slightly, while those with heart troubles were most susceptible. By means of certain objective signs of heat stagnation—the surface temperature of the forehead and the temperature and moisture of the clothed parts of the body—it was determined that subjective symptoms appeared only when the surface temperature reached a certain height. This was, for healthy people, 93° F. to 95° F. on the forehead; for the more susceptible and diseased, 89.5° to 91.5°; and with the moisture of the skin increased by 20 or 30 per cent. Under these conditions the normal dissipation of body heat is interfered with, and it is under these conditions that symptoms appear which are in every way similar to those developed in overfilled and “stuffy” rooms.

Now, when these people in the cabinet suffering from such symptoms were allowed to breathe the fresh outside air through a tube, such air being raised to the temperature and relative humidity of that within, it gave them no relief whatever; nor did the internal air produce any symptoms when breathed through a tube by one outside of the cabinet. But the symptoms of discomfort and illness experienced by the person within could be almost immediately relieved either by drying the air of the cabinet or by cooling it, or by putting it in rapid motion by means of a fan, without any chemical change being made in the air. The effect of these measures is simply by purely mechanical means to enable the body to throw off its heat more rapidly, and thereby all symptoms disappear; heat stagnation is the cause of the discomfort.

From the long series of experiments, carried out with great care as to all the details of observation and control, it is concluded that all of the symptoms arising in the so-called vitiated atmosphere of crowded rooms are dependent on heat stagnation in the body, and that the thermic conditions of the atmosphere, its moisture, and its stillness are responsible for the effects. To change any one of these elements is to change the rapidity of the loss of heat. If the change is such as to increase this loss, comfort is restored. It is also considered proved beyond any reasonable doubt, by their own as well as by previous research, that there is no gaseous excretion into the surrounding air, either from the breath or from other sources, deserving of the name of poison.

Angelici,⁸ working independently at about the same time, concurs in these opinions; and Reichenbach and Heymann⁹ later determined that objective evidence of heat stagnation in the body always precedes the development of subjective symptoms of discomfort under natural conditions, in the same way it does under the artificial conditions of the cabinet. Leonard Hill¹⁰ of England also has confirmed these general results and conclusions.

The New York Commission on Ventilation¹¹ found that the power to do mental or physical work, measured by the quantity and quality of the product

⁸ Quoted by Reichenbach and Heymann, *Ztschr. f. Hyg.*, 1907, 57: 23.

⁹ *Ibid.*

¹⁰ *J. Physiol.*, 1911, 41: 61.

¹¹ *Am. J. Pub. Health*, 1915, 5: 85.

by subjects doing their utmost, is not at all diminished by a room temperature of 86° F., with 80 per cent relative humidity, but the inclination to do physical work is diminished by high temperatures. The only known effect of stagnant air, even when it contains twenty or more parts of carbon dioxid, is slightly to diminish the appetite.

The experiments seems to indicate that overheated rooms are not only uncomfortable, but produce well marked effects upon the heat regulating and circulatory systems of the body and materially reduce the inclination of occupants to do physical work. The most important effects of even a slightly elevated room temperature, such as 75° F., are sufficiently clear and important to warrant careful precautions against overheating. A warm atmosphere, especially if moist and still, causes a rise in bodily temperature and this in turn disturbs metabolism, decreases working power, and causes early onset of fatigue. This is now stated best and most concisely in terms of effective temperature. *It is now clear that most of the symptoms caused by poorly ventilated rooms are due to the prevention of heat loss owing to the physical conditions of the air* (see also pages 816, 830).

Reinspiration of Expired Air.—By this phenomenon is meant the immediate reinspiration of a portion of our expired breath. This occurs quite commonly; in fact, may also be regarded as a normal accompaniment of respiration during the major part of our lives. Lehmann¹² and also Heymann¹³ determine the carbon dioxid of the inspired air, compared this with the carbon dioxid of the surrounding air, and from the difference computed the proportion of the breath which was reinspired. They found this proportion to vary greatly. It was sometimes more than 6 per cent, but dropped to zero in the open air, and in a breeze of three meters per second. Crowder¹⁴ confirms these observations and extends them much further. He shows that under many conditions the air about the face contains much more carbon dioxid than the surrounding air. The path of the expired air may roughly be seen by watching the course of smoke blown from the nostrils. The expired air leaves in a cone-shaped expansion, part of which lies quite close to the body, and then rises slowly by convection currents. Inspiration follows expiration immediately, therefore every chance is offered for some of the expired air to be again drawn in, except when facing a breeze of from two hundred to three hundred feet per minute, or when walking, riding, or fanning. When the back is turned to a lively breeze a little of the expired air is often reinhaled in spite of the current.

The position of the head influences the amount of air rebreathed. Thus Crowder has shown that while sitting upright the reinspiration was 2.3 per cent; lying down with a pillow tilting the head forward, 1.3 per cent; lying flat, head thrown back, none at all. In the ordinary position in bed, with the head on the side and flexed, with pockets or dead spaces for the air to stag-

¹² *Arch. f. Hyg.*, 1899, 34: 315.

¹³ *Ztschr. f. Hyg.*, 1905, 49: 388.

¹⁴ *Arch. Int. Med.*, 1913, 12: 420.

nate, there will be a greater retention of expired air. This occurs especially when the head sinks into a soft pillow.

It may be concluded that when one lives indoors and remains quiet he will immediately rebreathe from 1 to 2 per cent of his own expired air; in bed it will be more, from 1 to 5 per cent, and even 10 to 18 per cent, depending on the position in which he lies. Nor does sleeping in the open insure "pure" air, for breathing, especially when one buries his head between pillows and bed-clothes for the sake of warmth. On the other hand, it is plain that "a little extension of the dead space beyond the tip of the nose is of no consequence."

Summary.—It is now perfectly plain that the ill effects resulting from a vitiated atmosphere are not due to an increase of carbon dioxide nor to a diminution in oxygen. Upon this point all are agreed. The general consensus of opinion also excludes poisonous bodies in the expired breath as a factor.

Sanitarians are satisfied, with the evidence presented, that most of the discomfort is due to physical changes only. If a normal heat interchange can be maintained between the body and the air the symptoms which are commonly attributed to poor ventilation do not develop. According to this view the vital element of the ventilation problem becomes that of regulating the temperature, moisture, and motion of the air. When the air is still we are surrounded by an "aërial envelope" with a temperature and moisture resembling the open air on a hot and humid day. The symptoms caused by crowd poisoning, such as oppression, malaise, headache, vertigo, nausea, vomiting, and even collapse, indeed resemble those of heat exhaustion.

Metabolism is reflexly retarded by a warm aërial envelope, and stimulated by cool moving air, the consumption of oxygen by the tissues and the production of carbon dioxide by them being much less in warm air than in cold air.

Even those who look upon the physical changes in the air as the sole cause of the discomfort rather than the possibility of chemical changes admit that a certain amount of fresh air must be supplied. Flügge himself urges that life in the open should be more and more resorted to, but he would have the motive correctly understood, not that the chemical condition of inside air is harmful, but that it is the overheating of rooms that causes disturbances of health. Flügge states that one should go into the open not because he may breathe chemically purer air, but because its almost constant motion carries away the body heat and causes a beneficial stimulation of the skin and reflexly brings about a heightened cell activity that aids in the development of sturdy health. The chemistry of air and "crowd poisons" have little or no part to play in the explanation of outdoor benefits or of indoor discomforts. These are both dependent upon physical conditions, and their explanation rests with the physics of heat interchange between the body and its surrounding medium. The oft-quoted and admirable statement of F. S. Lee is that "the physiological problems of ordinary ventilation cease to be chemical and pulmonary and have become physical and cutaneous."

There is some danger in regarding the ill effects of poor ventilation as due

to thermal and other physical factors alone. According to this theory it is only necessary to keep the temperature and moisture down and keep the air in motion; a closed office with an electric fan would take the place of any system of ventilation. There is already a clamor against the laws requiring fresh air in workrooms, which is a natural corollary of Flügge's views. If re-breathing the same air is not hurtful, the ventilation of living rooms may be greatly simplified by simply keeping the physical conditions of the air within the limits of comfort. Furthermore, a great economy would be effected. It is, however, not scientific to insist that the chemical changes in a vitiated atmosphere may be disregarded, because we cannot at present demonstrate immediate relationship between cause and effect; neither is it safe to deny dogmatically the existence of injurious substances in a vitiated atmosphere simply because in the present state of our knowledge chemistry has failed to demonstrate them, and because most of the symptoms may be explained upon disturbances of thermic interchange.¹⁵ The effect of odors in a vitiated atmosphere is another problem little understood.

Furthermore, most of the observations have been under laboratory conditions and with short exposures; it is very probable that a decrease in mental and physical efficiency would result from a prolonged exposure to a vitiated atmosphere, even though it were kept dry and cool. The improvement in appetite, nerve vigor, blood quality, and muscular tone which follows open air life, even in the rich and well-fed, shows the paramount importance of fresh air, sunshine and exercise.

¹⁵ See also Sewall, *Interstate M. J.*, 1916, 23: 1.

CHAPTER VI

VENTILATION AND HEATING

VENTILATION

The problem of ventilation is apparently a very simple one; all that is required is to furnish a never-ending stream of fresh air from the inexhaustible supply without to replace that which is constantly being vitiated. To do this under the artificial conditions of house and factory life is often extremely difficult, and under certain circumstances practically impossible. Further, the problem of ventilation must take into account not only the quantity of air, but its physical condition, in order that the human machine may operate at the highest level of health and efficiency.

Ventilation must serve a number of purposes and comply with a number of conditions before it can be considered satisfactory: (1) it must bring pure air from without in order to dilute and remove the products of respiration, as well as other sources of vitiation; (2) it must maintain the air within the room at a proper temperature and humidity, and, further, must keep the air of the room in gentle and continuous motion; (3) it must remove the gases, odors, bacteria, dust, and other substances that contaminate the air of inclosed spaces; (4) it must dilute and remove the impurities produced by the burning of gas, candles, lamps, and other sources.

The problem of ventilation is physical rather than chemical, cutaneous rather than respiratory.

The object of ventilation is comfort and health. The purpose of ventilation is not to bring outdoor conditions indoors; the art of ventilation consists in adapting indoor conditions to indoor life. Indoor life is necessary in order to perform the delicate manipulations which cannot, as a rule, be effectively conducted outdoors. Indoor life, then, involves quiet and protection from sudden changes or extremes.

It is a simple mechanical problem to condition the air of an apartment. The ventilating engineer finds no difficulty in regulating the temperature and humidity within narrow limits, and in furnishing definite quantities of fresh, moving air. To maintain these conditions, however, the doors and windows must be kept shut, except with modern equipment. Herein arises the first difficulty between the theory and the practice of ventilation, for it is plain that the simplest and often the best way to ventilate a room is through open windows. The second difficulty arises from the fact that the conditions within and without the room to be ventilated are not constant. The principal factors here concerned are the force and direction of the wind, changes of outdoor

temperature, and, to a less degree, movements within the room. It is, therefore, much easier to maintain constant air conditions in a sub-basement than in a room exposed to wind and weather. Air conditioning is now an established engineering science, and the engineer is prepared to supply any kind of air that is desired.

The efficiency of any system of ventilation must be measured by the results obtained at the breathing zone. It matters little what the composition or the condition of the air is near the ceiling, provided the heated, moistened, and vitiated aerial blanket which surrounds us is constantly removed and replaced with a fresh supply properly conditioned.

Ventilation is far from satisfactory if the air brought into the room is smoky, dusty, or bacteria-laden, or if it is contaminated with gases or odors from cellars or surroundings. Attention should, therefore, be given to the sources of the air, and it is always an advantage to wash or filter it. There is a practical limit to the amount of fresh air that may profitably be forced into a room, especially warmed air in the winter time. Ventilation and heating naturally go hand in hand.

The belief is growing that it is not dangerous to rebreathe air, and the view is spreading that the hygienic value of ventilation for the purpose of maintaining a chemically pure atmosphere in dwellings, schools, and hospitals is not so great as is commonly supposed. According to this view it is more important to ventilate in the interest of the heat economy of the body, by the establishment of a suitable temperature and air movement, and by the regulation of the humidity in the atmosphere. The established facts, that the principal causes of the ill effects of vitiated air are due more to the heat and humidity and stillness of the air than to changes in its chemical composition, have led some hygienists to recommend rebreathing the air, provided the physical conditions are kept favorable.

Satisfactory ventilation should not only take into account the physical conditions of the air, but also demands a generous supply of fresh air in order to keep the chemical composition within reasonably normal limits. Clean air in motion and of proper temperature and humidity is necessary to indoor comfort.

The rigor of a cold climate makes of its inhabitants a house-dwelling race. Under these conditions houses are commonly overheated: if not by fire and steam, then by the heat of the inhabitants' bodies. When people do this they complain of poor ventilation, regardless of whether the air supply is large or small.

The problem of ventilation is immediately related to clothing, bathing, diet, exercise and other factors in personal hygiene that stimulate the vasomotor mechanism and make it vigorous and effective. In other words, the problem of ventilation concerns itself chiefly in conditioning the air so as to favor the heat regulating mechanism of the body.

Dwelling houses are usually constructed with little regard for ventilation. It is desirable that adequate provision should be made for the ventilation of

every house that is built. This requires just as much care and forethought as the system of heating the house, or furnishing it with water, gas, electricity, plumbing for the disposal of wastes, and other household conveniences. Whatever system of ventilation may be adopted, it is wise to flush rooms frequently with fresh air and flood them with sunshine. This helps to blow out the accumulated dust and bacteria, to oxidize organic matter that collects as a film on all surfaces, to diminish odors, and generally to purify the apartment. Window ventilation is simplest and often the best.

In all systems of ventilation the factor of faithful and intelligent operation is essential to success. No method is fool proof and no method will work by itself. Even window ventilation requires watchful attention of those in charge of individual rooms. "Constant vigilance is the price of pleasant and wholesome air conditions" (Winslow).

Air Washing.¹—The process of air washing consists of passing the air horizontally through a chamber in which water is falling in drops as rain, or into which it is sprayed. The sprays are obtained by forcing the water out of perforated pipes or through nozzles placed across ducts. When the sprays intersect they are said to form a "water curtain." The object is to bring the air and water into intimate contact. Besides the washing chamber there are heating or tempering coils in the ducts, or in a separate chamber, and devices for controlling the temperature. The water used for washing is circulated by means of a pump so that it may be used over and over in the spray chamber for a considerable time.

The New York State Commission on Ventilation found that air washing does not completely remove body odors.

Washing takes out many of the impurities in the air, as bacteria, molds, dust, epithelial scales, particles of various descriptions, also some odors and gases, but not carbon dioxide. Washing imitates nature's process through a rain shower. If desired, the water may be cooled in the summer time so as to influence the temperature of the air. Humidity can be controlled by air washing so as to add any given amount of moisture to the air. Several forms of air washers are on the market, essentially similar in principles, but differing in details of construction.

Air Filters.—Formerly the air was drawn through bags and other filtering material to take out dust. These offer resistance and have been superseded by better types of filter, such as the Midwest and Reed, in which the filtering agents are screenlike substances, or small tubes jumbled together and coated with a sticky material. The air takes a tortuous path through this device and the dust particles are caught on the sticky surfaces. Most of the dust and soot are removed, and they have the advantage of being cheaper than air washers to install and operate. Their chief disadvantage is that they do not add moisture to the air.

¹ For the mechanical equipment of buildings, for principles and methods of heating and ventilation, see Harding and Willard, *The Mechanical Equipment of Buildings*, Vol. I, J. Wiley, N. Y.

Recirculation.—The cost of heating large volumes of cold air has naturally stood in the way of efficient ventilation of schools and factories during the cold weather. Another question has been the low indoor relative humidity produced by heating outdoor air to a comfortable room temperature. Washing and recirculating the air overcomes both of these objections because it furnishes an ample supply of conditioned air in motion. The method has attracted favorable attention. Naturally there must be a limit to the continued use of the same air, but ordinary leakage and the use of a certain percentage of outside air prevent the concentration of any substances not removed by the washer.

The only advantage of washing and recirculating the air lies in the great saving of fuel in cold weather. At the gymnasium of the International Y. M. C. A. College at Springfield, it was estimated that a saving of from 40 to 50 per cent of coal resulted from recirculating the air. The success with the method at Springfield was probably due to the enormous air supply of the gymnasium. The method has many objections for schools and workshops.

Recirculated air is not equal to outside air washed. The great objection is that washing the air does not remove odors; it is therefore not very practical. There are factors still imperfectly understood. Recirculation may be subject to abuse, and if used must be carefully watched.

Vitiation by Respiration.—An adult individual at rest breathes at the rate of about seventeen respirations a minute. At each respiration about five hundred cubic centimeters (30.5 cu. in.) of air pass in and out of his lungs. The air in the lungs loses about 4 per cent of oxygen and gains about 4 per cent carbon dioxid. The nitrogen remains unchanged. In addition the expired air is raised in temperature to nearly that of the blood, 98.4° F.; it is also practically saturated with aqueous vapor.

The amount of carbon dioxid which is given off by an adult male person at rest can be calculated from the above figures, and will be found to be 0.71 cubic foot in one hour. Thus:

$$\frac{17 \times 30 \times 60}{1728} = 17.7 \text{ cubic feet breathed per hour}$$

$$4 \text{ per cent of } 17.7 = 0.71 \text{ cubic foot per hour of CO}_2$$

From actual experiment it has been determined that an average adult gives off 0.9 of a cubic foot of carbon dioxid during gentle exertion, and possibly as much as 1.8 during hard work. The adult female gives off about one-fifth less under similar circumstances, and an infant is said to give off about 0.5 cubic foot of carbon dioxid per hour. In a mixed assembly at rest, including male and female adults and children, the carbon dioxid given off per head is, therefore, taken as 0.6 of a cubic foot.

Vital Capacity of the Lungs.—The volume of air inspired and expired depends on the rate and extent of the respiratory movement, but in an adult man of average size and vigor about five hundred cubic centimeters of air are

inspired and expired during quiet breathing. This volume of air is known as the *tidal air*, and since the total volume of air in the lungs is about 3,500 cubic centimeters, it is evident that in normal breathing a large amount of air—3,000 cubic centimeters—remain in the lungs at the end of expiration. The air which remains behind is known as *stationary air*.

By forced expiration about half of the stationary air, *i.e.*, 1,500 cubic centimeters, can be expired, and this portion of the stationary air is known as the *supplemental or reserve air*, while the final 1,500 cubic centimeters, which no effort can expel, is known as the *residual air*. The total of 3,500 cubic centimeters of air in the chest, then, at the end of ordinary inspiration is made up as follows:

Tidal air	500 c.c.
Stationary air { residual air.....	1,500 c.c.
{ supplemental or reserve.....	1,500 c.c.
	<hr/>
	3,500 c.c.

When, however, inspiration is forced, another 1,500 cubic centimeters of air, known as *complemental air*, can be inspired, making altogether 5,000 cubic centimeters.

The total amount of air (complemental, tidal, supplemental) which can be inspired after forced expiration is known as the "respiratory capacity" or "vital capacity" or "extreme differential capacity," and the amount varies considerably according to height, weight, vigor, age, etc.

Peabody and Wentworth² furnish the following standards for the vital capacity of the lungs of normal men: for men over 6 feet tall, 5,100 cubic centimeters; between 5 feet 8½ inches and 6 feet, 4,800 cubic centimeters; between 5 feet 3 inches and 5 feet 8½ inches, 4,000 cubic centimeters. In women of the same height, it is about one-fifth less.

Vital capacity is a good index of vigor, endurance and reserve power.

Dead-Space Air.—With each breath, we take back into the lungs the air contained in the nose and larger bronchi—the "dead-space" air. This dead-space air constitutes about one-third of the whole volume of quiet inspiration, and not less than one-tenth of deep breathing. To all intents and purposes it is expired air which is constantly reinspired. Rebreathing of the ordinary dead-space air is a normal and conservative process; it prevents pure cold air from entering the lungs and reducing the carbon dioxid below the amount required for stimulating the respiratory center; it makes of breathing a regular and continuous rather than an irregular and interrupted function. Douglas and Haldane have shown that the volume of the dead space, instead of being a fixed quantity, is automatically altered so as to give greater or less resistance to the air-flow to and from the lungs with changing exertion. They

² *Arch. Int. Med.*, Sept., 1917, pp. 433, 443.

go so far as to state that rather marked variations may occur; and, while the mechanism is not fully understood, they think the regulation is as perfect as is that of the vasomotor mechanism for controlling the blood flow.

Factor of Safety.—Bernard and Mantoux³ have shown that the lungs are capable of performing the respiratory function even when the capacity is reduced to one-sixth of the normal. Furthermore, we should remember that the possibility of increase in the depth of inspiration is 400 to 500 per cent, and that by changing the rate and the completeness of expiration the alveolar ventilation may be increased considerably more than 1,000 per cent. From this great margin of safety it is easy to understand why a slight increase of carbon dioxide in the inspired air falls far below the limits of our conscious effort. From the experiments of Haldane and Priestly⁴ an actual increase of 100 per cent in the pulmonary ventilation passes almost unnoticed. The factor of safety which Meltzer⁵ has so well described as belonging to all well understood physiologic processes, is here a very generous one.

The Amount of Air Required.—A certain amount of fresh air is necessary for good ventilation. This amount is determined from the number of people, gas jets, electric fixtures and other sources of heat; also from the construction and orientation of the building; that is, the air requirements are based on thermal rather than gaseous considerations. The standard figure now used by ventilating engineers is 1,800 cubic feet per person per hour. Formerly, this factor was determined from the amount of carbon dioxide taken as an index of the impurities from respiration and combustion. The factor ascertained either by direct observation or from physiological data was 2,000 cubic feet. There is thus a happy coincidence between the old-fashioned chemical method and the modern thermal method of determining the amount of fresh air needed for good ventilation. The volume of air in itself is not as important a factor in ventilation as the necessity for the maintenance of temperature, moisture and air movement to facilitate evaporation and the elimination of body heat. It is interesting to note that it requires just about as much air to regulate heat interchange as to dilute the carbon dioxide to permissible limits. The amounts necessary to remove odors and dust are about the same as that required to keep the carbon dioxide within reasonable limits. The amount of air needed in good ventilation, therefore, remains about the same as formerly, but our reasons for supplying it have changed.

The amount of air required varies greatly with circumstances. Theaters holding 2,000 to 5,000 persons may require as much as 2,400 cubic feet per person per hour, especially in the summer time. In certain special hospital requirements, the figures range as high as 12,000 cubic feet. The standard figure of 1,800 cubic feet is the minimum, for it is difficult to obtain adequate distribution with lower rates, which are also apt to give stagnant pockets.

³ *J. de Physiol. Exper.*, 1913, 15: 16 (Ed. Abstr. in *J. Am. M. Ass.*, 1913, 60: 1794).

⁴ *J. Physiol.*, 1905, 32: 225.

⁵ "Factors of Safety in Animal Structure and Animal Economy," *Harvey Lectures*, New York, 1907-8, p. 139.

The avoidance of drafts is imperative in any good system of ventilation. The best way to avoid drafts is to distribute the air very uniformly and to regulate the temperature of the incoming air at about the temperature desired. Drafts are usually felt on account of poor distribution and temperature differences (page 248).

The Size and Shape of the Room.—These are exceedingly important factors in any system of ventilation. It at once becomes evident that a man in a diving suit with a good circulation of fresh air is better off than occupants of a spacious but poorly ventilated compartment in which the air has become vitiated through long occupancy. The air in a small cabin on a steamship may be infinitely better than the air in a large room of a country home. A rathskeller in the sub-basement may, with a modern system of ventilation, have much better air than that found in a department store with acres of floor space and high ceilings. In other words, a small space is sufficient if properly ventilated; a large space inadequate if improperly ventilated.

	Minimum Space per Head, Cubic Feet	Authority
Common lodging houses (sleeping rooms)	300	Local Government Board (Model By-Laws)
Registered lodging houses—		
Rooms occupied by day and night.	400	Ditto
Rooms occupied by night only.....	300	Ditto
Non-textile workrooms.....	250	Factory Act, 1901
Non-textile workrooms during over-time	400	
Underground bakehouses.....	500	Order under Factory Act, 1901
Above-ground bakehouses where night work is carried on by artificial light other than electric light between 9 P. M. and 6 A. M.....	400	Ditto
Army barracks.....	600	British Army Regulations
Army hospital wards.....	1,200	Ditto
Public elementary schools.....	80	Educational Department
London County Council Schools.....	130	London County Council
Canal boats (persons over 12 years)	60	Local Government Board,
Canal boats (persons under 12 years)	40	Regulations under the Canal Boat Act, 1877
Seamen's cabins.....	72	Merchant Shipping Act
Cows in cowsheds.....	800	Local Government Board, Model Regulations under the Dairies, Cowsheds, and Milkshops Order

The size of rooms for dwellings and workshops is somewhat of an economic question, but they should be large enough to allow the air to be replaced two or three times an hour without causing perceptible drafts. The minimal space, in accordance with this standard, is about one-third the quantity of air required per hour; that is, from seven hundred to one thousand cubic feet per person. The amount of space naturally varies with dwellings, factories, schools, theaters, prisons, hospitals; also with the length of time the room is occupied

and the nature of the work there carried on. Thus, in hospitals where ordinary cases are cared for, from 1,800 to 2,000 cubic feet of air is desirable for each patient, while no less than 2,500 cubic feet should be allowed for each fever patient. Soldiers in barracks are allowed six hundred cubic feet per head, and the limit for lodging houses is usually fixed at from three hundred to five hundred cubic feet. The United States Immigration Law requires five hundred cubic feet per head in the steerage. In figuring the amount of air space in a room allowance should be made for furniture, projecting surfaces, and other objects which diminish the available space. The preceding table from Parkes and Kenwood shows the attempts made by Great Britain to fix the minimum space allowed per head by legislation. If the space for occupancy is less than five hundred cubic feet, forced or mechanical ventilation is necessary.

A little consideration, however, will show that such regulation of space is by itself of little value. Unless there be movement of air, space alone is futile. However large the space may be, the air will become impure unless fresh air circulates through it, and however small the space the air may be kept pure by sufficient circulation.

As the result of many analyses that have been made by Haldane and Osborne, they found that the carbon dioxid bears no relation to the amount of air space under practical conditions. In fact, the most highly vitiated air found was in a room with an air space of about 10,000 cubic feet per person.

It is not alone the air space but the shape of the room that influences ventilation. It is a mistake to suppose that a lofty room is, therefore, an airy room, for a stratum of warm vitiated air soon occupies the upper portion of such a space, and, so far as good air is concerned, has the effect of lowering the effective height of the ceiling to the top of the door or nearest outlet. Anyone may convince himself of this fact by getting up on a stepladder in a room with a high ceiling, improperly ventilated, and occupied for some hours. The upper stratum of air in such rooms is frequently stifling. Ordinarily twelve feet is high enough for the ceiling of schoolrooms, museums, hospitals, etc., and nine feet for the rooms of private dwelling houses. Where there is little or no movement of the air it soon becomes offensive, no matter what the height of the ceiling.

Floor space is more important than height. The necessity for an abundant floor space is shown by the fact that a small inclosure with four high walls and without a roof, if crowded, speedily becomes oppressive. In fact, the four walls are not necessary to demonstrate this, for so-called "crowd poisoning" or heat stagnation in the open air upon a still, warm day is a common experience. According to Harrington, when the allowance is only 500 cubic feet per inhabitant, the floor space should be forty-two square feet ($8\frac{1}{2} \times 5\frac{1}{2}$). In the English barracks the soldiers are allowed fifty square feet of floor space. For schoolrooms the British Educational Code requires 120 cubic feet per child in average attendance and a floor space of ten square feet.

Inlets and Outlets.—Whether a room is to be ventilated by natural or mechanical means, proper inlets for the fresh air and outlets for the vitiated

air must be provided. No general statement as to the best size and position of these openings will apply under all circumstances.

Knowing the velocity of the incoming air, the area of the inlets may be proportioned so as to permit the movement of the necessary amount of air. The size of the openings under specified conditions is, therefore, a matter of simple arithmetic. In measuring the effective area of inlet and outlet tubes allowance must be made for friction and for the guards or fretwork which protect the openings. These often diminish the effective area about one-half.

In order to provide the air supply of thirty cubic feet per minute, the inlet registers in any room should have a total area equal to 0.1 square foot per capita. The air ducts must be so constructed as to lead the air evenly to the different parts of the building. Each room should be controlled independently by individual ducts and dampers.

It is usually better to admit the incoming air into a large apartment through a number of openings rather than through one large one; the same holds true of outlets. Outlets should be about the same size as inlets and should be placed with reference to them.

All air ducts tend to become soiled with dust and soot and should, therefore, be guarded with wire gratings, or other protecting devices, and they should also be cleaned periodically; further, it should be borne in mind that ventilating ducts are favorable highways for mice, roaches, and vermin. Inlets opening upon the floor are objectionable, as they collect unusual amounts of dirt and dust, which are then blown into the room.

Whether the air is to be admitted near the floor and taken out near the ceiling or *vice versa* is a question much discussed among ventilating engineers. Various possibilities are shown in the diagram, Fig. 76. The natural course of the warmed vitiated air is upward, and it would seem that the upward system has advantages over the downward system. However, a little study will soon convince one that if the incoming air is warm it will rise at once, and the maximum efficiency will be lost at the breathing line, which, after all, is the essential stratum of air in the room. A good arrangement under certain circumstances is to have the inlet above and the outlet below—both upon the same side of an inner wall. For crowded spaces the best system is doubtless the upward plan, which takes advantage of the natural currents. When this plan is used, the air should be admitted to various parts of the room; in theaters, under each seat. The present tendency in theaters and large auditoriums is to use the downward system of ventilation in winter and the upward in summer, by merely reversing the electrical connections on the fan motors.

Outlet ventilation may be arranged by placing a bell cover or glass globe over the gas lights and conveying the heated air thence to the outer air by means of ascending tubes. This not only removes the products of combustion, but, if the outlet tubes have a sufficient area, affords a very good system of ventilation. An automatic system of taking the air out of a room may also be provided by placing a shaft either around the chimney flue or against one side of it. The column of heated air in the ventilating duct will rise and

draw the vitiated air out of the room with which it is connected. The same may be accomplished by placing a steam jet or a gas burner within the ventilating duct to create a draft.

Ventilating ducts usually extend up the walls of the building through the roof, and should be in as direct a line as practicable. The openings upon the roof may be protected by an umbrella-like covering against rain, or they may be cowled to prevent down drafts. It appears that none of the exhaust cowls cause a more rapid current of air than prevails in an open pipe under similar circumstances.

Too little attention has been paid in the past to the cleanliness of the air supplied to our buildings. Fresh air inlets are often located with the grossest disregard for the quality of the incoming air. It is not uncommon to see them placed on the sidewalk level; or facing a vacant piece of ground that is swept by clouds of dust; or where smoke, the spent gases from automobiles, or objectionable odors may be taken in.

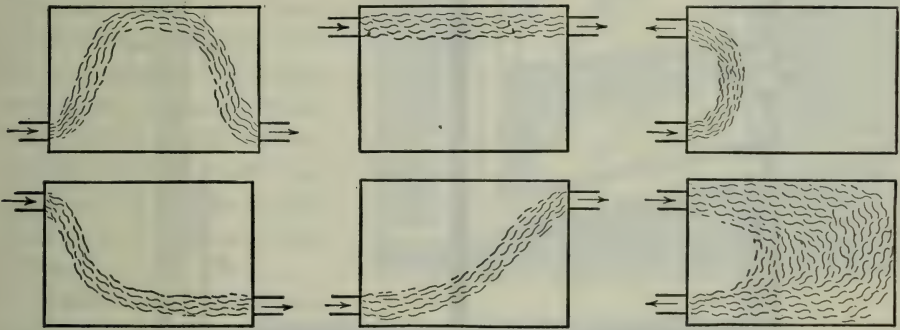


FIG. 76.—THE POSITION OF INLETS AND OUTLETS, AND THEIR RELATION TO THE AIR CURRENTS IN A ROOM.

Crowded buildings and dusty city streets will often render it impossible to secure clean air from the outside atmosphere without resorting to artificial purification.

External Ventilation.—Model city planning should provide streets of sufficient width, and should regulate the height of buildings and also limit the extent upon which the land may be built, so as to allow a free circulation of air about all structures and admit a flood of sunshine for at least a few hours during the day. Some of our metropolitan streets resemble canyons rather than city thoroughfares. Crowded tenements, facing upon narrow streets with shafts for courts and backing almost directly upon the houses in the rear, and further surrounded by tall buildings which prevent the free movements of the outer air, and shut out the sunshine, should be prohibited whether used as dwellings or workshops. In such places the ground stays moist, the air becomes stagnant, natural ventilation is greatly retarded, and the conditions upon a hot, still, moist day in summer become almost intolerable.

Generous parks, which are the lungs of a great city, should be scattered throughout the residential and business sections; playgrounds, boulevards, and

small open areas treated as parkings not only beautify but help to ventilate a city and add to the comfort, happiness, and health of its inhabitants.

Natural Ventilation.—Natural ventilation depends upon openings, such as doors and windows, also upon the air that comes through the pores of plaster, brick, and stone and through floors and ceilings and through the cracks and crevices about window frames, etc.

Natural ventilation depends mainly upon three principal factors: (1) perflation and aspiration; (2) gravity or thermal circulation; (3) diffusion of

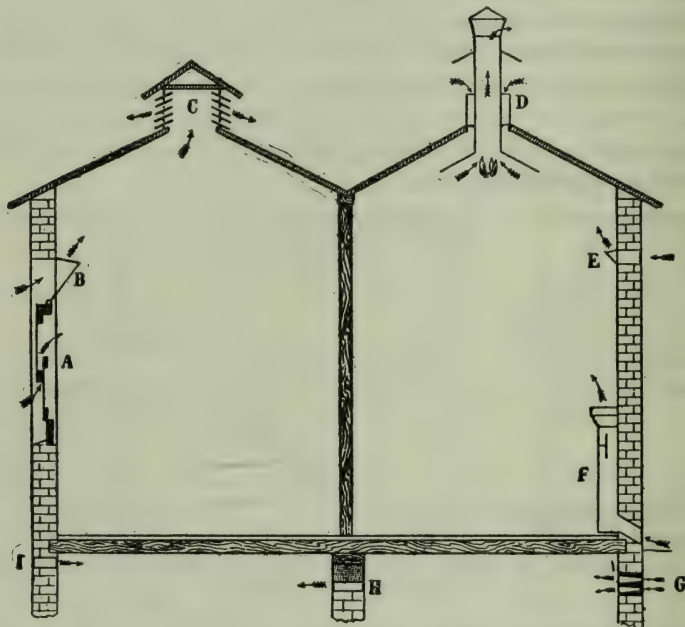


FIG. 77.—DIAGRAMMATIC SKETCH OF VARIOUS PROVISIONS FOR VENTILATION.

A, Sash window with Hinckes-Bird's arrangement. B, Hopper sash-light falling inwards. C, Louvered outlets. D, McKinnell's ventilator. E, Sheringham's valve. F, Tobin's tube (showing valve open). G, Ellison's conical bricks. H and I, Grid ventilators below floor joists. (From *Hygiene and Public Health*, by L. C. Parkes and H. R. Kenwood, London, H. K. Lewis, Philadelphia, Blakiston, 1911.)

gases. These factors constantly operate, whether in the presence or absence of any mechanical system. In fact, most schemes for mechanical ventilation are simply an application of these natural forces.

Perflation is simply the blowing of the air into the room as a result of the movement of natural air currents. Aspiration is the sucking action of the wind which draws air out of a space that it is blowing across. Thus, a wind blowing across an open tube carries along with it some of the air in the upper part of that tube. This causes an upward movement of the air in the tube. The same phenomenon takes place when wind blows by a window. The aspirating action of air is well demonstrated in the construction of an ordinary atomizer.

The air is kept in almost constant motion through changes in temperature. Warm air expands, is therefore lighter, and rises. This is a familiar phenomenon in the hot-air balloon. Thermal circulation, though often imperceptible, is constantly in operation, especially in occupied rooms. Even in calm weather there is considerable ventilation owing to differences in temperature, and hence differences in pressure between the air of the room and the outside.

Air leakage and infiltration are important factors and can be estimated with reasonable accuracy. The orientation of the building and the direction and velocity of the prevailing wind must be known.

More air than is commonly supposed enters or leaves a room through the cracks about doors and windows and other crevices. From the standpoint of natural ventilation it is, therefore, not advisable to have windows fit too snugly. The use of weather-strips, tongue and grooved metal strips, and similar devices to keep out the cold air saves coal bills, but is a considerable hindrance to natural ventilation.

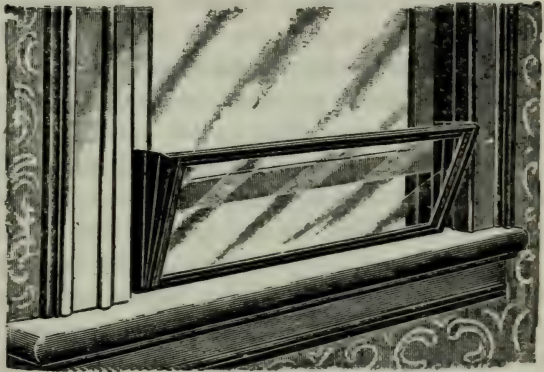


FIG. 78.—WINDOW VENTILATOR.

Under certain conditions very considerable amounts of air pass through the building materials used in the construction of walls, floors, and ceilings. Ordinary mortar is most permeable, then comes brick, then sandstone, next plaster-of-Paris, while enamel and tile are impervious. Under a pressure of 108 millimeters of water the following amounts of air pass in one hour through one square meter of:

Mortar	3,264 liters
Plaster-of-Paris	146 liters
Bricks	312 to 1,396 liters
Sandstone	426 to 496 liters

A pressure of 108 millimeters of water is equivalent to the pressure of a strong wind. The amount of air that will pass through porous materials varies, of course, with the temperature, moisture, and other factors.

Märker and Schultze, in their researches on the spontaneous ventilation of stables, found that the following interchange of air occurred per hour over one square yard of free wall at 9.5° F. difference of temperature:

With walls of sandstone	4.7 cubic feet
Quarried limestone	6.5 cubic feet
Brick	7.9 cubic feet
Tufaceous limestone	10.1 cubic feet
Mud	14.4 cubic feet

It is possible to force sufficient air through an ordinary brick to deflect the flame of a candle on the other side. This demonstration is usually accomplished by coating the edges and exposed portions of the brick with sealing-wax and arranging glass funnels on either side. Air forced with a bellows through one funnel may be measured either as to its amount or velocity as it comes out of the opposed funnel.

Natural ventilation is better in winter than in summer, owing to greater differences in temperature. It may be almost *nil* on a hot calm day. Too much moisture in the air of a room settles upon the surfaces and thus stops the pores of building materials, and also prevents the escape of carbon dioxide. Rain has a similar effect on the outside. An ordinary brick will soak up a pint of water. Ventilation through the walls is also hindered by oil and enamel paints and by wall paper. Outside obstacles, such as excessive foliage and narrow streets, are also considerable factors.

Natural ventilation may be greatly favored by simple devices. This may be demonstrated by placing a lighted candle in a bottle with a narrow neck. The flame soon dies out, but by placing a partition in the neck of the bottle, so that the products of combustion will escape on one side and the fresh air enter upon the other, natural ventilation proceeds so that the candle remains lighted. There are numerous simple devices that may be placed at the top or bottom of windows which favor the entrance of fresh air or the exit of vitiated air. An arrangement shown in Fig. 78 gives very satisfactory results. One of the upper window panes may be valved or fitted with a fan to permit the entrance of fresh air or the exit of vitiated air. A somewhat similar arrangement used at Fairfield, Connecticut, is shown in Fig. 79. Openings in ceilings, ridged ventilators, Sheringham's valves, Ellison's bricks, Tobin's tubes, and Stevens' drawer ventilator are all useful accessory devices to aid natural ventilation.

Ellison's bricks are bricks with conical perforations, the widened end of the conical opening debouching on the interior of the wall. The holes through the bricks are about two-tenths of an inch in diameter externally and one and a quarter inches internally.

Tobin's tube consists of a large upright tube, about five or six feet high, which conducts outside air into the room through the wall.

The Sheringham valve is a small vertical flap door in the wall near the ceiling, balanced by a counterpoise, and hinged so as to fall forward toward the room; it is cased in at the sides and front, so that the current can only pass upward.

Stevens' drawer ventilator is like a drawer lacking its back. It is made to

fit into a hole in the wall in such a way that when the drawer is shut the hole is air-tight, and when the drawer is open air can enter.

Hinckes-Bird ventilator is made of the opening between two ordinary window sashes when the lower is raised, and the lower opening closed by means of a specially high sill or by an accurately fitting block of wood.

These and various devices are useful aids to ventilation. They should be protected with valves so that they may be regulated. Sometimes it is advisable to provide gauze or cotton filters to keep out the dust.

Natural ventilation is greatly aided by means of warming the air in the outlet duct. The best example of this is the open fireplace, or other devices for warming the air in outlet tubes already referred to.

In dwelling houses, where there is no overcrowding, natural ventilation through cracks and crevices, through walls and windows, is ordinarily sufficient for practical purposes.

Wherever possible, open windows are the best and simplest means of ventilating a room. Any system of mechanical ventilation at best is costly and frequently unsatisfactory. Open windows are cheap and adequate, but the limitations and disadvantages of natural ventilation are obvious, and, therefore, we are frequently required to resort to mechanical means. The determining factor in dwellings, hotels, factories, etc., is the number of people: few people, natural ventilation; crowds, mechanical ventilation.

The Fairfield System.—An ingenious and simple system of modified window ventilation, devised by S. H. Wheeler of Bridgeport, Connecticut, and first installed at the Sherman School, Fairfield, Connecticut, eliminates the disadvantages of the ordinary natural ventilation while retaining some of its peculiar advantages. According to this plan, fresh air is admitted through the windows, but direct drafts are prevented by placing slanting window boards on the sashes so that the incoming air is deflected upward and mixed with the general good air of the room. This incoming current is furthermore tempered by placing the radiators used for direct heating under the windows and by making these radiators large enough to extend across the entire width of all the windows. Finally, a duct is provided for the egress of warm, vitiated air passing from near the

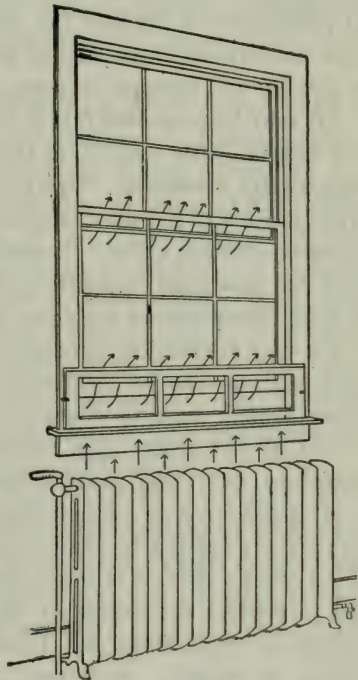


FIG. 79.—FAIRFIELD SYSTEM OF WINDOW VENTILATION.

ceiling of each room to the outer air, the upward current in this duct being maintained by the temperature difference between the outdoor and indoor air.

In industrial establishments where crowding is not great the same general principle has been applied by providing special air inlets to individual rooms with heating coils placed directly in front of them.

This system is very uneconomical because most of the heat is carried up the duct to the outside, without circulating around the breathing zone.

The King System.—An interesting system of ventilation, known as the King system, is in use in cow stables, which secures much better air conditions than those to which human beings are frequently exposed. Louvered openings at the ridgepole furnish an exit for the warm, vitiated air, while fresh air is admitted through ducts in the walls. These ducts open to the outside at the bottom of the wall and to the inside of the stable four or five feet above the floor, the inflowing current of air being induced by the difference in temperature between the stable and the outer air.

Mechanical Ventilation.—All “artificial” systems of ventilation depend upon one of three methods: (1) plenum system, which consists in the mechanical propulsion of air into the room; (2) the vacuum system, which consists of the mechanical extraction of the air out of the room; (3) a combination of the plenum and vacuum systems.

Air may be propelled into a room either by means of a warming apparatus or by mechanically propelling the air by means of rotary fans. The former is mechanically inefficient. Every heating apparatus is secondarily a ventilating device, especially hot-air furnaces, and the direct-indirect systems in use with hot-water or steam pipes. Stoves, open fireplaces, and similar heating arrangements are also good ventilating devices in that, if well constructed, they take out large quantities of air from the room.

For the mechanical propulsion of air either fans or “blowers” are used. These may be run by electricity, gas, or steam power. The air is forcibly driven through ducts to where it is wanted. Without this system of mechanical ventilation the great office buildings, basement restaurants, large passenger steamships, and other modern structures would not be habitable.

If dependence is placed solely upon drawing the vitiated air out of a room we are leaving to chance where the fresh air is coming from to replace it. In other words, it is impossible when the so-called vacuum system alone is used to control the source of the fresh air and insure its purity. Exhaust systems of ventilation are therefore of value only in connection with a positive air supply. As a rule, all well-ventilated structures depend neither upon the plenum nor the vacuum systems alone, but combine the two.

A complete plenum system consists of (1) screens which strain out dust and large particles, (2) a fan to force the air through the system, (3) tempering coils to heat the air to a moderate degree—50° to 70° F., (4) spray chambers to humidify and wash the air, (5) baffle plates or eliminators to remove entrained water particles, and finally (6) heating coils to bring the temperature of the air to the final point desired.

The disadvantages of the mechanical systems of ventilation are that they are expensive as to first installation and as to maintenance; furthermore, they are most effective when the doors and windows of the room are kept closed. They require expert supervision. The advantages are that they are effective in all kinds of weather, and require less space for the air ducts than natural ventilation.

HEATING

Heating and ventilation go hand in hand. A large share of the cost of heating is chargeable to ventilation; hence, if ventilation is overdone, it is an unnecessary expense. The artificial warming of houses has a similar action to clothing. "Burning fuel in the furnace saves fuel in the human machine." It especially saves the strain upon the metabolism of the young, the old, and the feeble. The tendency in winter is to wear too much clothing indoors. This results in coddling—that is, loss of vasomotor tone of our peripheral capillary circulation, from the constant bathing of the skin in a still, warm, moist layer of air. This in turn results in susceptibility to drafts and liability to colds. It is quite unnecessary to wear heavy winter clothing in rooms and offices properly heated and ventilated. Dependence should be placed on warm overcoats when going out of doors.

Most of our American buildings are overheated with abnormally dry air in the winter time. This is a mischievous combination. It causes excessive evaporation from the skin and mucous membranes, which gives rise to a feeling of chilliness. It also causes dryness of the skin and mucous membranes, irritation of the throat, and thus predisposes to colds and respiratory infections. Warm dry air does not give the same sense of warmth and comfort afforded by a cooler moist air. Thus, air at 68° F. and a relative humidity of 70 per cent feels warmer than air at 71° F. and a relative humidity of 20 per cent or less. (Compare the effective temperature and comfort zone, page 820.) It is easy to regulate temperature automatically by thermostats. Furnace heat, hot-water, and steam pipes tend to dry the air, and thus it becomes necessary to overheat our offices and houses before they become comfortable.

Heat is measured by the *British thermal unit* (B. T. U.), which is the quantity of heat required to raise the temperature of a pound of pure water one degree at its point of maximum density, 39° F. The *French thermal unit* is the calorie and is the amount of heat required to raise one kilogram of water one degree centigrade at corresponding temperature (4° C.). One calorie equals 3.968 B. T. U.

Heat travels by *radiation*, *conduction*, and *convection*. All three routes are constantly in operation in any system of heating. Thus, with an open fireplace the heat radiates in straight lines to the nearest objects, where they are absorbed or reflected, just as light passes through space independent of the atmosphere. That is why our face toasts and our back freezes before an open fireplace. The heat absorbed by any object passes through that object from particle to particle by conduction. Most metals are good conductors; air is a

very poor conductor of heat. Convection is the process by which heat is communicated through gases and liquids as a result of their mobility. Thus, the air is warmed by our bodies, by hot-water pipes, and by all heated objects, and therefore rises and establishes convection currents.

There are five main methods of heating: (1) open fires; (2) stoves; (3) hot-air furnaces; (4) hot-water or steam pipes; (5) electricity. The control of the temperature of a building is more a question of management than of the system used.

Open Fires.—The open fireplace heats mainly through direct radiation. It has the advantage of being cheerful and a good ventilator. It has the disadvantage of being wasteful, drafty and very unequal if depended upon as the chief source of heat. Although not economical, the open fireplace is deservedly popular.

Parkes and Kenwood estimate that, in an ordinary medium-sized sitting room with an ordinary fire, from 10,000 to 15,000 cubic feet of air are drawn up the chimney in an hour, the current being generally from three to six feet a second. "As ventilating agents," say Notter and Firth, "the best types of open fireplace cause some 2,600 cubic feet of air to pass up the flue per pound of coal consumed, or the passage of about 18,000 cubic feet up the chimney per hour." A *fireplace* will change the air of an ordinary room in one or two hours.

Franklin Stoves.—Franklin stoves consist of coal fires in a cast-iron stove, the products of combustion being carried off through a stovepipe. Such stoves, standing free in the room, are very efficient, so far as heating is concerned, and also favor ventilation through the circulation of air, which is drawn into the stove to support the burning of the fuel. The heating of the room is unequal, as it depends largely upon radiation and somewhat upon convection. Such stoves are apt to become red hot, in which case the red-hot iron acts as a catalyzer, reducing carbon dioxid to carbon monoxid. The organic dust in the air falling upon the hot stove is burned and produces an unpleasant smell.

Open Gas and Oil Heaters.—Open gas and oil heaters without flues to carry off the products of combustion are objectionable, from a sanitary standpoint. These heaters consist of a variety of designs. The heat is imparted to the room by convection and also by radiation. Such devices may contaminate the air with carbon monoxid from leakage or from unconsumed gas, or from the formation of soot, which becomes incandescent. Such heaters also contaminate the room with carbon dioxid and other products of combustion. The "rubber" tube feeding these gas heaters often leaks, and there is frequently a perceptible odor of gas in rooms where these devices are used. The radiant type of gas heater is especially apt to form carbon monoxid. Open heaters burning oil are less objectionable than those using gas.

Hot-Air Furnaces.—A hot-air furnace consists of a coal fire which heats a series of tubes or plates in the dome of the furnace. The air, which is usually taken from the outside through a duct, flows into this dome, where it comes in contact with very hot surfaces, and is thus conducted by thermal circulation

through a series of ducts into the rooms of the house. A hot-air furnace of this kind constantly pumps fresh air into the house and is, therefore, a very efficient system of ventilation. The objection to the hot-air furnace is that the air is excessively dry and often partly "burned" in passing over the heated surfaces in the dome. The odor caused by the burning of the organic particles in the air may frequently be noticed in houses heated with a hot-air furnace. The heated air entering the rooms is usually allowed to escape as it will. In order to overcome the disadvantage of the dryness of the air furnished by the hot-air furnace, water pans are always provided, from which the water is supposed to evaporate. These pans are ridiculously small and cannot possibly furnish sufficient moisture for the great volume of air constantly passing through one of these furnaces. For instance, according to Harrington, air at 25° F. saturated with moisture and then heated to 70° F. would need half a pint in every thousand cubic feet to give it a humidity of 65 per cent. Ingersoll calculates that a house containing 17,000 cu. ft. of space would require for a relative humidity of 40 per cent at 70° F. in the air already containing 20 per cent humidity and changed once an hour, about 15 gallons of water a day. The little water pockets in the average hot-air furnace are insignificant and inadequate.

The air from a hot-air furnace is drier than that furnished by any other system of heating or ventilation. Thus, an out-of-door air in winter at a temperature of 0° F., with a relative humidity of 50 per cent, when heated to 70° F., will have a relative humidity of only 3 per cent. This is drier than the air of the driest climate known, which is seldom less than 30 per cent. The average relative humidity in Death Valley, California, is 23 per cent; at Yuma, Arizona, it is 35 per cent during the driest month of the year; at Santa Fé, New Mexico, it is 29 per cent; and at Pueblo, Colorado, it is 38 per cent. It is not unusual for the excessively dry air of a furnace-heated house to cause the woodwork to shrink and fall apart, the bindings of books to crack, etc. Living in such an atmosphere is not normal and must be harmful (page 829).

Hot-Water and Steam Pipes.—This is a very simple and effective system of heating buildings. The hot-water system is especially applicable to small buildings and steam pipes to large buildings. The hot water is more readily controllable than steam, which has a tendency to overheat. Special furnaces are found on the market to heat the water or to generate the steam, which then circulates through pipes to the rooms where wanted. If the hot-water radiators or steam coils are exposed directly in the room, the system is known as the "direct." In the indirect system the hot-water pipes or steam coils are placed in a special box where the air from the outside is heated, and this heated air flows by thermal circulation through ducts into the rooms where wanted. The best practice uses a combination, namely, the direct-indirect system. In the direct system the air of the room is simply heated and reheated over again, while in the direct-indirect system the fresh warmed air is constantly pumped into the building and it is, therefore, an efficient method of

ventilation. In both these systems the air is abnormally dried, just as it is in the hot-air furnace, though not to the same degree. Actual tests during the winter time of the air of a steam-heated office in a modern building at Topeka, Kansas, showed the average indoor relative humidity to be 23 per cent, with an average temperature of 72° F. The outdoor humidity at the same time averaged 82 per cent.

Electric Heating.—Electric heating is clean, easily regulated, but expensive. It has the disadvantage of being insufficient as a ventilating device, unless special inlets and outlets are provided. Electric heaters consist simply of resistance units which heat the room mainly through radiation and convection.

The Cooling of Rooms.—Much attention has been given, through necessity, to the heating of rooms in winter time, but heretofore little attention has been given to the cooling of rooms in the hot season. It is quite as practicable to cool rooms at it is to heat them, and sometimes quite as important to health.

The principle of practically all cooling devices depends upon the fact that when a fluid evaporates to its gaseous state it absorbs a considerable amount of heat—latent heat. This heat is taken from the surrounding objects which, therefore, become correspondingly cold. Cold may also be produced by the expansion of air. This was pointed out in 1845 by Joulé. Thus, if a jet of air at 60° F. were blown into a room under pressure of ten inches of mercury above the ordinary barometric pressure, the sudden expansion of this compressed air would reduce it to a theoretical temperature of 13.3° F. below freezing. This principle of dynamic cooling has been applied to refrigerators.

Ammonia gas and also sulphur dioxid are now almost universally employed in freezing machines. These gases are readily condensed into a liquid. The compressed gas is allowed to expand into tubes, and the cold thus produced utilized directly; more frequently an indirect method is used by which the expanding gas first cools a freezing mixture consisting of a saturated solution of calcium chlorid; this chilled brine is then pumped through a series of pipes to the refrigerator or compartment where it is desired. Humidifiers and air washers are also used to cool rooms and buildings.

A simple method of cooling a room is by the rapid evaporation of water. Manning was able satisfactorily to cool a large room in the Government Printing Office at Washington by blowing air by means of an electric fan over a moist sheet. This sheet, about a yard wide, was hung near the ceiling, and constantly wetted by a stream of water flowing over it.

The Mt. Sinai Hospital in New York provides a specially cooled room for babies with summer complaint, in view of the direct relation between heat and infant mortality.

SECTION VIII

SOIL

CHAPTER I

GENERAL CONSIDERATIONS

The upper layer of the earth's crust, known as the soil, is derived from the disintegration of rocks and the decay of animal and vegetable matter of all kinds. It varies from a few inches in depth to several feet. The sub-soil also varies from a few feet to hundreds of feet in depth, to hard pan or an impermeable stratum.

From a sanitary standpoint the soil must be regarded as our friend rather than an enemy. Enormous quantities of organic matter and infections of all kinds find their final resting place in the soil and are there disposed of and rendered harmless by nature's beneficent processes. In fact, a closer study of the functions of the superficial layer of the soil shows that it is not only the organ of digestion and respiration of the earth, but, like the liver, it is the great organ in which toxic substances of all kinds are neutralized or destroyed.

The sanitarian does not look upon the soil as dead and inert, but rather as a living being, for it presents many of the vital phenomena that characterize life: digestion, metabolism, assimilation, growth, respiration, motion, and even reproduction. The soil breathes, it absorbs oxygen and exhales carbon dioxide; it is capable of digesting and assimilating vast amounts of organic matter by a complex process of metabolism; the waste products are excreted. If these wastes are retained the soil may be choked or killed by an accumulation of its own poison—a sort of auto-intoxication. The soil, like all living things, demands water, but it may be drowned by an excess. A water-logged soil dies in very much the same sense that an individual dies with dropsy. A dry soil is a desert waste. Sedgwick speaks of the "living earth" in the sense that it is teeming with life; bacteria, molds, amebæ, and many of the primitive forms of the animal kingdom, as well as worms, insects, snakes, birds, rodents, and many other animals, make their temporary or permanent homes in the upper layers of the earth. Earthworms by their plowing action, so beautifully shown by Darwin in 1881, constantly turn over the upper layers of the earth. Sand dunes illustrate lateral motion. The soil, therefore, is in constant peristalsis, which helps its digestive functions. The rise and fall of the ground water is analogous to the movements of the diaphragm and assists the respiratory functions of the soil.

Classification of Soils.—Soils are variously classified, depending upon the amount of sand, gravel, clay, loam, humus, peat, muck, rock, alkali, etc., which they contain. The difference between a sandy and gravelly soil depends mainly upon the size of the particles. These soils interest the sanitarian because hookworms live and flourish in them better than they do upon clay or rock formation. Soils are also wet or dry depending upon the level of the ground water and the composition of the upper layers.

Clay exists in particles of the smallest possible size. It is very cohesive, possesses a high degree of plasticity, and plays a very important part in determining the fertility of soils, their texture, and their capacity for holding water. Its plasticity is due to the presence of a small proportion of hydrated aluminium silicate, and is modified very greatly by the addition of less than a hundredth part of caustic lime. It is exceedingly impermeable to water, and when wet dries with great slowness. Clay may be regarded as a plastic colloid, but its special properties are only seen when a certain amount of water is present. The separate particles of clay are so small that, when placed in water, they assume a state of Brownian movement and sink only very slowly in spite of their high specific gravity. Traces of electrolytes have a profound effect on these properties; small quantities of acids or salts cause the temporary loss of plasticity, impermeability, and the property of remaining long suspended in water without settling; the clay is now said to be flocculated. The change can be watched if a small quantity of any flocculating substance is added to the turbid liquid obtained by shaking clay with water; the minute particles are then seen to unite with larger aggregates which settle, leaving the supernatant liquid clear. There is, however, no permanent change; deflocculation takes place and the original properties return as soon as the flocculating agent is washed away. Alkalies (caustic soda, caustic potash, ammonia, and their carbonates) deflocculate clay, causing it to remain suspended in water for long periods. Clay is thus an electronegative colloid, its reaction probably being conditioned by a trace of potash liberated by hydrolysis. It shows the general properties of electronegative colloids as elucidated by Schultz and by Hardy; thus, it is flocculated only by a solution containing ions or particles of opposite electrical sign, and the extent of flocculation increases rapidly with the valency and concentration of the ion.

Loam consists of a mixture of sand, clay, and humus. If the sand predominates, the soil is said to be light; if the clay predominates, heavy. A rich soil contains an abundance of humus.

By *humus* is meant the products of vegetable decomposition in their various intermediate stages of decay. It is the essential element of vegetable mold, and is necessarily of most complex composition. It is composed of a great number of closely related definite chemical compounds.¹ Humus contains a high percentage of nitrogen, especially marked in some of our prairie soils and in the "black soil" found in the provinces of the Ural Mountains, which,

¹ See Bulletins of the Bureau of Soils. Department of Agriculture.

according to von Hensen, contains as much as from 5 to 12 per cent of organic matter.

Surface Configuration.—*Geodesy*, or surface configuration, has an important relation to health. Low and swampy ground is a breeding place for the malarial mosquito. Highlands are apt to be drier and more healthful than lowlands. A slope affords better drainage than flat lands, and thus diminishes the dangers from soil pollution, but increases the risk of infection being washed down from those living above. In narrow valleys the air stagnates, the moisture is excessive in both the soil and the air, there is lack of sunshine and there is an unpleasant blanket of cold layers of air at night. Mountain sides are notoriously windy. High plateaus suffer from extremes of temperature. Thus, at Mexico City (about 8,000 feet above sea level) there is a sharp contrast between the temperature during the day and night, and even during the daytime between the sunshine and the shade. At Quito, which is 9,350 feet above the sea level, the daily variation of temperature at some periods of the year is no less than 34° F. Northern exposures may not get enough sunshine, and southern exposures sometimes too much.

The relation of the surface configuration of the land to health is intimately interwoven with the whole question of climate, and must take into consideration temperature, air movements, humidity, sunshine, barometric pressure, precipitation, and the seasons with their endless varieties from tropical to arctic.

Composition of the Soil.—Much attention was formerly given to the hygienic importance of the chemical constituents of the soil. The presence of organic substances was regarded not only with suspicion, but even as a serious menace to health. It was claimed that organic pollution of the soil made a good culture medium for the germs of infectious diseases. The gaseous products of decomposing organic matter in the soil have long been looked upon as particularly injurious. These gases, with other ill-defined but unknown volatile substances, are spoken of as *miasma* or *effluvia*.

We now know that very few, if any, of the bacteria pathogenic for man grow and multiply in the soil under natural conditions. The spores of tetanus, malignant edema, botulism and anthrax may live in garden earth for many years, but it is doubtful whether these microorganisms, especially the anaërobes, ever find conditions favorable for growth and multiplication in the soil. Ordinarily typhoid, dysentery, and cholera bacilli and other non-sporing aërobes do not flourish in the soil; on the contrary, they soon die there. It has been shown that cities built upon polluted soils have sometimes suffered relatively less from typhoid and cholera than cities built upon rocky or virgin soil. In some cities (as Budapest) it has been pointed out that the greatest morbidity and mortality rate was in that part of the city built upon made ground filled in with trash and much organic waste. These instances have been coincidences, for, as a rule, the low-lying, polluted soil happened to be the poor, crowded tenement district. A sanitarian does not recommend polluted soils for building sites, but it seems that their influence upon health has been over-

stated, especially where cellars are properly constructed. While a polluted soil may not be hazardous in the ways just indicated, it may be dangerous so far as hookworms and other parasites are concerned, or indirectly it may lead to contamination of drinking water, food, etc. (See Pollution of the Soil, page 919.)

Mineral Matters in the Soil.—By far the most abundant element in the soil is oxygen. According to various estimates, from 33 to 50 per cent of the solid crust of the earth consists of oxygen. The other elements found in abundance in the soil are: silicon, carbon, sulphur, hydrogen, chlorine, phosphorus, fluorine, aluminium, calcium, magnesium, potassium, sodium, iron, manganese, and barium. Aluminium silicate or clay makes up perhaps two-thirds of the inorganic components of soils. Other compounds are lime and magnesia carbonates (limestone) and numerous chlorides, sulphates, phosphates, oxides, etc., of the various bases.

Iron is universally present and gives the red color to soils. Nitrogen exists in soils in three distinct forms: (1) protein and its split products, (2) ammonia and its salts, and (3) nitric acid and nitrates or nitrous acids and nitrites.

Plant Matter in the Soil.—The vegetable matter exists in the soil in various stages of decomposition. One result of the decay of vegetable substances is the formation of organic acids, which have considerable power to dissolve mineral substances, accounting in part for the plumbisolvant action of acid-reacting surface waters from swampy lands.

Peat or muck results from the incomplete decay of vegetable matter under water.

Animal Matter in the Soil.—Organic matter of animal origin in soils results chiefly from the decomposition of carcasses or from contamination with the excreta of animals. As a rule, animal matter is neither so abundant nor so widely distributed in the soil as vegetable matter. From a sanitary standpoint soils polluted with organic matter of animal origin present a greater danger than soils polluted with vegetable matter. The wastes from man present the greatest hazard.

Physical Properties.—In general it may be said that the physical properties of a soil are more important, from the standpoint of health, than its chemical composition. A good soil is a spongy mass, radio-active and contains numerous colloidal bodies.

Porosity.—By the porosity, or pore volume, of a soil is meant the volume of the interstices between the particles, which may be filled with water or air, or both; in other words, the power to absorb water. Porosity is expressed as a percentage of the whole mass. Ordinarily the pore volume in soil amounts to about 40 per cent; some apparently compact masses, such as sandstone, have as much as 30 per cent. The pore volume of the soil is independent of the size of the individual grains.

Permeability.—The permeability of a soil is its ability to allow the passage of water; it does not depend upon the pore volume, but upon the size of the

individual pores. Rocks may have a high porosity, but slight permeability, due to the extreme fineness of the pores. Clay has a high porosity, but its permeability is slight, owing to the extremely small size of the pores, although their aggregate capacity is rather large. The presence of fissures and water in the rock greatly increase its transmitting property.

Water Capacity.—The water capacity of the soil is the amount of water held in the interstices of the soil when saturated, while the *water-retaining capacity* is the amount of water held back after a saturated soil is drained.

Soil Temperature.—The sun is the principal source of the soil temperature. Some heat is produced from chemical changes, but not in considerable amounts. The original heat of the earth's interior furnishes a constant source of heat that is of much importance.

The heat absorbed and given off by the soil has a notable influence upon the atmospheric temperature. Some soils and moist surfaces absorb heat from the sun and give it off again when the sun has set. The most heat-absorbent soils are sandy soils. The sand of the desert may be heated to 200° F., and when this hot sand is raised by simoons the temperature of the air in the shade may reach 125° F. or more. The power of absorbing or reflecting solar heat also depends upon the color of the soil.

Adsorption.—The soil has, to a remarkable extent, the property of adsorbing odors and gases, and ordinarily it is very hygroscopic. The soil is also capable of holding toxins, colors, and other substances through the physico-chemical property of adsorption. In this respect it acts like charcoal. Illuminating gas from leaky mains may be divested of its odorous constituents in its passage through the soil, so that its presence in houses may be undetected, thereby greatly increasing the danger. In the experiments made by Abba, Orlandi, and Rondelli about the filtering galleries of the Turin water supply the property of the soil to hold back substances in solution was shown. Cultures of *Bacillus prodigiosus* in large volumes of water poured into the ground at various points made their appearance 200 meters away in forty-two hours, whereas dyes, such as methyleosin and uranin, could not be detected until after seventy-five hours.

Soil Air.—Air is present in all soils, even in the hardest rocks. Sandstone may contain from 20 to 40 per cent, sand from 40 to 50 per cent, and humus as much as two to ten times its own bulk. The soil air differs markedly in composition from that of the atmosphere. It is usually very moist and contains various gases, especially carbon dioxid, resulting from the decomposition of organic matter. For the same reason soil air contains less oxygen than the free atmosphere. The soil air varies greatly, according to the character of the soil, the climate, the season, and rainfall. There is a continual interchange between the air of the soil and the air of the atmosphere. This interchange is influenced by differences in temperature, by rainfall, and by the movements of the ground water and by barometric pressure. Rain chokes the pores and checks soil ventilation. The soil air is in constant motion.

Following the teachings of Pettenkofer, the amount of carbon dioxid in

the soil air was for years taken as an index of the amount of soil pollution. It is now well known, however, that this is not a reliable index, for the reason that many conditions influence the amount of carbon dioxid in soil air. A soil recently manured may contain from two to five or even ten parts of carbon dioxid per thousand. In a gravelly soil the proportion may be as high as eighty parts per thousand.

Soil air may influence health when contaminated with poisonous gases, such as carbon monoxid. This occasionally happens. In the open these gases would be so greatly diluted that they could scarcely exert a deleterious influence, but when concentrated, as they sometimes are in dwellings, and breathed for a long period of time they may be responsible for anemia, headache, and other symptoms. Soil air containing carbon monoxid may be sucked into a dwelling from long distances in a lateral direction. Leaky gas pipes may thus render the air of a dwelling impure if the cellar is permeable. This is favored by the pumping action of the furnace, especially when the surface of the ground is frozen.

Soil air is practically sterile; that is, under ordinary conditions it contains few bacteria. Odors sometimes contained in the air from a polluted soil have no known injurious effect.

Soil Water.—The passage of water through the soil is essential to soil activity. The moisture favors the bacterial growth by which soils purify themselves and favors vegetation. Nitrates, chlorids, and other soluble substances are dissolved in the water and pass into the subsoil, or furnish food to the roots of plants. A soil absolutely dry, as a desert soil, is lifeless. A soil with an excess of moisture, that is, one in which the ground water level is at or near the surface, delays and alters the natural decomposition of organic matter. In the deeper layers of the soil, where no bacterial action takes place, vegetable matter may remain almost permanently without change. Thus, wooden piles are not attacked after centuries.

Water exists in the soil in two principal forms: (1) *soil moisture*, which comprises the water present in the interstices of the upper partly saturated layer, as well as the watery vapor contained in the soil air; and (2) *ground water*, or subsoil water, in which case the interstices of the soil are completely filled.

The soil moisture is estimated by determining the loss of weight by drying 10 grams of soil at 100° C. to constant weight. The dry sample may then be exposed to air saturated with moisture under a bell jar and again weighed. The increase in weight indicates the absorptive power of the soil.

Water may also be regarded as existing in the soil under three conditions, viz., *hygroscopic*, *capillary*, and *gravitation*. The hygroscopic water is that which adheres to the surface of the soil particles in the presence of air. The capillary moisture is that which is held within the spaces that are capillary in size. The gravitation water is that which drains through the soil and accumulates in the subsoil over an impermeable stratum. For a discussion of ground water see chapter on Water.

It is generally stated that a persistently low ground water level, viz., fifteen to twenty feet, is healthful, and that a persistently high ground water level, viz., three to five feet, is unhealthful, and that a ground water level that fluctuates suddenly is still more unhealthful. Pettenkofer found that typhoid fever was more likely to occur at Munich, Berlin, and Leipzig when the ground water level was at its lowest. His explanations to account for this were ingenious, but we now know that the relation was only a coincidence, for the same does not hold in other places.

Subsoil drainage is usually considered more of an agricultural necessity than a public health question. Large tracts of our land in the Middle West and in other parts of the world have normally a high ground water level, and it is necessary to bring this down in order to increase the fertility of the soil. This is done by draining the subsoil, which also abolishes marshy and swampy lands, and thus puts a check upon malaria.

One of the principal influences of the soil upon general health is through soil moisture. Dampness in or near the surface of the soil may affect the health of those dwelling nearby: Such a soil is cold, and the atmosphere immediately above it is liable to be damp, and this a desert soil, is lifeless. A soil with an excess of moisture, that is, one in which the ground water level is at or near the surface, delays and alters the natural decomposition of organic matter. In the deeper layers of the soil, where no bacterial action takes place, vegetable matter may remain almost permanently without change. Thus, wooden piles are not attacked after centuries.

THE NITROGEN CYCLE

The most interesting of the vital phenomena taking place in the soil is the disposal and utilization of organic matter. This may best be illustrated by the nitrogen cycle, which must be understood in order to have a clear conception of *soil pollution*, *water purification*, and *sewage disposal*.

The nitrogen cycle is a complex series of events which protein matter undergoes, in which it is reduced to simple and stable inorganic compounds, and then returns through plant life to the animal kingdom. One phase of the cycle, namely, the breaking down of animal and vegetable matter, is due almost entirely to bacterial action. The other phase, namely, the building up of complex living organic matter from simpler compounds and elements, is mainly a function of living plants.

The nitrogen cycle is a process in which the anabolism or synthesis occurs in plants, while the catabolism or analysis is brought about chiefly through bacterial action. Hence the series of events constituting the nitrogen cycle largely depends upon the plant kingdom. The important phases of the cycle occur upon the soil and in its superficial layer. It will presently be seen that this cycle has a fundamental importance in sanitary science, and has a special significance in preventing soil pollution, in the purification of water, and in

the disposal of sewage. It is evident that any permanent break in this cycle would result in the cessation of life upon the earth.

The supply of combined nitrogen on the earth is comparatively small, and it is possible to calculate approximately the time necessary for its exhaustion if not replenished. In its free form, nitrogen occurs in enormous quantities, four-fifths of the atmosphere being composed of it. According to Hopkins, the amount of nitrogen over each acre of the earth's surface is enough to meet the needs of a 100-bushel crop of corn every year for 500,000 years. Large quantities of nitrogen are required by all living organisms, which are constantly returning it to its inert atmospheric form. Nitrogen exists in plants and animals mainly in the form of protein. Enormous quantities of nitrogen flow away in sewage; explosives return vast amounts from its fixed to its gaseous form. This fact led Sir William Crooks² to predict dire calamity to the human race if science were not able to utilize atmospheric nitrogen. Fortunately, this happens in nature in the process of nitrogen fixation which starts nitrogen again on its cycle from the dead to the living. Furthermore, methods have been discovered to fix atmospheric nitrogen which are now in successful operation on a large commercial scale.

Decomposition of Nitrogenous Matter.—As soon as an animal or plant dies its protein constituents are at once attacked by putrefactive bacteria. The proteolytic microorganisms growing in and upon the nitrogenous matter break it up into secondary and simpler products, which have a striking correspondence to the cleavage products of gastric and pancreatic digestion. Some of the putrefactive bacteria, of which the *Bacillus subtilis* and the *Bacillus proteus* are important types, liquefy protein matter during the process of putrefaction. Other bacteria, of which the colon bacillus is an example, break down organic matter without evident liquefaction. Very many other species of bacteria take part in this stage of the cycle. Anaërobes are also active. For the most part the microorganisms pathogenic for man are killed during the process of putrefaction; they die in the struggle for existence. The processes of decomposition are essentially the same, whether the organic matter is the carcass of an elephant, a beetle, a tree, or a leaf, provided that the necessary moisture, warmth, and other conditions for bacterial activity are present. The breaking down of vegetable matter is slower and more difficult than the breakdown of animal matter. This is due in part to the fact that the latter contains larger percentages of putrescible protein and also usually contains more moisture, which favors bacterial activity. Decomposition and putrefaction are fully discussed on page 663.

The breaking down of the complex protein molecules to simpler and stabler compounds is usually spoken of as mineralization, and may be regarded as a series of oxidations. According to our present chemical conception, it is really a series of hydrolyses. The complicated molecular structure of protein matter is analyzed into amino compounds of simpler and simpler composition, until

² Address before British Association for the Advancement of Science, 1898.

nitrogen appears in the form of ammonia. We know little of the chemistry of the early stages of protein decomposition. The process seems hopelessly complicated from the intricate structure of the molecule. Eventually from the seething caldron of molecular disintegration there appear simpler substances, such as proteoses, peptone, ptomains, amins, leucin, and tryosin, and other amino substances, as well as organic acids, indol, skatol, phenol, and finally sulphurated hydrogen, mercaptan, carbon dioxid, and ammonia.

One of the final products of the process is carbon dioxid, part of which passes into the atmosphere and part of which is retained in the soil as carbonates of alkaline bases. The ammonia, as such, cannot be used by plants. Some of it may escape into the atmosphere, but for the most part it is retained in the soil as ammonium chlorid or ammonium carbonate. In the soil the ammonia is oxidized by the action of nitrifying bacteria first into nitrites and then into nitrates. This nitrifying action of bacteria, revealed by Winogradsky,³ in 1888, was one of the brilliant discoveries in bacteriology. Through his ingenious work and that of later workers, it is now known that this process is usually accomplished in two distinct steps. The ammonia as carbonate, which is rapidly oxidized by nitrosomonas into nitrite, and this by nitrobacter into nitrate.

Nitrosomonas and Nitrites.—All attempts to isolate bacteria which possess the power of nitrification proved futile until Winogradsky's epoch-making discoveries. He used ingenious methods and keen reasoning in his experiments. He was finally able to isolate in pure culture a bacterium which he called *Nitrosomonas*, which was capable of availing itself of the abundant energy from the oxidation of ammonium compounds. *Nitrosomonas* is a rod-shaped or spherical cell, usually at rest but at times capable of motion; if motile, with polar flagella. They grow best on media containing no organic substance. The type species is *Nitrosomonas europaea*, described by Winogradsky in 1892.

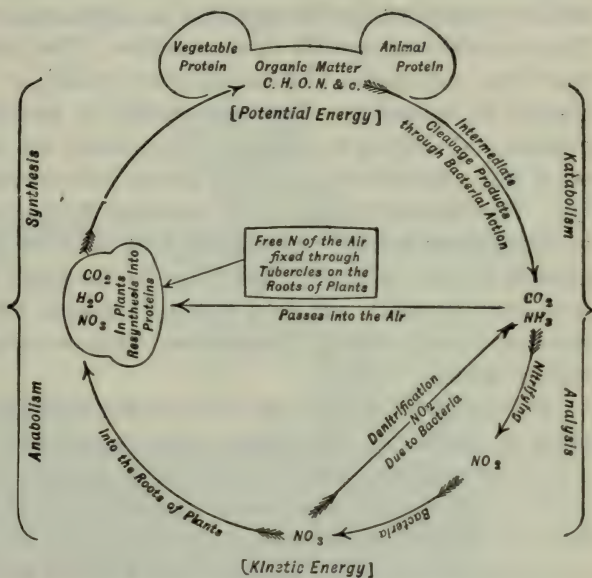


FIG. 80.—THE NITROGEN CYCLE.

³ *Ann. de l'Inst. Pasteur*, 1890, 4: 275, 760; also *Arch. d. sci. biol.*, St. Petersburg, 1895, 3: 207.

These and other nitrifying organisms are found in abundance in all cultivated soil and in soil water containing a due proportion of magnesium carbonate, sulphate, etc., which affords a favorable medium for their development. The ammonia is converted into nitrous acid (nitrites), and the nitrites into nitric acid (nitrates) by means of the abundant special ferments.

The nitrous acid combines with bases and the nitrites exist in the soil mainly as salts of potassium and sodium. They remain as the lower oxid a very short time and therefore never accumulate, and thus are never found in any large amount for they are unstable and readily oxidized to nitrates.

Nitrobacter and Nitrates.—The nitrate-forming organisms were also accurately described by Winogradsky in 1892, and called by him nitrobacter. They are also very specific in their action. They are rod-shaped cells, aërobic, spore-free, not motile and do not grow readily on ordinary media or in the absence of ammonia. They are capable of securing growth energy by the oxidation of nitrites to nitrates. Nitrobacter are widely distributed in cultivated soil. No other organism is known with certainty to produce nitrates in soil. The nitrates are stable and represent the final stage of the mineralization of nitrogenous matter. In certain arid parts of the world large deposits of nitrates (KNO_3 , saltpeter) are found as the result of the nitrification of bird excrement (guano), which is rich in available nitrogen. These collections, however, do not occur in places where there is enough rain to carry away the readily soluble nitrates.

Ordinarily the nitrates go into solution in the ground water and are either taken up by the roots of plants or are washed away in the ground water. In a sanitary analysis of water taken from the soil the presence of nitrates and nitrites, therefore, has a special significance. If nitrites are found in soil water it indicates pollution and signifies active bacterial action and the presence of organic matter. Nitrates in soil water, without nitrites, are an index of past pollution (see Water Analysis).

Denitrification.—In 1886, Gayon and Depetit described two organisms, *B. dentrificans* α and β , capable of reducing nitrates with the evolution of gaseous nitrogen. The organisms are widely distributed in soil. Many bacteria have this power of denitrification, a sort of reversible process by which nitrates are reduced to nitrites, but those capable of reducing nitrates to ammonia and further to gaseous nitrogen are not very numerous. The reduction of nitrates to nitrites is characteristic of very many of the well-known microorganisms, such as the colon group, pyocyanus, subtilis, and many other bacteria. Maasen found this action in 85 of 109 kinds of microorganisms studied. Denitrification, however, does not occur in a well-ventilated soil, for it requires lack of air, presence of much decomposable organic matter and of nitrates—a combination rarely found in nature.

Nitrogen Fixation.—Within the soil two great groups of bacteria possess the power of fixing nitrogen. The course of the reaction by which these organisms effect the fixation of nitrogen is entirely unknown. The members of one group live free in the soil and are able with the energy they obtain from

the oxidation of organic carbon to build up complex nitrogen compounds. This is known as non-symbiotic nitrogen fixation. The bacteria of the other group are able to fix nitrogen by living in symbiosis on the root nodules of certain plants (the symbiotic group).

Non-Symbiotic Group.—The first group consists of free-living bacteria in the soil. They have the ability to fix the free nitrogen of the air independently of plant life and evidently get their energy from the organic carbon. One of the first known of this group was a large spore-forming anaërobe described by Winogradsky in 1895, and named by him *Clostridium pastorianus*. Beijerinck,⁴ in 1901, described a group of large aërobic bacteria which are also able to utilize free nitrogen for the purpose of building up complex protein. This

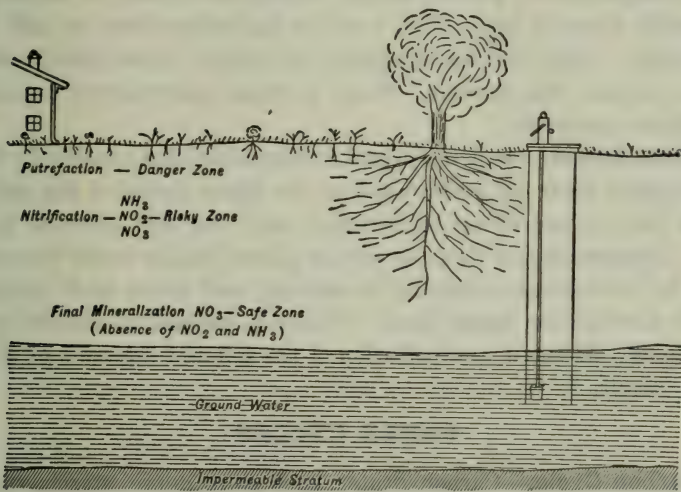


FIG. 81.—THE NITROGEN CYCLE IN DIAGRAMMATIC VERTICAL SECTION.

group goes by the general name of *Azotobacter*, several species of which have been described.

Symbiotic Group.—The second group of organisms grows on the root nodules of certain plants, especially legumes (peas, beans, clover, lupines). It has long been known that such crops enrich the nitrogen of the soil, while ordinary grains deplete it. Thus, a crop of crimson clover is able to add more than two hundred pounds of nitrogen per acre. Woronin,⁵ in 1866, discovered that root nodules are crowded with rod-shaped bacteria, which we now know are living in symbiotic relation to the plant. Beijerinck⁶ in 1888 grew these bacteria in pure culture and named them *Bacillus radicicola* (now *Rhizobium radicicola*). They are very pleomorphic and have the remarkable

⁴ *Centralbl. f. Bakteriöl.*, 1901, 2: 561.

⁵ *Bot. Ztschr.*, 1866, 24: 329.

⁶ *Bot. Ztschr.*, 1888, 46: 725.

ability of fixing nitrogen. Bacterial inoculation of soils to replace nitrogenous manuring is a fascinating possibility, but has not been realized in practice.

Specificity of the Reactions.—It will be noted that in the nitrogen cycle all the essential steps, from proteolysis to mineralization of the organic matter, nitrification, oxidation, and reduction, as well as the fixation of free nitrogen from the atmosphere, are all the result of bacterial action. Each stage of the complex process is specific, in the sense that it requires a particular species or group of bacteria to effect the result, and also specific in the sense that special conditions of environment are necessary for its action to take place. *Nitrosomonas* will oxidize ammonium carbonate and nothing else; it will not touch nitrites, urea, or the substituted ammonias. *Nitrobacter* is equally specific in oxidizing nitrites to nitrates.

The decomposition of protein occurs in stages as a result of bacterial action largely specific for each step. Thus very few microorganisms are able to attack native protein. After the decomposition is started, these specific organisms or groups continue the process. Some of these putrefactive organisms are aerobic, others anaërobic.

A Surface Action.—It is important to remember that practically the entire cycle takes place upon the surface and in the upper layers of the soil. A few feet below the surface of an undisturbed area the soil contains few or no bacteria. Carcasses buried deep, or sewage placed too far below the surface, do not profit by the nitrogen cycle in its entirety, and under such circumstances incomplete nitrification takes place. Nature's method of disposing of dead wastes is thereby defeated, and pollution of the soil and infection of the ground water may result.

OTHER CYCLES

The Carbon Cycle.—Carbohydrates, such as cellulose, starch, sugars, and similar constituents of vegetable and animal matter, are fermented, with the formation of carbon dioxid, alcohol, and various organic acids. The carbon in carbohydrates passes through a series of changes, which may be regarded as the carbon cycle. The carbon dioxid resulting from fermentation unites with water in plant life, and under the action of chlorophyll and sunlight is again built up to starch and sugars.

The fermentation of the carbohydrates is likewise due to the action of microorganisms. In a mixture containing both carbohydrates and protein, as a rule, the microorganisms act upon the carbohydrates first. In other words, the putrefaction of protein is delayed or hindered by the presence of fermentable carbohydrates. For this reason sewage containing wastes from breweries always presents difficulties at disposal plants.

Fats are also attacked by bacteria, with the consequent production of acids. The hydrocarbons are broken down with more difficulty than either the carbohydrates or protein. An excessive amount of fat in sewage always gives trouble on a filter. For instance, the drainage from a wool-scouring mill containing lanolin and the discharges from slaughterhouses and the

wastes from creameries, laundries, and cheese factories containing animal fat present special problems in sewage disposal.

The nitrogen cycle, as well as the carbon, sulphur, and phosphorus cycles, are all processes of oxidation—at least the terminal products are nitrates, carbonates, sulphates, and phosphates.

CHAPTER II

THE SOIL AND ITS RELATION TO DISEASE

Bacteria in Soil.—Countless millions of bacteria occur in the upper few inches of the soil. The enormous overgrowth of bacteria in the upper layers of the soil gives it the sticky, moist feeling which rich soils possess. The odor of the soil, such as that which is particularly noticed after a rainstorm, is due in large part to *Cladothrix odorifera* and other organisms which are commonly found in the soil. Few bacteria are found in an undisturbed soil below a depth of four to six feet. A sand bed used for filtering sewage shows a similar vertical distribution of bacteria. Below six feet the statement is made that the soil is usually sterile. This is not strictly true, but the numbers are much diminished and bacterial activity has practically ceased. As a rule, living bacteria are not obtained from samples of soil obtained ten to twelve feet below the surface, except in soils with large pores or crevices, or in cases where the bacteria have been carried by burrowing animals. It is exceedingly difficult to determine the number of bacteria in the soil, as so many of them are anaërobes and vast hordes belong to the nitrifying groups, which grow only upon selective media. The soil is also the home of many other species, requiring special conditions for growth in artificial culture media.

Of the ordinary bacteria that grow upon the usual laboratory media Houston found an average of 100,000 per gram in an uncultivated sandy soil, 1,500,000 per gram in a garden soil, and 115,000,000 per gram in a sewage soil. Peaty soils have smaller numbers. The actual numbers must be vastly greater, for many microorganisms in the soil do not grow upon the common media. In fact, the soil is the home of the greatest number and variety of bacteria found anywhere. It is the bacteria in the upper layers of the soil that make it resemble a living gland. Each particle of earth is coated with a zoögleal envelope. The sand and mineral particles form the supporting structures, the coating of bacteria corresponds to the glandular epithelium, and the interspaces between the particles are the capillary and lymph channels.

Most of the bacteria in the soil are saprophytes. The microorganisms pathogenic for man do not find conditions favorable for growth and development in the soil. For the most part the temperature is too low; further, they are crowded out by the overgrowth of the saprophytes. Koch has demonstrated that anthrax and other pathogenic bacteria may be grown in sterile soil, but cannot be grown in unsterilized soil, that is, in living soil. They die in the struggle for existence. Experiments have shown that the soil of graveyards contains no more bacteria than the corresponding soil in the same

locality, and is noticeable by the absence of pathogenic microorganisms. The soil often contains the bacteria (or their spores) of certain wound infections, such as malignant edema, anthrax, *B. aerogenes capsulatus*, and tetanus. The relation of the soil to typhoid, cholera, dysentery, hookworm disease, Cochin-China diarrhea, and other infections will be discussed presently.

Pollution of the Soil.—The soil is capable of disposing of great quantities of organic matter. However, if it is overburdened it remains polluted and may endanger health through contamination of the drinking water and in other ways. It is not only the amount but the kind of pollution, and also the manner of its disposal, that plays a very important part. It must first of all be remembered that the purifying action of the soil is largely dependent upon bacteria, and that this action takes place almost solely in the upper layers. If carcasses are buried deeply, or if sewage is allowed to enter the soil at several feet below the surface, the process of purification is long delayed or checked. A leaky cesspool or broken drain which discharges its contents into the soil at a depth of five feet or more may seriously pollute the ground water, whereas the same material placed upon or just beneath the surface may be entirely mineralized and all infection destroyed before it reaches the depth of five feet. Vegetable matter in a water-logged soil undergoes a partial and unusual decomposition into muck or peat. Trees buried deeply, where bacterial action is practically absent, remain for many hundreds of years practically unchanged. Many factors retard the purifying action of the soil. Among these the temperature and moisture and absence of oxygen predominate.

When organic matter falls upon the soil it is consumed and digested by the hungry earth. Without this property the surface of the earth would long ago have become clogged with vegetable and animal matter.

Pathogenic bacteria that may be thrown upon the soil in feces or otherwise are usually detained in the upper layers and finally destroyed there. Under ordinary conditions pathogenic microorganisms are caught in the upper layers of the soil, just as they are caught upon the "schmutzdecke" of a slow sand filter. The soil does not act simply as a mechanical trap. The bacteria are detained and destroyed by a combination of physical, chemical, and vital processes taking place in the upper layers of the soil.

All polluted soils are not equally dangerous. Soils polluted with human feces and urine present the greatest hazard to man. The special menace of soils polluted with human excreta is from hookworms, and other infections discharged in the feces or urine. Hookworm infection is contracted directly from soils polluted with human feces, and the eradication of hookworm disease depends primarily upon preventing pollution of the soil. The danger in the case of typhoid, dysentery, cholera, and other bacterial infections is usually indirect through infection of drinking water or occasionally through flies or other mechanical means of transference. A soil polluted with typhoid may endanger either the surface water or the ground water, particularly in limestone formations. Pathogenic microorganisms in a polluted soil may also

find their way back to man upon vegetables. Definite proof of any direct relationship between soil contamination and the spread of typhoid, cholera and dysentery is meager or wanting. Kligler¹ concludes from experimental and field observations that in moderately compact clay, sand-clay and sandy soil, free from cracks, the possibility of subsoil pollution of the ground water is negligible, provided the ground water is more than ten feet below the polluted area.

Tapeworms and other intestinal parasites pass part of their life cycle on or in the soil, and may infect man directly or indirectly in various ways. The question of soil pollution and the particular ways in which it is related to health have been discussed separately under each disease concerned.

Dirt.—The soil is often spoken of as dirt. The soil in the field is "earth," but in the parlor or on our hands it becomes dirt; that is, matter out of place. The word "dirt" is from the old Saxon "drit," meaning excrement. Dirt in the ordinary sense becomes a potential danger, especially when containing human excretions or soil bacteria associated with wound infections.

To the sanitarian dirt includes rubbish, manure, and organic wastes of all kinds. It may be the vehicle, but not the source, of infection. It harbors flies, fleas, lice, rats, mice, and vermin of all sorts that breed in it and act as intermediate hosts or carriers of infection. While dirt cannot originate typhoid fever or other infections, it favors conditions which encourage the spread of such diseases. Rubbish in vacant lots, in backyards, in alleys, in cellars, garrets, and other places may be taken as an index of the failure to appreciate the modern teachings of hygiene and sanitation. It was once the chief duty, and still an important one, of the health officer to insist upon cleanliness of premises and surroundings, both in country and city. There are still many who think that "the dirt rate of a city determines its death-rate"; that "to clean up a city means to clean out disease"; and "dirt and disease go hand in hand." We now know this is not so, for there is a wider and deeper significance to cleanliness.

Cleanliness.—Cleanliness is the heart and soul of sanitation. We are inclined to place it even before godliness, for cleanliness of body, cleanliness of mind and soul, and cleanliness of our surroundings are essential to a full appreciation of the spiritual virtues. Our conception of cleanliness has greatly changed with our advance in knowledge of the kinds of dirt, the degrees of dirtiness, and the nature of these dangers. We can no longer be satisfied with physical or æsthetic cleanliness, but must insist upon biological cleanliness. A tetanus spore upon the shining blade of a surgeon's knife makes that instrument filthy, whereas many such spores on the skin of a chicken may be harmless when ingested. We cannot see the infection upon the common drinking cup, upon the roller towel, upon the point of a pencil that has just been moistened with saliva, or in water, milk, or food, although we well know the danger of such invisible "dirt" that these objects may harbor.

¹ *Monogr. Rockefeller Inst. M. Research*, No. 15, Oct. 10, 1921.

It requires a bacteriologist to tell the difference between clean dirt and dirty dirt. We lack a sixth sense, or microscopic eye, to see and distinguish the harmful germs. We, therefore, must practice scrupulous cleanliness and educate the people to the biological meaning of this term. Long experience has taught the lesson that cleanliness offers a protection against disease; that clean surroundings are apt to be free of infection; and that clean food is apt to be safe food.

Cleanliness of person and environment results in the diminution of the crop of many pathogenic microorganisms, such as streptococci and staphylococci. Cleanliness of the type that approaches asepsis would prevent sickness and save lives through diminishing the risk of such infections. Hill believes that the mildness of modern infectious diseases is due to the lessened virulence, smaller stock, and reduced distribution of the streptococci and staphylococci formerly bred in hospitals and in wounds promiscuously. The public health officer should, therefore, campaign for personal and communal cleanliness.

Many houses, especially in poorer sections of cities, lack proper facilities for laundry work. Public laundries, such as are provided in many European countries, would materially help in the campaign for general cleanliness.

At one time the theory of the filth diseases reached the dignity of a special name—the pythogenic theory, first propounded by Murchison in 1858.² Typhoid fever was long regarded as the type of a filth disease, and, while we are now dropping that term, we should not forget that typhoid fever is really a filthy disease, for each case means that a short circuit has been established between the anus from one person and the mouth of another.

SOIL AND DISEASE

Soil was formerly accused of being responsible for many diseases. The list, however, has dwindled with our increase in knowledge. Infestations rather than infections may come from soil pollution. The number of infections directly associated with the ground is small and the indirect influences are less evident than formerly supposed. Definite proof of the direct relation between soil contamination and the spread of bacterial intestinal infection is meager. Apart from the one real danger, namely, soil polluted with man excrement, the sanitarian is now inclined to minimize the influence of the soil upon health. We now know that comparatively few, if any, of the bacteria of the intestinal group grow and multiply there. The following diseases associated with the soil need discussion.

Hookworm Disease.—Hookworm disease comes from soil pollution. It is the best example we have of a prevalent and widespread infestation contracted from soil polluted with human feces. It occurs especially in moist

²"I shall bring forward what I consider positive proof that this fever (typhoid) is produced by emanations from decaying organic matter; and I would therefore suggest for it the appellation of 'pythogenic fever'!" *Contributions to the Etiology of Continued Fever*, p. 219; *Medico-Chirurgical Transactions*, London, 1858, 41: 221.

sandy soils rather than on clay or rocky soils. This is due to the fact that hookworm larvæ soon dry up and die upon hard rocky or clay surfaces, whereas they find favorable conditions for development upon moist sand or loam. Under these conditions the larvæ develop as far as the second ecdysis, which have the power of penetrating the skin (see page 145).

Other Animal Parasites.—Many other animal parasites of man are deposited on the soil in the discharges and reinfest man during one of the stages of their cycle of development, after a varying journey, sometimes through an intermediate host. In the case of *Tenia solium*, for instance, man pollutes the soil with feces containing the eggs. Hogs devour this infection and return the disease to man. Soil pollution likewise results in the infection of cattle with the eggs of another tapeworm of man, *Tenia saginata*. Various parasitic protozoa have resistant stages that may remain in the soil for long periods of time following their introduction by fecal contamination.

Ascaris lumbricoides, the eelworm, is the commonest of the lesser intestinal parasites. It is a large nematode or round-worm, sometimes a foot and a half in length, while the diameter is about that of an ordinary lead pencil. The worms are found especially in children in all parts of the world. In the tropics most children harbor anywhere from two or three to several hundred. The worm lives in the intestinal tract, and the female passes a prodigious number of eggs which have a hard shell with a rough, warty appearance, often bile-stained. The embryo develops in the egg only after it is passed. Under favorable conditions, that is optimum temperature (33° C.), moisture and oxygen, the eggs develop in ten days to a month. Under unfavorable conditions of low temperature, dryness or insufficient oxygen, they may live in the soil as long as five years, so that soil once contaminated with these parasites may continue to serve as a source of infection for considerable periods of time. If fresh eggs are ingested, they pass through the intestinal tract without hatching. Man does not infect his neighbor directly. In other words, it is necessary that part of the life cycle occur in the outer world for these worms to continue their existence.

When the ripe eggs are ingested, the larvæ hatch and some of them penetrate the wall of the intestine and go to the liver and lungs and occasionally other organs. They may reach the liver as early as two days after experimental infection in rats, and the lungs on the third day. They pass from the blood-vessels into the air sacs and bronchial tubes of the lungs, and thence through the trachea to the mouth. Here most of them are swallowed. A serious and often fatal pneumonia in rats, pigs and other animals is produced by larvæ developed from eggs experimentally fed to them. It is now known that *Ascaris* larvæ may produce bronchitis and pneumonia in man. The mature worms occasionally creep forward into the throat and nose. Sometimes they wander into other organs through ducts leading from the intestine or into the body cavity through the intestinal wall, thus giving rise to serious abscesses. *Ascaris* does not pierce the skin as does the hookworm larva.

Santonin was for centuries the classical drug for expelling *Ascaris*, but

recently it has been found that oil of chenopodium is much more effective and dependable.

Ascaris depends mainly on soil infection with human feces, and its prevention corresponds to that of hookworm disease.

Trichuris trichiura, the whip-worm, next to hookworms and *Ascaris lumbricoides*, is the most common intestinal worm parasitic in man. It has a similar distribution and life history to the eelworm and is therefore primarily associated with soil pollution. Whip-worms are difficult to dislodge, probably due to their firm attachment. Oil of chenopodium appears to be the most effective remedy.

The following table³ gives a list of animal parasites having a relation to the soil during some part of their life history, and which may, therefore, be more or less associated with soil pollution. It will be noticed that for the most part protozoa and nematoda are contracted directly, whereas trematoda, cestoda and acanthocephala are contracted indirectly and through an intermediate host.

LIST OF ANIMAL PARASITES OF MAN WHICH MAY BE SPREAD BY SOIL POLLUTION

I	II	III
Transmitted: without Intermediate Host	Indirect: with Intermediate Host	Requires Further Study; Probably I or II as Marked
PROTOZOA		
37 <i>Endamœba* coli</i> 37 <i>Endamœba* gingivalis</i> 37 <i>Endamœba* histolytica</i> 37 <i>Endamœba* muris</i> 38 <i>Councilmania* lafleuri</i> 39 <i>Endolimax* nana</i> 40 <i>Iodamœba* williamsi</i> 41 <i>Caudamœba* sinensis</i> 42 <i>Dientamœba* fragilis</i> 48 <i>Vahlkampfia limex</i> 48a <i>Karyamorbina* falcata</i> 49 <i>Hartmanella hyalina</i> 50 <i>Sappinia diploidea</i> 52 <i>Craigia* hominis</i> 54 <i>Wasielewskia* gruberi</i> 56 <i>Trimastigamœba philippinensis</i> 69 <i>Chlamydomphrys stercorea</i> 72 <i>Leydenia gemmipara</i> 84a <i>Oicomonas</i> 84b <i>Pararhizomastix hominis</i>		

The numbers refer to the key-numbers in Stiles and Hassall's *Key-Catalogues to the Parasites of Man*, issued by the United States Public Health Service.

Asterisks indicate parasites reported for the United States; some of these imported. Italics, more important; Roman, less important

³ This table was prepared for me by Ch. Wardell Stiles.

LIST OF ANIMAL PARASITES OF MAN—*Continued*

I	II	III
Transmitted: without Intermediate Host	Indirect: with Intermediate Host	Requires Further Study; Probably I or II as Marked

PROTOZOA—*Continued*

108 Bodo		
109 Prowazekia		
111 Cercomonæ		
117 Diplocercomonæ		
118 Enteromonæ		
119 Embadomonæ * intestinalis		
123 <i>Chilomastix</i> * <i>mesnili</i>		
124 Tetrachilomastix		
126 Tetramitus		
128 Copromastix		
129 Tricercomonas		
131 <i>Trichomonæ</i> * <i>confusa</i>		
131 <i>Trichomonæ</i> * <i>vaginalis</i>		
136a Tetratrichomastix		
139 <i>Giardia</i> * <i>lamblia</i>		
161 Isospora		
163 Eimeria		
204 <i>Balantidium</i> * <i>coli</i>		

TREMATODA—FLUKES

246 <i>Fasciola</i> * <i>hepatica</i>	231 <i>Gastrodiscus hominis</i>	II
246 <i>Fasciola gigantica</i>		
248 <i>Fasciolopsis buski</i>	237 <i>Watsonius watsoni</i>	II
250 <i>Paragonimus</i> * <i>westermani</i>	255 <i>Echinostoma ilocanum</i>	II
267 <i>Opisthorchis felineus</i>	255 <i>Echinostoma malayanum</i>	II
268 <i>Clonorchis</i> * <i>sinensis</i>	260 <i>Artyfechinostomum sufrartyfex</i>	II
272 <i>Heterophyes heterophyes</i>	267 <i>Opisthorchis caninus</i>	II
272 <i>Heterophyes nocens</i>	267 <i>Opisthorchis noverca</i>	II
273 <i>Metagonimus yokogawai</i>	267 <i>Opisthorchis viverrini</i>	II
273a <i>Stamnosoma armatum</i>		
273a <i>Stamnosoma formosum</i>	277 <i>Dicrocoelium lanceatum</i>	II
281 <i>Schistosoma bovis</i>		
281 <i>Schistosoma</i> * <i>hæmatobium</i>		
281 <i>Schistosoma</i> * <i>japonicum</i>		
281 <i>Schistosoma</i> * <i>mansonii</i>		

LIST OF ANIMAL PARASITES—*Continued*

I	II	III
Transmitted: without Intermediate Host	Indirect: with Intermediate Host	Requires Further Study; Probably I or II as Marked

CESTODA—TAPEWORMS

314 <i>Hymenolepis</i> * <i>nana</i>	295 <i>Diphyllbothrium</i> * <i>latum</i>	291 <i>Braunia jassyensis</i> II
	295 <i>Diphyllbothrium cor-datum</i>	295 <i>Diphyllbothrium</i> * <i>parvum</i> II
	308 <i>Dipylidium</i> * <i>caninum</i>	296 <i>Diplogonoporus brauni</i> II
	314 <i>Hymenolepis</i> * <i>diminuta</i>	296 <i>Diplogonoporus grandis</i> II
	315 <i>Drepanidotea lanceolata</i>	305 <i>Davainea asiatica</i> II
	325b <i>Tænia</i> * <i>solium</i>	305 <i>Davainea formosana</i> II
	325d <i>Tænia</i> * <i>saginata</i>	305 <i>Davainea madagascariensis</i> II
		319 <i>Bertiella satyri</i> II
		325c <i>Tænia infantis</i> II
		325d <i>Tænia africana</i> II
		325d <i>Tænia</i> * <i>confusa</i> II
		325d <i>Tænia philippina</i> II

NEMATODA—ROUND-WORMS

337 <i>Anguillula</i> * <i>aceti</i>	462 <i>Gongylonema</i> * <i>hominis</i>	339 <i>Rhabditis fecalis</i> I
339 <i>Rhabditis pellio</i>	462 <i>Gongylonema</i> * <i>pulchrum</i>	339 <i>Rhabditis</i> * <i>hominis</i> I
355 <i>Strongyloides</i> * <i>stercoralis</i>		400 <i>Æsophagoetomum brumpti</i> I
370 <i>Trichuris</i> * <i>trichiura</i>		400 <i>Æsostephanostomum thomasi</i> I
387 <i>Ancylostoma braziliense</i>		403 <i>Ternidens deminutus</i> I
387 <i>Ancylostoma</i> * <i>duodenale</i>		407 <i>Syngamus kingi</i> I
387 <i>Ancylostoma malayanum</i>		414 <i>Trichostrongylus</i> * I
390 <i>Necator</i> * <i>americanus</i>		416 <i>Mecistoicrus</i> I
417 <i>Hæmonchus</i> * <i>contortus</i>		426 <i>Diocetophyia renale</i> II
481 <i>Ascaris</i> * <i>lumbricoides</i>		469 <i>Physaloptera caucasica</i> I
484 <i>Toxocara</i> * <i>canis</i>		469 <i>Physaloptera mordeus</i> I
484 <i>Toxocara</i> * <i>cati</i>		
486 <i>Toxascaris limbata</i>		
492 <i>Enterobius</i> * <i>vermicularis</i>		

ACANTHOCEPHALA—THORN-HEADED WORMS

	505 <i>Moniliformis</i> * <i>moniliformis</i>	
	508 <i>Macracanthorhynchus</i> * <i>hirundinaceus</i>	

Goiter.—Soil deficient in iodine is the cause of endemic goiter, for in such regions there is lack of this important element in water, in food, or both (see page 1042).

Tetanus.—Spores of the tetanus bacillus (*Clostridium tetani*) commonly occur in the soil of inhabited regions. They have been found not only in the

superficial layers, but sometimes at a depth of several feet. The normal habitat and the great reservoir of tetanus are the intestines of the herbivora. The spores are also contained in the intestinal discharges of man and other animals.

Tetanus increases as we approach the tropics, where puerperal tetanus and tetanus of the newborn are relatively frequent. Tetanus spores are much more abundant in certain localities than others. The cultivated soils of Flanders were the cause of many cases of tetanus during the World War. Certain parts of Long Island and New Jersey have become notable for the number of cases of tetanus caused by small wounds.

The tetanus bacillus probably does not grow and multiply in the soil. It cannot there find the necessary anaërobic conditions, temperature, and other factors necessary for multiplication. The remarkable resistance of the spores accounts for their persistence and accumulation in soil. They are especially numerous in manured soils.

The prevention of tetanus has been discussed on page 96.

Anthrax.—Like tetanus, anthrax does not grow in the soil under natural conditions. Its persistence is accounted for by its resistant endospore. Anthrax spores have been found in pastures where infected animals have been confined.

The anthrax bacillus requires oxygen in order to sporulate; the spores, therefore, do not form in the blood, and it is very important not to open the carcass of a sheep or cow dead of this disease before it is buried. The classic researches of Pasteur on anthrax should be studied in this connection. Pasteur examined the field where animals dead of anthrax had been buried twelve years previously. He found the specific bacillus in the soil and demonstrated its virulence by inoculations into guinea-pigs. Pasteur thought that the spores were brought to the surface of the soil by earthworms, and proved the possibility of this by sowing virulent cultures in soil and recovering the bacillus from worm casts. It seems, however, in the light of subsequent investigations that the danger from this source is slight, so that anthrax, with a few exceptions, can hardly be called a soil infection. This is the case at least with man, for there is no instance on record in which human anthrax has been contracted from contact with the soil.

Botulism.—Spores of *Clostridium botulinum* are found almost everywhere in the soil and thence get into our food. The relationship to the soil is indirect (see page 657).

Malignant Edema.—The bacillus of malignant edema is found in the superficial layers of the soil. It is very widely distributed. This organism is also found in putrefying substances, in foul water, and in the intestinal tract of various animals. In 1877, Pasteur first recognized an organism belonging to this group by injecting animals with putrefying liquids. He called the organism the *vibrion septique*, recognized its anaërobic nature, but did not obtain it in pure culture. Koch and Gaffky, in 1881, studied it carefully and renamed it the bacillus of malignant edema. The bacillus has lateral flagella, an oval spore, and is a strict anaërobe. It is very pathogenic for almost all

animals, causing extensive hemorrhagic edema without the production of gas, which distinguishes it from the gas bacillus of Welch. Wound infections with malignant edema occur, especially with deep punctured or lacerated wounds, which favor anaërobic growth. Before the days of antiseptics this complication was frequent, especially during wars.

Bacillus Welchii (also known as Welch's gas bacillus, *B. aërogenes capsulatus* and *B. perfringens*).—This organism is a member of a populous and widely distributed group of bacteria, which have in common the ability to ferment sugars with the production of butyric acid. *B. welchii* is a large rod, Gram-positive, and usually grows singly or in pairs. Spore formation is inconstant and occurs only in alkaline media, never in pure cultures in media containing a fermentable sugar or free acid. It causes stormy fermentation of milk; that is, the milk is quickly coagulated and gas formation is so abundant as to break up the curd and even to force parts of it above the cream ring. A rabbit injected intravenously with this organism, killed within two or three minutes and incubated, presents in twenty-four hours a body enormously distended with gas which will burn with a pale blue flame.⁴

This bacillus is found in the intestinal canal of man and animals, in soil and dust which distributes it widely. When introduced in wounds it causes a serious infection with the production of gas. In the World War many wounds contaminated with the soil of the trenches were complicated with this infection.

Many other microorganisms, especially those belonging to the hemorrhagic septicemic group, occur in the soil and occasionally complicate wounds.

Intestinal Bacterial Infections.—*Typhoid Fever*.—There was a widespread belief, even among sanitarians, that this disease is frequently connected with soil pollution. This belief was given scientific recognition by Pettenkofer, who believed that the gases or effluvia produced rise, and in some way were capable of provoking disease. Pettenkofer's mistake concerning typhoid in relation to the height of the ground water has already been mentioned.

Typhoid bacilli frequently find their way upon and into the soil along with human excreta. Multiplication, however, rarely, if ever, takes place there. As a rule, they die quickly; they may possibly live a month under unusual circumstances. When frozen they may live and remain virulent for several months, as in the case of the Plymouth episode and the New Haven epidemic. While typhoid fever in cities and towns has no evident direct relation to soil pollution, it is possible to conceive an indirect relation in many cases, especially in camps and in rural districts.

There are numerous ways by which typhoid bacilli may be returned from the soil to the mouth of a susceptible person. It is possible, though not likely, for this to occur directly. So far as typhoid is concerned, perhaps the greatest danger from a polluted soil consists in infection of the drinking water. The ways in which it may occur are discussed in the chapter on

⁴"Studies in *Bacillus welchii*, with Special Reference to Classification and to Its Relation to Diarrhea," J. P. Simonds, *Monogr. Rockefeller Inst.*, No. 5, Sept. 27, 1915.

water. The transfer of typhoid bacilli from the soil to the mouth may also occur mechanically by means of flies, dust, and dirt. Vegetables grown in a polluted soil may carry typhoid bacilli to the very tips of their leaves.

The pollution of soil with human feces is always a danger and should be prevented. The worst offense in this particular occurs in country districts, where the potential danger is greater than in the city.

Cholera.—There is every reason to believe that the cholera vibrio dies quickly when deposited upon or in the soil under natural conditions. The cholera vibrio may be transferred from the soil to the mouth in the ways mentioned above in the case of typhoid. Formerly cholera was believed to be associated with polluted soils, but it now appears that the disease is rarely contracted from the soil, and that the physical and chemical conditions of the ground play little, if any, rôle in the epidemiology of this disease.

Bacillary dysentery has not been traced to soil pollution, but there is a suspicion that *amebic dysentery* may in some way be thus connected.

Tuberculosis and Other Diseases.—In 1862 H. I. Bowditch formulated the law of soil moisture from studies which seemed to indicate that tuberculosis was more common in Massachusetts over moist soils than dry ones. If there is any connection between tuberculosis and the soil, the relation must be indirect. Exposure to cold and damp depresses vitality and lowers resistance to tuberculosis. It does not necessarily follow that habitations or workshops are cold and damp because the ground on which these houses are built is wet and cold.

Dampness and cold may favor rheumatic and neuralgic conditions, and also predispose to respiratory infections. In this way association with a cold, damp soil may be prejudicial to health. Clay soils are apt to be damp; sand and gravel soils are readily drained and may be kept dry by means of simple devices. Such soils, therefore, make the best building sites for habitations. As a rule, the foundation of a house should be at least two or three feet above the level of the ground water.

The soil greatly influences the character of the water which rests upon it and which passes through it. This is discussed in the section on water.

SECTION IX

WATER

CHAPTER I

GENERAL CONSIDERATIONS

Water is a prime necessity of life, not only as an article of diet, but also for the proper cleanliness of person, clothing, and things. In centers of population it is essential further for manufacturing, for the conveyance of waste, and for protection against the ravages of fire.

Though water is not technically classed as a food, it is an essential article of diet. In nature water comes in contact with many surfaces and substances and, therefore, is particularly liable to contain impurities. Pure water is a chemical curiosity; it does not exist in nature. By virtue of its marked erosive and solvent properties, all water in nature contains impurities, in solution and in suspension. Some of these are organic and some are inorganic. They consist of various gases, liquids, and solid substances. Water may contain many bacteria and is a frequent medium for the transmission of infection.

From the remotest antiquity the highest value has been placed upon an abundant and pure water supply. Centers of populations sprang up in ancient times around those points where it was most readily available, and great expenditures of labor and treasure were made to carry it to places where it was not naturally plentiful.¹

It is interesting to note that the number of towns in this country before 1800 having a public water supply was only 16, supplying about 2.8 per cent of the population existing at that time. In 1850, there were only 83 public waterworks, supplying about 10.6 per cent of the census population. In 1897, the total number was 3,196, supplying about 41.6 per cent of the population.² In 1915, there were 4,872 publicly and privately owned waterworks.³ In 1924, there were approximately 9,850 cities and towns with waterworks systems in the United States exclusive of its possessions; of these 2,800 were privately owned, and served from 10 to 15 per cent of the total population supplied. The water systems of practically all our larger cities are publicly owned

¹ The date of construction of the Appian aqueduct carrying water to Rome is placed at 312 B. C. Eighteen other aqueducts were constructed at various times until A. D. 226. The one commenced by Emperor Caius and completed by Claudius, according to Pliny, cost 350,000,000 sesterces, or about \$12,700,000.

² M. N. Baker, *Manual of American Water Works*, 1891 and 1897.

³ *McGraw Waterworks Directory*, 1915.

and operated. Between 85 and 90 per cent of the total population in these places was supplied by publicly owned works.⁴

CLASSIFICATION OF WATER

From a sanitary standpoint water is either good or bad, according to whether it has or has not the quality of wholesomeness. Commonly water is spoken of as pure or impure. These simple classifications, however, are not tenable, for it is not possible in the present stage of our knowledge to draw such a sharp line of distinction. It is seldom possible to detect infection directly in water. The likelihood of its existence may be inferred from the degree of contamination. *Contamination* is the introduction into water of human or animal wastes, which may transfer infection, or of other substances, as chemical poisons, which may also render it unwholesome. *Pollution* is a term used primarily to denote impairment of physical attractiveness. Pollution is the introduction into water of substances of such character and in such quantity that they render it objectionable in appearance, taste, or odor. They may or may not contribute to contamination.

For practical purposes, then, water is classified as good, polluted, or contaminated. (1) *A good water* is one which is at all times free from contamination and safe for human consumption, as determined by laboratory analyses, sanitary survey, and continued use. It is also one which is attractive in its appeal to the senses. (2) *A polluted water* is one which has suffered impairment of physical qualities through the addition of substances causing turbidity, color, odor, or taste. (3) *A contaminated water* is one which carries potential infection by reason of the addition of human or animal wastes or which has been rendered unwholesome by poisonous chemical compounds.

Many cities in Europe have a double water supply with faucets plainly labeled "potable" or "non-potable," the first being suitable for drinking, cooking purposes, and personal use, while the second is intended for miscellaneous household and industrial uses.

According to source, waters are considered under three classes, viz., rain water, surface water, or ground water. That which collects in streams, lakes, ponds, and reservoirs is surface water, and that which percolates through the soil and collects in underground places is ground water.

PROPERTIES OF WATER

Water is a remarkable substance with many unique properties. It is a clear, transparent, tasteless, and odorless fluid; colorless in small quantities; pale blue through a deep column. It freezes at 0° C. and boils at 100° C. under a barometric pressure of 760 millimeters. It is practically incompressible; it has its greatest density at 4° C.

Water is the most widely distributed substance. The hardest crystals and

⁴American Water Works Association, *Manual of American Water Works Practice*, 1925.

the driest rocks contain appreciable quantities; in fact, crystals could not form were it not for the action of water.

Practically all substances yield to water; it is the most universal solvent known. It dissolves gases; in fact, one of the most important constituents of all natural waters is carbon dioxid. Carbon dioxid is always present in the air, and all rain waters contain some of it. Still more is taken up by the water as it percolates through ground covered with vegetation. The presence of this gas increases the solvent powers of the water, enabling it especially to dissolve limestone and other mineral and organic substances.

Water rises to a greater height in a capillary tube than any other substance. Owing to this property it clings very tight in the soil and rises to a considerable height, thus making vast areas of the earth arable instead of dry desert.

The difference in the amount of water vapor the air can hold at varying temperatures is far greater than the difference in the amount of vapor of any other substance that the air can hold. For example, air containing 75 per cent of saturation of water vapor at 50° F. will supersaturate (150 per cent) at 32° F. The excess precipitates as rain.

Water is unique in many of its properties. It does not boil or freeze at the expected temperatures. It has high specific heat and remarkable capillarity; its greatest density is above the freezing point. It exhibits special properties in its gaseous form. Many of these peculiarities are explained on the assumption that the molecule of water consists of aggregates of H_2O .

Mobility and Circulation.—The mobility of water permits it to circulate in both the inorganic and organic kingdoms. This property constitutes one of the most fundamental and important activities of the world in which we live. Both water and carbon dioxid circulate rapidly and penetrate everywhere, and they cling when they get there; it is owing to this fact that vegetation is widespread and organic activity intense on the earth. The mobility of water and carbon dioxid, which are the principal foods of plants, makes it possible for vegetation to cover the land even to the mountain tops. The circulation of water combined with other properties, such as evaporation, latent heat, etc., influences climate and health.

THE USES OF WATER IN THE BODY

Water is the principal constituent of our bodies, the principal substance that enters our bodies, and the principal substance that leaves our bodies. It is almost exclusively in solution that things can penetrate into our body and the waste products are turned out mainly in solution. There is a good deal of resemblance between the salts in our blood and in sea water. "Our blood is, so to speak, descended from sea water."

As a rule, water is not considered a food, for it has little or no value when estimated as a force producer within the body. Much of the water which is either drunk or ingested as a part of other foods passes through the body

unchanged, although a little of it is undoubtedly altered or split up into elements which unite with other compounds. The nature of these processes is obscure, and as yet very little understood. Water is entitled to rank as a food because it enters into the structural composition of all foods as well as all the tissues of the body; it is an essential element of diet, even though it does not of itself build tissue, repair waste, or produce heat or energy.

Water composes about 70 per cent of the entire body weight, and its importance to the system, therefore, cannot be overrated. The elasticity or pliability of muscles, cartilages, tendons, and even bones is in a great part due to the water which these tissues contain. "The cells of the body are aquatic in their habits." The amount of water required by a healthy man in twenty-four hours is, on the average, between 1,800 and 2,100 cubic centimeters, besides about 600 cubic centimeters taken in as an ingredient of solid foods,⁵ thus making a total of 2,400 to 2,700 cubic centimeters. Twenty-eight per cent of the water lost from the body takes place through the skin, 20 per cent through the lungs, 50 per cent through the kidneys, and 2 per cent through other secretions and the feces.

The use of water in the body may be summarized as follows: It enters into chemical composition of the tissues; it forms the chief ingredient of all the fluids of the body and maintains their proper degree of dilution, and thus favors metabolism; by moistening various surfaces of the body, such as mucous and serous membranes, it prevents friction; it furnishes in the blood and lymph a fluid medium by which food may be taken to remote parts of the body and the waste material removed, thus promoting rapid tissue changes; it serves as a distributor of body heat; it regulates the body temperature by the physical process of absorption and evaporation.

One of the most universal dietetic faults is neglect to take enough water into the system. Water may be taken with meals, but never to wash down food; in other words, water must not be used to take the place of thorough mastication.

THE AMOUNT OF WATER USED AND WASTED

From a sanitary standpoint our aim should be to encourage a generous use of water, but to discourage waste. The conservation of pure water and the economic use of a purified water are pressing problems that a growing and expanding country must meet and solve as a matter of self-interest if not of self-preservation.

It is possible to get along with a surprisingly small amount of water. Thresh found that in a number of country places the amount used in cottages did not greatly exceed one gallon per person per day. This is not sufficient for modern requirements of cleanliness and health. On the other hand, where the supply is abundant and easy of access large quantities of water are heedlessly wasted.

⁵ Fully five-sixths of the food in an ordinary diet consists of water.

The average amount of water required per capita for domestic purposes is usually stated at about seventeen gallons a day. Rankine considers ten gallons sufficient. Parkes found that the average amount used by a man in the middle class, who may be taken as a fair type of a cleanly man belonging to a fairly clean household, is twelve gallons per day. This includes the amount used in cooking, drinking, ablution, utensil and house washing, and laundry. Davies' estimate of seventeen gallons a day is divided as follows:

Drinking, 3 pints; cooking, 5 pints.....	1 gallon
Ablution (including sponge bath, 2½ gallons)	5 gallons
Washing (laundry, 3; house, etc., 3).....	6 gallons
Water-closets	5 gallons
<hr/>	
	17 gallons

The actual per capita daily consumption of water in some cities is, in fact, not much above this figure. Thus, Manchester uses twenty imperial gallons and Berlin twenty-five gallons a day for each individual. Some small English towns, as Saffron Walden (population 6,108), use eleven gallons per capita per day, and Melrose (population 1,300) uses thirteen gallons. As a contrast to these low figures most cities in America are furnished with an extravagant quantity—Pittsburgh, two hundred United States gallons per capita daily, Buffalo, 213, Philadelphia, 168, Washington, 132. The small amount of water used by some European cities is not an ideal to strive for under American conditions. The European figures are steadily increasing, even where all water is sold by meter. In towns having a metered supply the per capita consumption varies from 6.6 imperial gallons daily for the lowest class of dwellings to fifty-nine gallons for the highest class of dwellings.

The following tables give the per capita consumption in some American cities, contrasted with similar figures abroad: *

WATER CONSUMPTION IN LARGE AMERICAN CITIES *

(Arranged in order of number of services.)

City	Gallons per Capita per Day	Per Cent Service Metered
New York, New York.....	131	26
Chicago, Illinois	275	10
Baltimore, Maryland	132	17
Boston, Massachusetts	113	89
Buffalo, New York	213	15
Washington, D. C.	132	87
San Francisco, California	81	100
New Orleans, Louisiana	100	100
Seattle, Washington	105	100
Rochester, New York	84	99
Atlanta, Georgia	95	100
Memphis, Tennessee	70	100
Springfield, Massachusetts	91	100

* *Manual of American Water Works Practice*, 1925.

* These figures include industrial uses.

WATER CONSUMPTION IN FOREIGN CITIES

City	Year	Consumption United States Gallons per Capita per Day
London	1924	45
Paris	1911	38
Berlin	1911	25
Vienna	1914	37
Rotterdam	1919	29
Zurich	1918	63

The amount of water expressed by the per capita consumption of a community is very misleading for purposes of comparison. The figures are often obtained by dividing the total theoretical amount of water pumped, by the population. The result, therefore, does not take into account many factors, for the actual amount of water pumped may not equal the theoretical possibilities; corrections for slip and other factors should be made. The figures also do not take into account the amount of water lost through broken pipes, leaky joints, etc. It is estimated that in some places almost half the water pumped is wasted in this way. In a well-maintained system the lost or unaccounted for water should not exceed 10 per cent of the volume delivered to the system. Further, there are great discrepancies when contrasting different cities in the amount of water used for business purposes. The amount of water used in trades and manufactures varies enormously. Certain industries, such as tanneries, paper mills, breweries, wool scouring, etc., require great quantities. Therefore, unless the per capita consumption is based upon the amount of water actually measured by meter for domestic purposes, the figures of one city cannot be properly compared with those of another.

Causes of Water Waste.—Few persons realize the immense amount of water that is wasted in almost every town. Taking it right through, probably one-half of the water supply of American cities is wasted. While some of this is unavoidable, the greater part of it could be stopped. It is necessary to allow a liberal supply, but there is no sanitary advantage in waste. Good clean water in large quantities is difficult to obtain and expensive. Economy and avoidance of waste are, therefore, essential.

There are three principal causes of this waste: (1) leakage from faulty mains and service pipes; (2) waste from defective house fittings; (3) waste resulting from an unmetered or unmeasured service. The first cause includes leaks from faulty mains and service pipes and all other hidden defects where the water escapes unperceived into drains and sewers or into the subsoil. It is possible to check a large part of this waste by the use of instruments known as detectors. With these instruments leaks may be located and defective taps and open stopcocks discovered.

It requires but a moment's calculation to figure out the great number of gallons wasted by forgetting to close a stopcock. In some cities, such as Wash-

ington, in the winter time the water in many houses is allowed to run continuously from the cold water faucet, in order to prevent freezing.⁷ The waste from this cause is enormous, and may be corrected by properly placing the service pipes so as to avoid the danger of freezing.

Water Meters.—It has been the universal experience that much water is thoughtlessly wasted where the service is not metered. The only objection to a metered service is the prejudice common to all innovations, but the advantages are soon realized and the saving is very considerable. The introduction of meters in the city of Washington has resulted in checking the waste by reducing the total amount of water consumed one-third, making a saving of from twenty to thirty million gallons of water a day without annoyance or inconvenience to any one. This great saving did not all result from the metering alone, but was aided by the use of detectors and an efficient system of inspection, which checked waste from other causes. In Milwaukee, before meters were generally adopted, the water used per tap was 1,781 gallons per day. After the majority of houses were furnished with meters, the amount used per tap was only 644 gallons. Buffalo reduced her water consumption during the war (1918) from over 300 gallons per capita to 260 with a survey that covered only part of the city. Another notable instance of checking waste was furnished by Liverpool, where the average amount supplied daily per head was 33.5 imperial gallons. Deacons' water waste detectors were introduced, and these, together with efficient inspection, reduced the supply to twenty-three gallons without any restrictions being placed upon the consumers. The good effect of measures for the conservation of water are apt to be temporary, owing to a careless return to waste, unless combatted by continued efforts of the water department to obtain the coöperation of water consumers.

DUAL WATER SUPPLY

The question of a dual supply of water, one cheap for general purposes and the other high class for personal use, has often engaged the attention of engineers and sanitarians. Ancient Rome had a sort of double supply, and Paris and other European cities have it at present. The advantages and disadvantages of the double system are evident. Even where the community served is intelligent and careful, the danger of a double system is very great, and it will probably never be resorted to except through stress of circumstances.

A number of cities have a separate water system under high pressure for fire fighting. Auxiliary water systems are often connected with the drinking water mains, and controlled by means of check valves, by-passes, etc. This arrangement is hazardous and a number of outbreaks of typhoid fever have been traced to faulty valves or failure to close by-passes in such a dual system.

⁷ In cities where this practice prevails, more water is used in the winter time than in the summer months.

Twenty outbreaks due to these causes have been reported in the United States in recent years.

SOURCES OF WATER

We may begin the circle by considering that all water comes to us from the aqueous vapor condensed in the form of rain or snow. Of this a certain amount returns to the atmosphere by evaporation; the rest collects upon the surface of the earth or soaks into the ground. Some of it flows off in the direction of surface slope to join the ponds, lakes, rivers, or seas, or some of it may penetrate the earth to variable depths. The sources of our water supply may, therefore, be classified as: (1) rain or snow water, (2) surface water, including ponds, lakes, streams, and rivers, and (3) ground water, including springs and wells. This classification is evidently an arbitrary one, used for convenience. There is no sharp line of demarcation between rain, surface, and ground water. Rain water soon becomes surface water, and surface water quickly passes into the ground; the ground water frequently reappears as springs to form streams and lakes and other surface supplies.

Rain water is nominally the purest, but is liable to irregularity of composition, and in built-up sections it is difficult to collect it so as to be free from pollution and fit for drinking. Surface water from inhabited watersheds is, in its raw condition, never entirely safe for drinking purposes. Ground water obtained from the subsoil of a catchment area, free from sources of pollution, is usually of a satisfactory character. Artesian water, which is ground water obtained from the deeper underlying strata, is often so rich in mineral matters that it is unsatisfactory for most uses. The various sources of pollution, its character, and dangers will be considered in subsequent pages.

RAIN WATER

Rain water is really "distilled water"; that is, it is water that has been vaporized and then condensed. The process of distillation is one of the best known methods for purifying liquids of all kinds. All the non-volatile substances are left behind; theoretically, therefore, rain water should approach nearer to absolute purity than any other kind of natural water. However, it receives impurities from the moment it condenses, for each droplet of mist is formed about a particle of dust in the air. The rain drop further absorbs gases, and as it drops through the air collects a large amount of the "dirt" floating in the lower portions of the atmosphere. It is a common observation that a shower will wash the air so that it becomes beautifully clear and clean. The impurities collected by the rain before it reaches the surface of the earth, while considerable in amount, are practically negligible from a sanitary standpoint. After rain touches the earth's surface it becomes a surface water. If collected before touching the ground from a clean, impervious surface in the open country, it is the purest of natural waters. The use of rain water for drinking purposes has met with little favor by sanitarians, despite its

exceptional purity, because it is so frequently collected and stored in such a careless manner that it is subject to impurities. It is true that rain water is not likely to be contaminated with sewage, nevertheless some of the dirtiest waters used for domestic purposes come from rain water tanks.

Because rain water is soft it recommends itself for use in the laundry, and the absence of lime salts renders it desirable for cooking. On the whole,

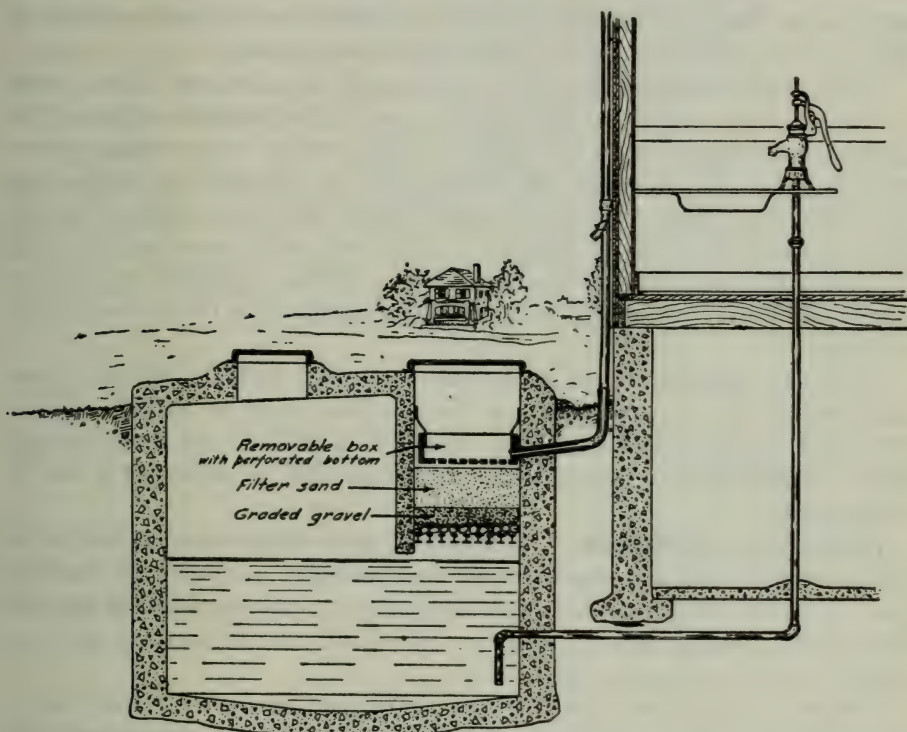


FIG. 82.—PROPERLY CONSTRUCTED CISTERN WITH SAND FILTER, located outside the house to protect rain water.

(From *Water Supplies and Sewerage Systems for Farm Residences*, Bd. Health Minn., March, 1925.)

however, it is not considered as practicable as a good ground or surface water for general domestic supply.

The storage of rain water in cisterns and containers about the house was the principal factor in keeping yellow fever alive in endemic foci. The yellow fever mosquito (*Aedes egypti*) breeds by preference in artificial containers holding rain water. It was the abolition of such breeding places that has protected Philadelphia, Boston, Havana, New Orleans, and many other seaports from yellow fever epidemics (see page 294).

Usually it is advisable to filter rain water collected from the roofs of buildings, especially if situated in towns, near dusty roads, etc. Filters for this purpose should be located accessibly, be constructed of coarse sand or other

proper materials and receive regular attention, else they are liable to become inefficient and filthy.

Amount.—The average annual rainfall on the globe is computed to be thirty-three inches. The mean annual rainfall for different portions of the United States has been tabulated by the United States Weather Bureau to average some thirty inches. In New England and the Middle States it amounts to forty inches. In Assam from 600 to 805 inches have been recorded, while in the Sahara desert, part of Arabia, the desert of Gobi, and portions of Mexico, Chile, and Peru it has seldom been known to rain.

The amount of water given by rain can easily be calculated if two points are known—the mass of rainfall and the area of the receiving surface. The amount is determined by a rain gauge and the area of the receiving surface must be measured. Roughly, the amount may be calculated by multiplying the area of the receiving surface in square feet by half ⁸ the rainfall in inches, the result being in gallons. Thus, one inch of rain on a house roof 20 × 20 feet area would be about two hundred gallons. With a rainfall of forty inches per annum this would amount to eight thousand gallons, or twenty-two gallons per day.

The total theoretical amount, however, is never available, for the reason that some is lost by evaporation and the first flow should be wasted. Only a very small proportion of water may be collected from a light shower spread over a considerable interval, especially in hot weather, as nearly all is lost by evaporation.

Collection and Storage.—The points of prime importance in the collection and storage of rain water for domestic purposes are: (1) the material and care of the surface upon which it is caught; (2) the separation of the first flow, which contains most of the gross impurities; (3) the location and construction of the storage cistern.

Storage cisterns for collecting rain water are frequently placed underground. In some places rain water cisterns are built of cypress wood and always above ground. Tanks of wood serve their purpose well, provided they be kept full. If there is great fluctuation in the water line the tank itself falls out of repair. Rain water attacks iron, lead, zinc, and other metals, and when metal cisterns are used the metal should be coated with a good asphaltum paint. This applies also to the delivery pipe. Under no circumstances should lead cisterns or lead service pipes carry rain water used for drinking purposes. It should not be forgotten that cisterns are liable to pollution, and require frequent inspection and cleansing.

Where the overflow pipes from rain water tanks are connected with sewage drains, precautions must be taken to prevent sewage backing up and entering the tank.

Composition.—Rain water varies in composition with the purity of the atmosphere through which it has passed. It always contains dissolved gases,

⁸ Six-tenths is a more accurate factor, but as about 10 per cent is lost, one-half gives more nearly the actual amount recovered.

an average of twenty-five cubic centimeters per liter. These gases are mainly nitrogen, oxygen, and carbon dioxid, taken up in proportion to the amount contained in the atmosphere. In addition ammonia is very commonly present. The amount of total solids varies; throughout England it averages 0.39 part per million. The principal inorganic constituents of rain water are sodium chlorid; nitric acid and nitrates, sulphuric acids and sulphate; a small quantity of nitrogenous organic matter is also present. The sodium chlorid comes mostly from the sea spray lifted into the atmosphere through wind action. The sulphuric acid comes largely from the waste products of burning coal. Rain water is soft on account of the absence of the alkaline earths, and is almost always acid in reaction. It has a mawkish taste.

Bacteria.—Rain water contains a variable number of bacteria and other microörganisms, the number and kind depending upon the germ population of the atmosphere through which the rain passes. Fortunately the various micro-organisms floating in the air and carried down mostly by the first shower are not of serious moment, as far as health is concerned. Pathogenic micro-organisms in the air are few in number, and these are soon killed by desiccation or the germicidal action of the direct sunlight, to which they are so thoroughly exposed.

Miquel, at the Montsouris Observatory in Paris, found rain water to contain bacteria, pollen, spores of fungi, protococci, etc.; these were especially numerous in the warmer months. In the first showers after a long spell of dry weather over 100,000 such organisms may occur in a pint. Miquel obtained an average of four bacteria per cubic centimeter of rain water in the country and nineteen per cubic centimeter in Paris.

SURFACE WATERS

Surface waters include rivers, creeks, and smaller streams, large and small lakes, ponds, and impounding reservoirs, all resting upon the bosom of the earth in contact with the atmosphere. Surface waters vary greatly in composition, depending largely upon the character of the catchment basin. A water flowing over an uninhabited rocky soil or through deep layers of sand and gravel is more likely to be free of organic impurities than one that is drained over loam or has stood in swamps.

From the way in which surface waters are exposed they are subject to impurities, and from a sanitary standpoint are frequently dangerous and almost always open to suspicion. Most cities, especially in America, depend upon surface waters for their supply. This is usually taken from rivers, lakes, or impounding reservoirs. It is scarcely possible, in a populous country, to obtain a large quantity of surface water free from pollution with human wastes. Sanitarians have, therefore, more and more come to the conclusion that, while surface waters used for drinking purposes should be protected from con-

tamination, if this is not practicable, they should be purified before they are used.

Rivers.—Streams are the natural sewers of the regions they drain, and, when used as a source of water supply, we have established a direct connection between the alimentary canals of the people living upstream with the mouths of those below. Most of our large rivers flow through more than one state; therefore, the interstate pollution of streams becomes a national problem. In some of the older countries of Europe, with more centralized power, laws to prevent the pollution of streams have long been enforced. In our country where the rivers furnish the chief source of water supplies for most of our large cities, laws to prevent pollution are now in successful operation.

Stream Pollution versus Water Purification.—The succession of cities and the combined use of the river as a sewer and source of water supply on such rivers as the Merrimack, Hudson, Delaware, Ohio, Missouri, and Mississippi are particularly impressive. The use of the waters of these rivers, raw and unpurified, has caused much unnecessary sickness and has cost thousands of lives.

No stream draining an inhabited region can be considered safe without some method of purification, even though the sewage flowing into the stream is treated and all reasonable precautions taken in connection with it. There are storm overflows and street wash that cannot pass through sewers and other sources of pollution which cannot be controlled.

Looking at the whole matter of stream pollution solely as an economic engineering problem, it is cheaper to purify the water supplies taken from the rivers than to purify the sewage before it is discharged into them. The volume to be handled is less and the cost of purifying water per million gallons is much less than the cost of purifying sewage. Further, in the present state of our knowledge water may be purified more effectively and with greater certainty than sewage. On the other hand, it is perfectly clear to the sanitarian that both methods are essential, that is, a reasonable protection of our streams against pollution and also purification of the water served to cities.

Composition.—The composition of river water varies greatly from source to mouth and often from bank to bank. This complexity is due to the mixture of surface and ground water, each of which contributes a variety of impurities. Ground water, as a rule, contains more mineral matter and is harder than surface water by virtue of its longer contact with rock materials. It is also liable to hold in solution large amounts of carbon dioxide. Surface water contributes organic matter to the stream from the soil, swamps, sewage, and industrial wastes; also some dissolved mineral substances; suspended particles of clay, soil, and waste; bacteria and other microscopic forms of life. In general, the mineral content of rivers is dependent upon the geological formations, the organic content upon the density of population.

Rivers are usually less polluted near their source, principally for the reason that head waters are found in uninhabited regions. Such pollution as is found is more liable to be due to natural causes, such as the leaching of swamps and the erosion of soft materials by rapidly flowing water.

The amount of impurities increases as we descend the stream. Population centers tend to increase in size and number as the stream grows in its flow seaward, and, in as much as the rivers are the natural drainage channels for all the wastes of human life and industry, pollution increases more rapidly than the dilution of the stream and more rapidly than it can be disposed of by natural forces of purification.

In broad, slow-moving streams great inequalities in composition are often found through the cross section. These inequalities persist below a point at which tributaries enter or large volumes of waste discharge. The effect upon the average composition of the river water may be either one of dilution or concentration.

Sudden and great changes in the character of river water occur at times of heavy rainfall. In the average stream this dilutes the dissolved mineral constituents, but increases the amount of organic substances, suspended solids, and bacteria. In a stream already heavily polluted a flood may cause dilution and relative decrease of all constituents.

General changes, extending over long periods of time and slow in their operation, which come with increasing population and greater pollution, often occur. The sanitary significance of such changes is of great importance for they measure a retrogression in quality, a trend toward incipient nuisance, and a sanitary menace.

Stream Flow and Water-borne Disease.—Frequent attempts have been made to correlate the flow of streams and the stages of a river with outbreaks of disease, especially typhoid fever. Typhoid may be, and usually is, independent of the stage of the river. Such correlation as appears to exist at high stages is due to the fact that outbreaks are often connected with sudden freshets, following a long dry spell. At such times the flow is rapid, infective material is quickly carried for long distances, and nature's forces have not time to destroy pathogenic bacteria in water. In other words, it is the rapidity of flow, or the time consumed for the quick transfer of fresh infection, rather than the stage of the river, that is most often responsible for water-borne epidemics of typhoid fever. During the spring and fall freshets when the water is cold this danger is greater.

The same infective material, if discharged at the same point at a low stage of water, might be rendered harmless by natural forces of purification because the elapsed time in transit would be much greater. On the other hand, if discharged at a point nearer the consumer's tap, during either low or high water, it would survive and cause disastrous results. Heavily contaminated streams are dangerous at all stages of water; those showing slight or occasional contamination may be hazardous only at high stages because the protective barrier of time has been eliminated.

Lakes and Ponds.—Lake water is apt to be soft and free from serious organic impurities. Fresh water lakes and ponds make admirable sources of water supply when kept free from pollution with the wastes of human life. This is much more practical than in the case of rivers, on account of the limited area of the catchment basin directly draining into a small lake or pond. In large lakes the dilution of accidental contamination is enormous, and the effects of time, storage, sedimentation, and other purifying factors have a good chance to exert their protecting influence. The sanitary problem in connection with large bodies of fresh water, such as our Great Lakes, is quite different from that of small lakes and ponds.

The Great Lakes.—The lake cities suffer most from the mingling of their own sewage with their own water supplies. This is avoided in part by building the intakes farther out into the lake or by placing the intakes in deep water at points where there seem to be fairly definite currents, bringing fresh, clear water from the body of the lake to the intake. The currents are never constant, being controlled by the wind, hence safety cannot always be secured in this way. Almost every lake city has at one time or another suffered from outbreaks of typhoid fever. Chicago has cut a drainage canal to keep her sewage from entering the lake, so that it now flows through tributaries to the Mississippi River. This sanitary reform cost the city of Chicago upward of \$40,000,000, and it eliminated the sewage of a large part of the city, but not including certain areas of Evanston and the north side. Despite this commendable piece of sanitary engineering designed to keep the water clean, Chicago disinfects its water supply with liquid chlorin. Plans have now been projected to care for the sewage of those areas not included in the original work.

Hazen points out that in the smaller cities upon the lakes the mingling of the sewage and water may be relatively just as important as in the larger ones. They have less money to spend, their intakes do not go out so far, their sewers are apt to discharge at the nearest point, sometimes directly in front of the waterworks intake. The water may be shallow and stirred by the wind to the bottom, and, in short, "Menominee's sewage in Menominee's water may be just as bad as Chicago's sewage in Chicago water."

The Great Lakes are so large, the flow so slow, and the dilution and time intervals and exposure to sun and air are so great that there is practically no chance of infection being carried from one of the great cities to another. Thus, Chicago sewage would scarcely endanger the purity of Detroit's water supply, even with no drainage canal. The little city of St. Clair, with 2,543 inhabitants, only forty-five miles away, is far more dangerous to Detroit. In the same way Detroit's sewage is probably harmless at Cleveland, and Cleveland sewage is harmless at Buffalo. The sewage of Buffalo, however, is a great menace to those drinking the water at Niagara Falls.

Pollution may travel a variable distance in large lakes. At the mouth of the Detroit River, for instance, serious pollution was shown, extending normally more than ten miles into the lake, and at other places sewage pollution

was shown, extending as far as eighteen miles from the shore.⁹ The pollution from boats passing near the intake may also be a serious menace.

Most of the cities on our Great Lakes find it impracticable to extend water pipes into zones of pure water on account of the great cost of these extensions and the engineering difficulties involved in placing intakes beyond a seventy-foot depth. Therefore, in most instances, our lake cities cannot obtain a safe water supply without purification.

Impounding Reservoirs.—Impounding reservoirs are artificial ponds or lakes, usually made by throwing a dam across a narrow valley. Most impounding reservoirs are made along the course of a small stream. The principal use of impounding reservoirs is to hold the excess water of the winter and spring flows in store for the summer and fall. At the same time, the quality of the water is equalized and improved by a mixture of the wet and dry weather flows. Time and relative quiescence permit the sedimentation of suspended particles, the coagulation and precipitation of colloidal coloring matter, and the destruction of objectionable aliens among the bacteria. Advantage is taken of the great sanitary safeguard of storage. Dangerous intestinal microorganisms tend to die a natural death during the time that the water is stored in a large impounding reservoir. This time usually varies from a few weeks to several months. Furthermore, there is the primary protection in that such reservoirs are very often located in sparsely settled or uninhabited districts where sanitary conditions on the catchment area are readily subject to control.

The chief disadvantage of impounding reservoirs as storage basins is that they are liable to heavy growths of algæ and other microscopic organisms responsible for objectionable tastes and odors. The stagnation of the lower layers of deep bodies of water also contributes to bad tastes and odors at certain seasons of the year.

Collection and Storage.—The proper development of a catchment area for collecting and storing water calls for a knowledge of many features of engineering science and a special degree of skill in applying these facts to a particular project. The amount of water that can be economically obtained is influenced by many factors such as, the total amount of rainfall, the distribution of rainfall throughout the year, the mean annual temperature and fluctuations from the mean temperature, the amount of water surface on the area, the nature of the cover and of the underlying geological structure, and the suitability of the area for dam sites.

In a region like that of New England it is customary to plan for a storage of two hundred days' supply in order to provide water at all seasons and to allow for fluctuations in run-off from year to year. In some parts of the West, however, several years of storage may be required to meet the deficiency of long periods of drought.

With a rainfall of forty-odd inches per year in New England an average

⁹ International Joint Commission of the United States and Canada.

run-off of about 750,000 gallons per day is expected for each square mile of watershed when there is economical development of storage. To obtain this amount a storage capacity equivalent to approximately 150 million gallons for each square mile of watershed area is required.

The impounding reservoir designed to furnish New York City with a new supply of water to supplement the Croton System is the largest artificial reservoir for water supply in America, if not in the world. It was made by damming Esopus Creek in the Catskill Mountains and holds 120 billion gallons of water. Boston is supplied from impounding reservoirs on small streams: the Cochituate (1848), the Sudbury (1878), and the Nashua (1898). The Wachusett Reservoir stores the water from the latter source of supply and has a capacity of sixty-three billion gallons of water. Numerous other large and small cities are served by impounded supplies.

Stagnation of Water in Impounding Reservoirs; "Overturn."—The stagnation of water in impounding reservoirs and small lakes and ponds deserves special mention. Hazen points out that in our climate, when a reservoir or lake is more than twenty to forty feet deep, the upper part of the water is usually in circulation under the influence of the wind and the lower part remains stagnant. There is little or no mixing between the surface layer and the bottom water, except for two short periods each year, one in the spring and one in the fall. These periods of circulation to the bottom are known to waterworks men as the spring "overturn" and the fall "overturn."

During the summer weather a stratum of warm water remains at the surface. This layer may be twenty feet in small reservoirs, and forty feet in great lakes. The temperature of this surface layer may reach 75° or 80° F. or more in midsummer. The wind stirs it up to a certain depth (about twenty to forty feet), depending upon the depth of the reservoir and the force, direction, etc., of the winds. Daily fluctuations in temperature also cause vertical currents by effecting changes in the density of the water.

The bottom layer is cool and quiet. As the air temperature falls with the approach of winter the surface water cools, becomes denser and mixes with water below. When the difference in temperature between the surface and bottom layers is decreased, the wind action extends deeper and vertical currents arise, and eventually all the water in the reservoir turns over and mixes from top to bottom. The mixing continues for a few weeks, until the temperature of the surface water falls below the point of maximum density, namely, 4° C. Then the colder water remains at the top and winter stagnation results. The top often freezes and entirely shuts out wind action, so that the period of winter stagnation may be even more quiet than the summer period. The spring "overturn" is caused by a reversal of the conditions causing the fall "overturn"; surface water is warmed until it reaches the temperature of the bottom water, when the upward and downward currents take place.

It can readily be seen that this phenomenon of stratification has much to do with the quality of the water. Thus, the organic matter upon the bottom of reservoirs decomposes, and in the absence of oxygen produces the vile odors

and nasty tastes of putrefaction. These odors and tastes accumulate in the bottom water until the fall "overtun"; then they become mixed with all the water in the reservoir. If the water is drawn from the reservoir near the top, as it usually is, there will be a great change in the quality of the water at the time of the fall "overtun." These fall changes are more intense than those which take place in the spring. The surface water is well charged with oxygen, and, as this falls to the bottom, it oxidizes and neutralizes some of these products of decomposition. Tastes and odors due to this cause may be removed by aerating the water by means of fountains, cascades, falling over a dam, or any other similar means. For a further discussion of this interesting subject see Hazen's *Clean Water and How to Get It*.

GROUND WATER

Water which is taken from the ground by means of wells or that which flows naturally from the ground, as in the case of springs, is usually satisfactory, as far as injurious impurities are concerned. The surface water is greatly purified as it percolates through fine, sandy soil. This is nature's process of filtration; the organic matter is oxidized, the bacteria are largely strained out. The soil can take care of a large amount of pollution, and, if not overburdened, or if it has no cracks or crevices, the ground water may be entirely free of objectionable organic substances and bacteria. In passing through the soil the water takes up a rather large amount of carbon dioxide, which is set free by organic decomposition. The water, thus acidulated, has a greater solvent action for lime and other mineral constituents, so that ground water is apt to be harder than surface waters, and to contain a larger amount of dissolved inorganic substances. In deeper waters the solvent action is favored by increased heat and pressure, so that deep wells and artesian waters are frequently unfit for domestic use on account of the large amount of lime, iron, salt, and other inorganic impurities which they contain.

The water that soaks into the soil finally rests upon an impervious stratum. Such water, as a rule, does not exist in the ground as a river¹⁰ or lake, but occupies rather the spaces between the sandy particles, except in limestone formations. Ground water, therefore, in any quantity is found, as a rule, in sandy, gravelly, or sandstone formations.

Ground water finally reaches a certain stratum where it ceases to pass downward, and is then directed in a horizontal plane, forming a more or less continuous bed of water. This is known as the *ground-water table*, which underlies practically all of the earth's surface. It is tapped when wells are sunk, and forms springs, lake and marshes, where it crops out on the surface.

It is only in limestone regions that the ground water exists as flowing rivers or in large bodies. In such instances, as, for example, the Mammoth

¹⁰ Leipzig and Pueblo both take their water supply from underground "rivers" flowing through coarse gravel. In Leipzig the stream is two miles wide, forty feet deep and covered by six feet of soil; it probably represents the bed of an old river.

Cave in Kentucky, the underground river may appear and disappear suddenly. The sanitary hazard of water from limestone crevices is much greater than that obtained from a sandy soil.

The surface of the ground water does not follow the surface of the land, but more approximately the contour of the impervious stratum on which

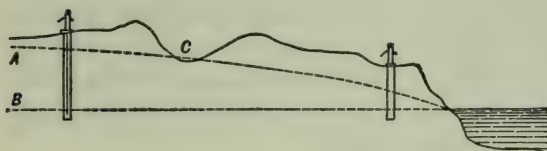


FIG. 83.—GROUND WATER.

A, High level. B, Low level. C, Intermittent spring.

it rests. It crops out at the surface here and there, to form rivers, ponds, lakes, and springs. The irregularity of the surface of the ground-water table is due to a certain extent to the rainfall. During drought the level becomes more and more uniform, until it may

become quite horizontal. It moves slowly towards streams or the sea, and this is why fresh water may usually be obtained by sinking a well at the seacoast.

Amount.—The amount of water that may be obtained from the ground can only be determined by means of actual pumping tests carried on for a sufficient length of time to bring about an approximate state of equilibrium between the supply and the demand, as determined by the level of the ground water. It is rarely practical to continue such tests until perfect equilibrium is reached, for in many cases several years of operation would be required to determine the ultimate capacity of a source. Pumping tests of short duration are apt to be very deceptive, as ground water may exist in the form of a large basin or reservoir with very little movement, corresponding to a surface pond with small watershed, and brief tests would give little more information than similar tests on a pond.

It is easier to get a little ground water than a large amount, and for this reason ground water supplies are more generally available for, and better adapted to, the needs of small places than of large cities. In the United States, many small Middle West communities are supplied with ground water from driven wells.

In Europe, ground water supplies have been secured for many large cities; there has been no corresponding development in America. The reasons for the greater use of this method of supply in Europe, especially in Germany, are: smaller quantity of water required per capita, more favorable geological conditions, more study of the subject and greater efforts to secure ground water. In some cities the amount obtainable has diminished in the course of years or has not kept pace with the needs of growing cities.

Ground water may be obtained from: (1) sand and gravel deposits, (2) sandstone rock, (3) limestone formations.

Ground Water from Sand and Gravel Deposits.—Water flows through sand with some difficulty due to friction on the sand particles. From a given

pumping station it is only possible to draw the water from a limited distance. This distance depends upon the depth and coarseness of the sand. Therefore, the only way to secure a large quantity of water from such formations is by the use of a number of comparatively small pumping stations, spaced so as not to draw from the same territory.

Only a given amount of water can be secured from a square mile of ground. The amount depends upon the rainfall, upon the evaporation from the surface of the ground, from transpiration of vegetation, and upon the amount of storage in the pores of the soil.

Most of the sand deposits of our country are not practically available for water supply purposes, because the grains of sand are too small and the flow of water through them is too slow. It is only the coarse-grained sands that are practically available.

A few large cities in America obtain their drinking water supplies from ground water obtained from sand and gravel deposits. The water supplied to Camden, New Jersey, is obtained from the ground through wells close to the Delaware River. This water filters through the sand slowly and is thus purified. This method of adding to the yield of wells is used in some places in Germany and France. Memphis, Tennessee, is probably the largest city of the United States supplied entirely with water drawn from sand and gravel deposits. In this case the water-bearing area is several hundred feet below the surface, and is below a clay layer. Lowell, Massachusetts, obtains ground water from three stations, draining different areas of glacial drift, while Baton Rouge obtains its water from an artesian well system two thousand feet deep.

Ground water obtained from sand and gravel deposits is usually clean and free from unwholesome impurities. Nevertheless, many towns and cities having such a supply were compelled to seek other sources, because sufficient water was not obtainable to supply rapidly growing population.

Filter galleries are excavations in sandy formations near river banks. Water from such sources corresponds in all practical respects to the ground water obtained from sand and gravel deposits by means of wells. The wells are preferable, as they allow water to be drawn at a lower level, and this tends to a drainage of a greater area, thereby securing a larger quantity of water.

Filter galleries are apt to furnish a diminishing supply, because the pores of the filtering material become filled with the sediment of the river water. When this happens there is no way of renewing the supply. In some torrential streams the filtering surface is renewed from time to time, but this usually does not occur.

Ground Water from Sandstone Rock.—The method of driving wells in sandstone rock differs from that in driving wells in sand or gravel, but the collection, storage, and flow of water are precisely the same.

The cementing material, which binds what otherwise would be loose sand into a solid rock, often seems to offer but little resistance to the flow of water, and the sandstone for water supply purposes acts as so much sand would act.

Water drawn from sandstone is always well filtered. It, however, is usually limited in amount, and, while of the greatest value for small supplies, is not sufficient for large communities. The Marshall and Potsdam sandstones underlying parts of Michigan, Illinois, Wisconsin, and Minnesota are used extensively for supplying towns and small cities. Thus, Jackson, Michigan, with a population of over 48,000, is one of the largest cities so supplied.

Ground Water from Limestone Formations.—In limestone formations the underground flow of the water is not through sandy or porous rock, for limestone is not porous. The water travels through fissures or passages. When these are large they are called caverns or caves, which are natural seams or cracks enlarged by the gradual solution and removal of the limestone by the passing water. Limestone is the only common rock that is soluble in this way, and, for water supply purposes, limestone formations must be distinguished from all others.

The crevices may be, and often are, continuous for many miles. They are remarkably tortuous and anastomose freely, and the direction and flow of the water bear no relation whatever to the surface drainage. Pollution at one point may, therefore, endanger those using the water at a far distant place.

Limestone formation has little ability to hold the abundant winter flows to maintain a supply through droughts. The difference between limestone and sand in this respect is striking, and, from a sanitary standpoint, the fact is significant that water flowing through sand is filtered and purified, whereas no such action takes place through limestone fissures. While much water is frequently available at one point in limestone formations, the amount is subject to great fluctuations, and the supply may fall short when most needed.

That contamination at one point may soon reappear at a far distant point may be demonstrated by the use of fluorescent dyes, or by the use of massive cultures of some harmless microorganism, such as yeast or *Bacillus prodigiosus*.

In our country San Antonio, Texas, is supplied with water from limestone springs. Indianapolis was at one time and Winnipeg in Canada was also supplied largely from this source. Paris in France is partially supplied with limestone water. Vienna obtains its supply from the wonderful Kaiserbrunnen and other limestone sources, which are all in the high mountains, where there is scarcely any population or pollution. This supply is mainly from the melting ice and snow of the high mountains which replenishes the springs, so that the amount of water obtainable is greater in summer than winter.

Typhoid fever has been caused rather frequently by the use of ground water from limestone formations. This has been demonstrated in Paris, Switzerland, France, England and elsewhere. A typical example was the Lausen epidemic (see page 1060). Water supplies from limestone formations must, therefore, be regarded with suspicion.

Wells.—A well is nothing more or less than a hole sunk into the earth to reach a supply of water and fitted with some mechanical arrangement for lifting the water to the surface. Wells may be either shallow or deep, dug,

drilled or driven. The type depends upon the nature of the material through which the well is sunk. By a shallow well is usually understood one which is dug and lined with stone or brickwork. The cylinder is usually five or six feet in diameter and rarely over thirty feet deep. Driven wells are made by driving an iron pipe into a sandy or gravelly soil. The iron pipe is perforated near its pointed end, for the entrance of the water. By deep wells are meant drilled or the so-called artesian wells. They consist of an iron pipe or tube six to eight inches in diameter, and may extend many hundred feet into the earth. If the water is drawn from a depth of one hundred feet or more without passing an impervious stratum, the well is usually spoken of as a deep well. If the well passes through an impervious stratum into a pervious one beneath, in which the water rests upon another impervious stratum, it is spoken of as an artesian well.¹¹ Water is usually pumped from the well either by means of the ordinary suction pump or by means of compressed air.

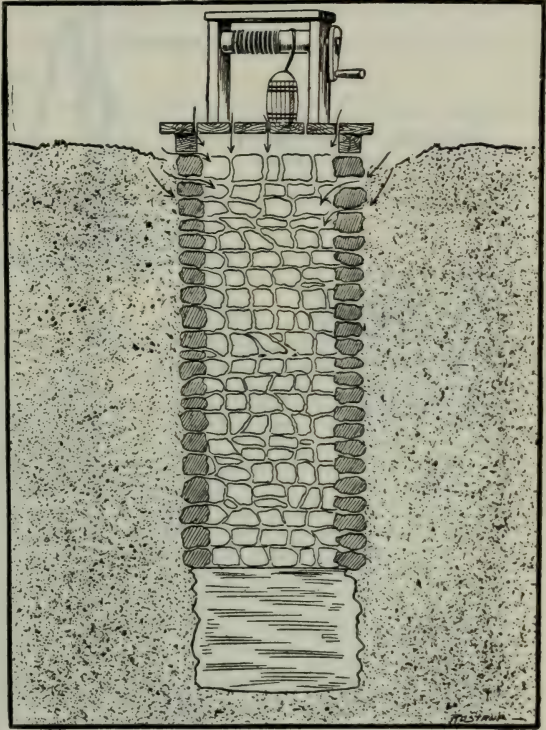


FIG. 84.—USUAL METHOD OF POLLUTION AND EVEN INFECTION OF WELLS.

Wells may be polluted

from the surface, and also from the subsoil drainage under certain conditions.

The filtering power of the soil is usually sufficient to protect the water drawn from a well, unless (1) the soil is overburdened with organic matter, or (2) a cesspool, broken sewer, or other gross source of pollution is close by, or (3) channels, fissures, or crevices exist in the soil and subsoil so that impurities reach the well without undergoing the process of biologic filtration.

In locating a well, therefore, much depends upon the surface configuration of the ground, the character of the soil, and the proximity of possible sources of pollution. The casing of a dug well should be sound and tight, preferably of brick laid in cement mortar, pointed on the inside. This impervious casing should extend as deeply into the well as practicable, and after it is laid the

¹¹ The word "artesian" is derived from Artois, an ancient province in France which was supplied with flowing wells. Artesian water may or may not flow spontaneously.

outer space between the casing and the earth should be filled in with well-tamped clay soil or concrete. One of the most important points in the construction of a shallow well is to extend the casing at least eighteen inches above the surface of the ground and to build around it a shield of concrete

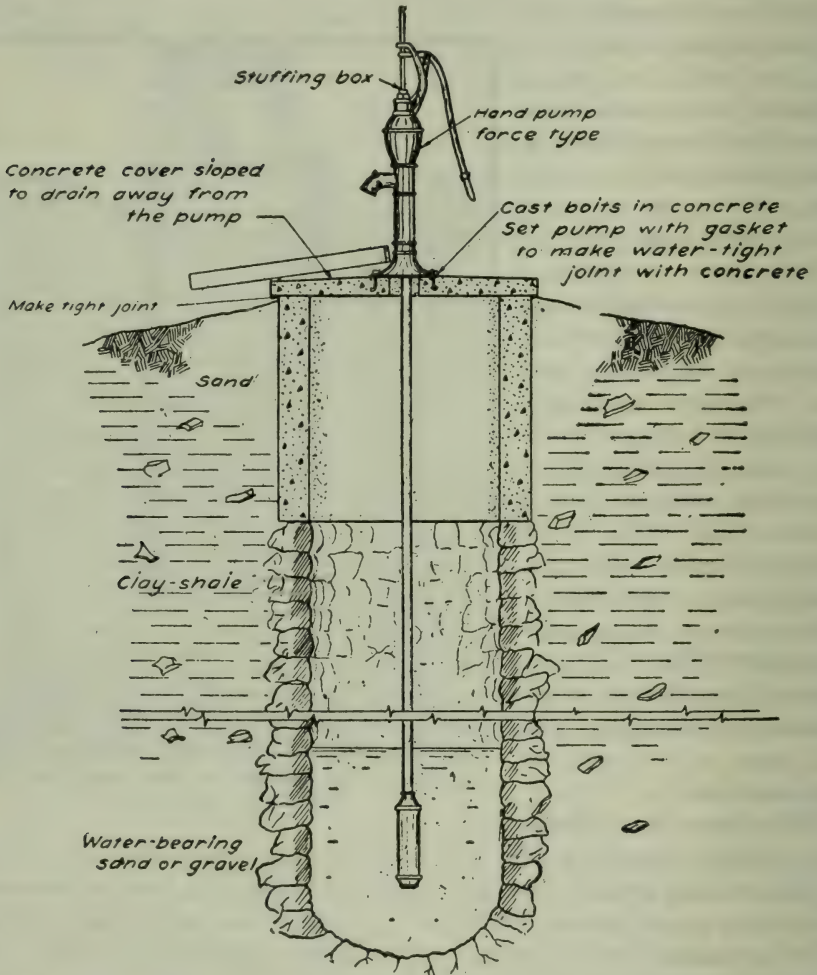


FIG. 85.—DIAGRAM SHOWING HOW A DUG WELL CAN BE PROTECTED AGAINST POLLUTION.
(From *Water Supplies and Sewerage Systems for Farm Residences*, Bd. Health Minn., August, 1921.)

or brick laid in cement extending in a circle from the top of the well three or four feet wide. This shield should join the well casing so as to make a tight joint with the casing. The floor of the well should rest upon the top of the casing, so that no space is left for frogs, mice, or bugs to crawl in. The floor should likewise be water-tight, and is best made of reinforced concrete

with a cement surface. If this is not practicable, it should be made of sound, hard, tongue-and-grooved boards well driven up, and the edges painted with white lead. Upon this should be laid another floor of similar material at right angles to the first. The pump should be let into the floor and firmly fastened to it, and protected with a flashing of tin to prevent water washing back into the well.

The widely prevalent idea that some form of ventilation must be provided for a well is entirely unnecessary. Well water keeps better in the dark and protected from the outer air and dust.

The top of driven wells should be as carefully protected as just described for a dug well, as otherwise polluted surface water may work down the sides

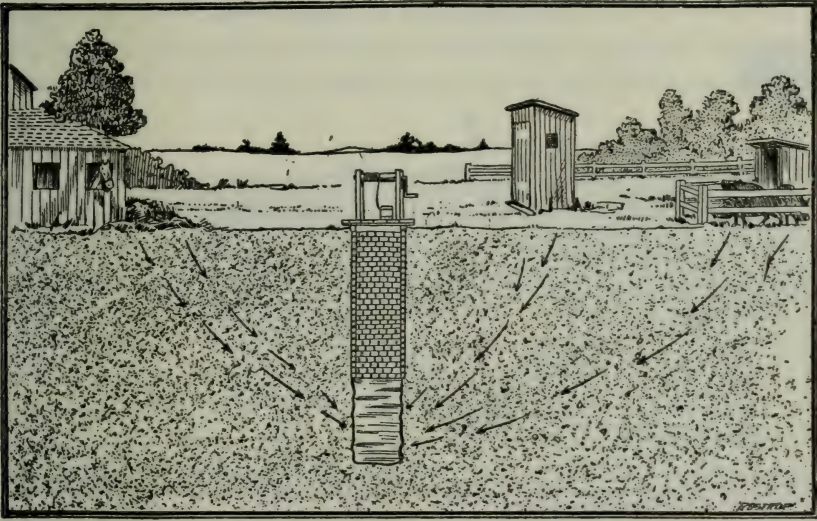


FIG. 86.—POPULAR IDEA OF HOW WELLS BECOME INFECTED FROM SURFACE POLLUTION.

This probably rarely takes place in rural districts, as the soil can usually hold back most of the impurities. The danger is great, however, where fissures, cracks, or crevices exist, or where sewage enters beneath the surface of the soil from broken drains or leaky privies, especially in limestone formations.

of the pipe. Care should be taken that the pipes of a driven well near the surface of the ground do not rust and become leaky. Such wells should be provided with a heavy top, to which the pump frame should be tightly bolted, in order to prevent the loosening of the joints in the pipe by the vibration of pumping. The space between the base and the well casing of driven wells should be filled with grouting and overlaid with cement near the top. The ground about all wells should slope away and be kept clean, and, where possible, should be turfed. The waste water should be carried by pipes to a considerable distance from the well.¹²

¹² On this subject see the admirable reports of the Minnesota State Board of Health: *Water Supplies and Sewage Systems for Municipalities*, March, 1923; *Water Supplies and Sewerage Systems for Rural Residences*, August, 1921.

Artesian water and water from deep wells furnish the safest and most satisfactory sources of supply we have. Such waters are usually clear and of high sanitary quality but sometimes contain a large amount of inorganic impurities, which render them unfit for domestic purposes. Frequently they contain iron or manganese, which soon oxidizes upon contact with the air and is thrown out as an insoluble salt, rendering the water yellowish or brownish. Deep well waters may also contain an excess of lime and magnesium salts, common salt, hydrogen sulphid, carbon dioxid, etc.

Location.—It is evident that in densely inhabited cities, with miles of sewers, some of them doubtless broken or leaky, and with the thousands of privy vaults which still survive in our American cities, we have a more or less sewage-polluted condition of the soil favorable for the contamination of shallow

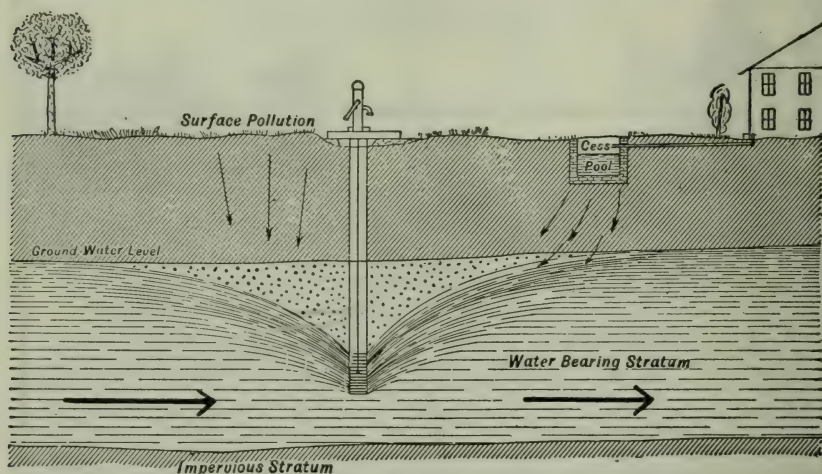


FIG. 87.—DEPRESSION OF THE GROUND WATER LEVEL BY PUMPING AND TENDENCY TO DRAW NEARBY POLLUTION FROM THE SOIL OR CESSPOOL, AGAINST THE NORMAL DIRECTION OF FLOW.

wells. Shallow wells, on general principles, have been gradually eliminated from all large cities having a good water supply. This danger was well shown in the studies upon typhoid fever in the District of Columbia, in which many of the shallow wells situated within the city limits were shown, by chemical and bacteriological analyses, to be polluted.

Water from shallow wells located in sandy or gravelly formations is entirely satisfactory, provided there are no nearby sources of pollution. The proximity of a well and privy may be especially hazardous. Shallow wells in limestone regions must be carefully guarded and always looked upon with suspicion.

Disinfection.—Wells may be disinfected with ordinary lime, which has been found to be fairly effective. The method of injecting steam under a pressure of two atmospheres has been used. The steam is forced into the water until the temperature is brought to near the boiling point. Bleaching

powder, chlorinated lime, however, is the cheapest and most practical substance for disinfecting wells that need such purification.

Springs.—A spring is a stream of water emerging from the ground, its flow being due to natural causes. Spring water does not differ in any essential particular from the ground water obtained from shallow wells. Springs may be regarded as natural wells, outcropping where the geological formation is favorable. Spring water, as a rule, is of a high degree of purity, and as the water flows spontaneously it can easily be utilized. It is less subject to contamination from lowering of the water table than is well water, if pumping is not resorted to and only the natural flow of the spring utilized. Spring waters differ greatly in character, depending upon the temperature of the water and the inorganic constituents which they contain. Springs may be perennial, the flow being constant, or intermittent.

Fuller classifies springs, according to their origin, as gravity and artesian; and according to the kind of passages traversed by the water, as tubular and fissure springs.

Some of the largest flowing springs are found in Florida, notable among these being the Silver Spring with an estimated flow of 368,913 gallons per minute, and Blue Springs with a flow of 349,166 gallons per minute.

Springs may be contaminated from various sources, and in much the same way that wells are contaminated. The overlying porous layer of soil may be too thin to remove the contamination of surface washings from privies, stables, hog pens, and other places. This is probably not a frequent source of danger in such waters. Springs may be directly contaminated from surface washings; that is, the material may be washed down and into the spring by heavy rains, and, unless the spring has a bold flow, the material may remain in it for some time. Leaky cesspools above a spring may carry dangerous material almost directly into the water, just as they endanger wells in precisely the same way. These are sources of danger, but the greatest hazard is found in spring water from limestone formations.

In limestone regions springs are subject to the danger already spoken of in the case of wells. A spring in such a region may be the same stream that runs through the neighbor's back yard and disappears in his meadow. A limestone spring that becomes muddy soon after a rain should be regarded as particularly suspicious.

The protection of a spring against contamination requires a careful study of each location. Stables, hog pens, and privies should be distant, and, if possible, on another slope. Soil pollution must be prevented in the neighborhood of the spring, and animals kept away, and special regard must be had for the location and character of the privy. The spring should be protected above with a masonry or concrete wall. This should extend well into the ground, so as to guard against surface washings. A ditch should be dug around both sides of the spring, to carry off the surface drainage. It is well to plant grass about the location so as to keep out dust and prevent erosion of the soil.

POLLUTION AND CONTAMINATION

Sources and Nature.—Pollution refers to the entrance into water of substances, of any nature whatsoever, which impair its physical qualities. Vegetable matter from swamps and certain manufacturing wastes come in this category and may have no influence upon health. Sewage may not only cause pollution but also contamination and has a very vital relation to health. Contamination is a specific kind of pollution. These terms as used in connection with water are defined on page 930.

Direct evidence of infection in water has so far baffled bacteriologists, except in the case of cholera. Typhoid bacilli have been isolated from drinking water only in a few rare instances, possibly because we lack methods of enrichment and possibly because of the transitory and fleeting nature of the infection. The evidence of water-borne infection is practically always of an epidemiological character gathered after cases of disease have occurred. Contamination, however, can easily be established on the basis of field or laboratory findings, which show that wastes from man or animals defiled the water. *Contamination is an index of possible infection.*

Practically all surface waters are polluted. Ground waters are usually free from pollution but often give chemical evidence of past pollution which has been oxidized. Many surface waters are contaminated, the degree representing the chance of their being infected. Although ground waters are usually free from contamination, they have on numerous occasions been responsible for outbreaks of typhoid fever, dysentery, and cholera.

Danger of Contamination.—The greatest hazard to man is found in a water contaminated with the discharges from the human body: *feces, urine, and sputum*. There is comparatively little danger from water containing the wastes of other animal life, for the reason that few of the infections of the lower animals are thus transmissible to man. There is still less danger in water polluted with organic matter of plant origin. Water containing small amounts of inorganic substances in solution plays a relatively minor rôle, as far as health is concerned.

Prevention of Contamination.—The prevention of the contamination of our streams, lakes, ponds, and other surface supplies is an important sanitary problem with a large economic side. In streams and large lakes, the most dangerous contamination is that which is nearby, that is, that which is quickly transferred in a fresh and virulent form. Cities taking water from an average stream should prevent the access of raw sewage for at least fifty miles, or better one hundred miles, above the intake. Partial protection may also be accomplished by installing sewage disposal works for all upstream towns and settlements and abolishing all overhanging privies upon the river and its tributaries. This requirement should be supplemented by sanitary inspection. When these measures are not feasible, intercepting sewers may be built, as on the Schuylkill at Philadelphia. Canals that parallel

a river, as the one upon the bank of the Potomac, may receive the sewage and surface drainage and thus to some extent protect the stream. It is comparatively easier to guard small lakes, ponds, and impounding reservoirs than long or large streams.

Simple Tests to Determine Sources of Pollution.—Sources of pollution and possibly of infection may often be determined by simple tests which may be carried out by a layman. These tests afford valuable information and consist in the addition of some chemical substance to the source from which pollution is possible and then in determining whether the same reappears in the water supply. For this purpose a large number of substances that may be readily recognized by their taste, odor, or appearance may be used, such as coal oil, carbolic acid, fluorescein, and common salt. Coal oil poured on the ground near an artesian well is an easy and convincing method of establishing the presence of defective piping and surface or subsoil pollution. Nördlinger recommends for this purpose saprol, which tastes like naphtha and is so penetrating that its odor may be readily recognized in proportions of 1:1,000,000 or by taste in solutions of 1:2,000,000. Trillat experimented with a large number of dyes and finds that fluorescein dissolved in alcohol and diluted with 5 per cent ammonia solution can be detected by a fluoroscope in proportions of 1:2,000,000,000. The fluoroscope is a tube of clear glass three or four feet long and one-half inch in diameter, closed at one end with a stopper. In such a tube natural waters have a somber blue color which changes to a clear green if fluorescein is present. Fluorescein can be detected by the unaided eye in dilutions of 0.625 part per million. This dye possesses the evident advantage of not being precipitated by the soil, a reaction that readily occurs with most anilin dyes brought in contact with calcareous solutions.

The conclusion must not be drawn that because these soluble salts reappear in the water microorganisms and dangerous pollution would likewise find its way through the soil for an equal distance, for the soil has well-known filtering power when free from fissures or open channels and is capable of removing bacteria and oxidizing large quantities of organic matter. However, these methods are of service in indicating the possibility of danger under certain circumstances and are particularly useful in discovering sources of pollution near wells or in limestone formations.

Massive cultures of prodigiosus, pyocyaneus, fluorescens, yeasts, and other microorganisms, if not normally present in the water under examination, may be used to detect pollution. The cultures are poured upon the ground or into suspicious places and the water tested at varying intervals to determine whether they reach the supply. Careful controls must be made beforehand to assure the absence of the particular organism used.

The Interstate Pollution of Streams.—Sanitarians have maintained for years that no community or individual has a right to dangerously pollute streams used for public water supplies, any more than a man has a right to poison his neighbor's well. England enjoyed the benefit of a Rivers Pollution Commission as early as 1855, in order to prevent, remedy, and remove the

danger of polluted water supplies. This commission adopted a comprehensive system for the disposal of sewage and for water purification, the fruits of which England is enjoying to-day. The United States has no law regarding the interstate pollution of streams, and with our growing population and increasing amount of pollution this is becoming a live and pressing sanitary question. In some of our more progressive states, as, for example, Massachusetts, Connecticut, Minnesota, New Hampshire, New Jersey, New York, Vermont, and others, the State Board of Health is given control over the pollution of streams within the borders of the state. In Pennsylvania the State Sanitary Water Board has jurisdiction and is coöperating with neighboring states to safeguard interstate streams. After the Chicago drainage canal was opened the city of St. Louis (state of Missouri) sued the city of Chicago (state of Illinois) through the federal courts, asking an injunction against the pollution of the Mississippi River, from which St. Louis draws its drinking supply. The testimony occupied many weeks, and in published form takes up many volumes. The verdict was "no cause for action," or "not guilty"; that is, it was not proved that typhoid bacilli or other organisms dangerous to health reached St. Louis from Chicago.

Speaking generally, jurisdiction over the pollution of waters in the United States is confined to the several states. There is no provision in the Constitution which gives to Congress authority in the premises. Hence, by the familiar principle in our Constitution that the several states retain full sovereign power, except so far as such powers are restricted by the national Constitution or expressly delegated thereby to the national government, the individual states have full control of this subject—a subject with which they are individually impotent to deal, but one with which much can be done through coöperation.

Pollution of International Boundary Waters.—Under terms of Article IX of a treaty of January 11, 1909, between the United States and Great Britain, the questions of extension of pollution of boundary waters and remedies were referred to the International Joint Commission under date of August 1, 1912. A sanitary survey was made of the Great Lakes District, particular emphasis being laid on sewage pollution from cities and towns, sewage from vessels, saw mill and other industrial and household wastes, and present methods for their control. In general, the conclusions embodied in their final report, 1919, are that it is "feasible and practicable without imposing an unreasonable burden on the offending communities to prevent or remedy pollution, both in the case of boundary waters and waters crossing the boundary. In case of city sewage, this can best be accomplished by installing collection and treatment works having special reference to the removal of bacteria and matters in suspension." The conclusion is made that vessels should treat their sewage before discharge, as for instance with live steam, also that water ballast discharge be regulated with due regard for drinking water intakes for any neighboring community; further, that restriction should be placed on disposal of garbage and carcasses in boundary waters.

The Care of Catchment Areas.—"Catchment area," "watershed," "drainage area," and "catchment basin" are terms used to include the area immediately surrounding a water supply so situated that water falling upon it will drain toward this supply. The ideal catchment area is free from human habitation and is covered with forests. The catchment areas supplying impounding reservoirs and the natural ponds and lakes used as reservoirs are limited in area when compared, for example, with the catchment areas of the great rivers, from which many public water supplies are drawn. It is, therefore, possible to inspect and control the former more readily than the latter.

It is often impossible to remove population from a catchment area, and, in fact, it is usually unnecessary to do so. Very good water may be drawn from areas upon which there is a large population, when proper and well-known precautions are taken. Thus, there are 282 people per square mile upon the Sudbury catchment area, and 49 upon the Wachusett, furnishing Boston's water supply, and 59 upon the Croton, furnishing part of New York's water supply. The use of the automobile has greatly increased the hazard upon all such watersheds, for it is not possible to effectively control the habits of an itinerant population.

The prolonged storage of the water in large protected reservoirs is a sanitary safeguard, and makes the Boston water and the New York water safer than it otherwise would be. The greatest danger lies in the fact that contaminated water may sometimes pass through a reservoir by short circuit, due to surface currents or unusual draft, and so reach the consumer before it has enjoyed the full benefits of purification by storage.

The proper sanitary care of a catchment area requires, first of all, sufficient laws granting suitable authority, especially concerning the disposal of human wastes.

Care must also be exercised to keep out manufacturing wastes and the surface washings that may carry contamination from human sources or undesirable pollution from other sources. This object may be accomplished in various ways. The city should own the shores of the reservoir and also as much of the land along the important streams as is necessary to carry out these objects. Old sources of pollution must be removed, and new sources prevented. Where the danger from human contamination is especially great, as around the impounding reservoir itself or at nearby suburban settlements, engineering projects, sometimes of considerable magnitude, are necessary to carry away the sewage and the surface drainage. A strict patrol of the catchment area, in order to supervise picnic and camping parties, the camps of construction gangs, and other sources of danger, must be exercised. A good man on the alert can patrol a large district, getting his information in various ways, and personally inspecting all suspicious localities frequently.

In the investigation of a stream and its watershed the chief points requiring attention are the relative proportions of the polluting matter and the flow of the stream when at its minimum; the general character of the stream, the

rate of flow, and the distance between the source of pollution and the intake of the water.

Water boards should plant their catchment areas with trees. Trees add to the retention of water falling as rain, lessen evaporation, and cool the adjacent atmosphere, perhaps aiding condensation. Trees prevent floods and regulate and help to purify the supply, for water draining through the soil of wooded areas is naturally cleaner than that scouring the surface of barren land.

CHAPTER II

SANITARY ANALYSIS OF WATER

A complete estimate of the sanitary quality of drinking water can be given only after: (1) examination in the laboratory; (2) inspection in the field; and (3) experience acquired from actual use of the water. Information obtained from any single one of these three sources does not furnish complete evidence upon which final judgment can be founded, or future predictions safely made.

Clinical Aspects.—The clinical result from protracted use is in a sense the final test of fitness. It furnishes past proof of the effect upon health in so far as clinical symptoms are recognized, but medical science is constantly discovering new causative agents of disease, living and inert, and placing new emphasis upon previously neglected conditions of health. As a consequence, standards of fitness for drinking water tend constantly to become more rigorous. Therefore, it is necessary to have the fullest possible information from laboratory and field upon the constituents of the drinking water used in any community in order to determine if this important article of diet is concerned with the new knowledge. Also, what is more important, past fitness, as judged from clinical results, gives no indication of deteriorating quality and threatening danger. Inspection and analysis furnish prior warning of this.

A change from a contaminated water to a pure supply may bring unlooked-for consequences in its wake. This was the case in Vienna where the new supply was followed by a great increase in the amount of goiter owing to the absence of iodine in the water.

The Field Survey.—A phase of the examination of water supplies which is of surpassing importance is the survey of sanitary conditions in the field. This calls for a consideration of a great many items and the work should be intrusted only to persons who have by training acquired a broad knowledge of those matters which are pertinent to the sanitation of water sources, to their physical development, and to the purification of water. The survey should deal among others with the following features:¹

I. *Ground Water Supplies*

1. Geology of the strata above and in which the supply is taken.
2. Slope of the ground-water table with reference particularly to known sources of pollution.
3. Extent to which the water table or underground supply is lowered by consumption.

¹ For a complete discussion of this subject see Appendix I, *Report of Advisory Committee on Official Water Standards*, U. S. Public Health Service; *Pub. Health Rep.*, 1925, 40, No. 15.

4. Study of known sources of possible pollution and the extent to which they contribute an element of danger.

5. The chance of contamination reaching the supply adventitiously, as by faulty casings, poor protection at the surface, the proximity of abandoned borings, etc.

6. Methods in use for protection of the supply, such as control of population on the contributing area, disposition of sewage, and limitations placed upon other users of the source.

7. Purification of the water. Nature and adequacy of the processes used.

II. *Surface Water Supplies*

1. Geology of the catchment area.

2. Character of the catchment area, flat or steep; its cover, forest, waste, or farm land.

3. Density of population and number of industries.

4. The disposition of sewage and liquid wastes.

5. Protective measures developed on the watershed, as adequacy of storage, flood control, location of intakes, sanitary inspection, and regulation of fishing, boating, etc.

6. Purification of the water. Nature of processes used, their adequacy, suitability and efficiency under all conditions of raw water quality.

III. *Distribution Systems of All Supplies*

1. Chance of contamination at pumping stations, in distributing reservoirs, or in mains.

2. Material, age, and condition of mains.

3. The use of lead pipe for service connections or house plumbing.

4. Connections with auxiliary supplies of inferior quality. The authority to which is delegated the power to draw upon such supplies.

5. Protective measures against leakage from auxiliary supplies; the pressure differences, use of check valves and gate valves.

The field examination, when properly made and repeated at intervals, furnishes information of the sanitary and hygienic aspects of a water supply of greater significance than can be obtained in any other way. It secures not only collateral evidence to be used in the interpretation of laboratory findings and morbidity statistics, but also brings together facts which indicate the hazards under which the supply is operating. Further, these facts form a basis for prediction of changes to come. They show the trend of conditions from time to time and so are a guide in shaping plans to meet future contingencies.

Laboratory Examination.—A sanitary analysis is intended to furnish evidence of the wholesomeness of water and its general fitness for domestic uses. The analysis of water is unlike that of many other substances in that many of its constituents have no great significance when considered alone and have limited values depending upon the amount of other constituents.

It is also a fact that waters of acceptable quality differ greatly from each other and that the same water may show wide fluctuations in quality from season to season. This is particularly true of surface supplies. For these reasons it has been difficult to establish fixed standards of quality. The interpretation of laboratory findings needs to be made in the light of all the analytical data obtainable and with the guidance of supplementary information from every possible source. When this is done the laboratory becomes an invaluable adjunct for estimation of sanitary quality.

Great stress should be laid upon the frequency of analysis. A single examination shows existing quality, but usually makes judgment difficult, and provides no evidence of the degree of uniformity in composition. The number of examinations to be made in a month, or a year, will be governed by the magnitude of fluctuations found and by the health hazards which beset the use of the supply.

Laboratory examination is one of the very important phases of water purification. The operation of no process can be made certain and effective without a knowledge of the improvement which is brought about. Analysis is the most delicate means of measuring this. The need for purification usually, though not always, implies the existence of contamination, which is potential infection, and, therefore, a menace to the well-being of consumers. The degree of contamination in the raw water and the extent to which it is removed by purification processes can best be judged by tests in the laboratory. Analysis also records changes, not of sanitary significance, which are important in the control of the processes.

Thus it is that the rôle of the laboratory functions midway between the field survey and the test of actual use. Sanitary analysis corroborates or modifies estimates of quality made in the field and indicates, within the bounds of present knowledge, the probable results of use of the water.

STANDARD METHODS

Up to the early years of the present century the methods employed for the examination of water were those dictated by personal preference on the part of the analyst. Much confusion, not to say inaccuracy, resulted and it was often impossible to check the work of one laboratory against another. The first attempt at standardization of procedures in water analysis came with the report of a committee of the American Public Health Association, published in the *Journal of Infectious Diseases*, May, 1905, Supplement No. 1. The work of this committee constitutes one of the most important contributions ever made to the subject. It was later printed in book form under the title of *Standard Methods of Water and Sewage Analysis* and distributed by the American Public Health Association. Additions and revisions have been made and published every few years, the last to appear being in 1925.

This book is the standard manual of procedure and the methods there laid down are accepted as official and are generally adhered to by water analysts.

Any deviation from them should always be noted in published reports. Standard methods represent group judgment and are adopted by common consent as the rule to be followed under ordinary circumstances, especially in routine work and by those who are not skilled in laboratory technic. For reasons that seem self-evident, it is of special importance to follow standard methods for bacterial examination. It should be pointed out, however, that a "standard" method is not always the best method to follow in some circumstances, and its adoption should not stifle initiative in developing improvements or a new method.

The Parts of a Sanitary Analysis.—A sanitary analysis of water resolves itself into four component parts: (1) the physical, (2) chemical, (3) microscopical, and (4) bacteriological examinations.

Physical Tests.—Physical tests have to do with the determination of substances which influence physical or æsthetic quality, as taste, odor, color, and turbidity. Such tests are empirical and arbitrary in method and form of expression. They do not record amounts of specific compounds which may be present, for any of the above-mentioned qualities may be due to a variety of compounds, many of which are of complex, organic structure and some incapable of detection in the light of present laboratory knowledge. The methods employed for the physical examination determine the total effect of all compounds present which can be measured by the sense of taste, smell, or vision. Arbitrary standards are used for comparison in the determination of color and turbidity.

Chemical Examination.—The chemical examination makes use of quantitative methods which measure the amounts of different chemical ingredients. Some of the methods determine directly specific compounds which are present. Others are in the nature of indirect or inferential tests and record substances derived by chemical reaction which indicate the presence and amounts of ingredients too complex for direct determination. The chemical examination is useful in detecting pollution and in estimating normal chemical constituents such as iron, salt content, and compounds which cause hardness.

Microscopical Examination.—The microscopical examination is designed to detect and measure the amount of living and of inert particles which are suspended in the water. It does not include the bacteria. Microscopical analysis is most useful in studying the numbers and kinds of plant and animal organisms, like the algæ and protozoa. It also detects and identifies fibers, cells, and solid particles of all kinds.

Bacteriological Examination.—The bacteriological examination deals with the numbers and kinds of bacteria present. From its findings a better appraisal of hygienic quality can be made than from any other part of a sanitary analysis. It has been possible from experience to define more closely limiting values for bacteria than for other constituents of water.

From the foregoing it will be noted that each of the four parts of a complete sanitary analysis deals with different qualities of the sample. It is, therefore, of prime importance that any estimate of character and quality

which is based upon laboratory findings should be made in the light of all the tests assembled and correlated.

Our standards by which the purity of water is judged are constantly rising. There is no doubt that many waters now considered satisfactory will not be acceptable in the future. A water must have more than the negative property of being safe; it should have positive qualities: it should be crystal clear, sparkling and inviting, cool and refreshing.

Collection of the Sample.—For a complete physical, chemical, and microscopical analysis of water one gallon is necessary. If the sample has been collected in a sterile container with precautions to prevent contamination the same sample may serve for the bacteriological examination. Usually the bacteriological samples are collected separately in special bottles holding at least 100 c.c., but not completely filled.

The bottles for bacterial samples should be of hard, clear white glass and have a glass stopper. They should be chemically clean and dry sterilized at 160° C. for one hour, or in the autoclave at 115° C. for fifteen minutes. For transportation they may be wrapped in sterile cloth or paper, but, better, the neck may be covered with tinfoil and the bottle placed in a tin box. Cork stoppers should never be permitted. Earthen jugs and metal containers are entirely unsuited.

Samples for physical, chemical, and microscopical analysis should also preferably be collected in bottles of good glass with glass stoppers. Cheaper bottles may be used for physical and microscopical samples under certain conditions and when only a short time elapses between collection and examination. Water must be analyzed as soon as possible after sampling.

Generally speaking, the shorter the time elapsing between collection and analysis, the more reliable will be the analytical results. If too long a time intervenes, it affects especially the bacterial tests, for bacteria multiply enormously when water is kept in a bottle at ordinary temperature. The dissolved gases, the oxygen consumed, the oxygen demand, and the nitrogen determinations are also materially affected by comparatively short delay. The following are the maximum times which may elapse between sampling and analysis (*Standard Methods* of the American Public Health Association):

PHYSICAL AND CHEMICAL ANALYSIS

Unpolluted waters	72 hours
Fairly pure waters	48 hours
Polluted waters	12 hours
Sewage and sewage effluents.....	4 to 6 hours
(If a longer time must elapse, 6 c.c. of chloroform per liter of sample must be added and the sample put on ice.)	

BACTERIOLOGICAL ANALYSIS

Pure waters	12 hours
Impure waters	6 hours
Samples to be kept at 10° C. or less.	

Samples for microscopical analysis are preferably examined in the field, especially if fragile organisms are present. Under no circumstances should the elapsed time be more than twelve to twenty-four hours, depending upon the purity of the sample.

Technic of Sampling.—Care should be taken to secure a sample thoroughly representative of the water to be analyzed. A pump should be operated five minutes, or water faucet allowed to run several minutes, before the bottle is filled. In collecting samples of surface waters the specimen should not be obtained too near the bank of the stream or pond and surface pollution should be avoided. A note should be made as to whether the specimen is collected from the surface or at what depth under the surface it is taken. It is always advisable to take the temperature of the water at the time of collection.

Methods of Analysis.—The official procedures in water analysis given in *Standard Methods of Water and Sewage Analysis* of the American Public Health Association should be closely adhered to unless there is special reason for using others or modifications.

In the following pages the constituents of water which have sanitary significance are discussed and their meaning interpreted; for the technical details of the methods the analyst will follow the standard procedures.

EXPRESSION OF CHEMICAL RESULTS

Formerly results were expressed in grains per gallon. After the introduction of the metric system results were expressed in parts per hundred thousand, but now results are commonly expressed in parts per million. The latter method has the advantage that 1 mg. is 0.000,001 liter, and, therefore, 1 mg. in 1,000 c.c. = 1 part per million. A liter or a fraction thereof of the water to be analyzed is used, which greatly simplifies the calculations.

Of course, the assumption is made that a liter of water weighs a kilogram. This is sufficiently accurate for potable waters, but introduces an error where mineral waters are dealt with, the specific gravities of which are appreciably higher than unity. In such cases the water may be actually weighed, or the weight may be estimated from the known specific gravity and volume.

The results expressed in parts per hundred thousand or in grains per gallon may be transformed to parts per million, or, conversely, by the use of the following table:

	Grains per U. S. Gal- lon	Grains per Imperial Gallon	Parts per 100,000	Parts per 1,000,000
1 grain per U. S. gallon	1.000	1.20	1.71	17.1
1 grain per imperial gallon	0.835	1.00	1.43	14.3
1 part per 100,000	0.585	0.70	1.00	10.0
1 part per 1,000,000	0.058	0.07	0.10	1.0

ODORS AND TASTE

The purest water is absolutely devoid of taste and odor, but it is also insipid. If such water is aerated by agitation or by filtration through a porous air-containing substance, it becomes sparkling and agreeable. The taste is imparted to most waters by the mineral matter as well as the gases held in solution; hence the flat, insipid taste of distilled water. After a person becomes accustomed to the taste of a particular water another does not appeal to him and does not satisfy his thirst to so great an extent. Once having been accustomed to a moderately hard water, a soft water seems very flat and tastes much like distilled or rain water.

Odors in waters are objectionable, rather than detrimental to health. As a rule, the most objectionable odors develop in surface waters and are caused by the growth of algæ, diatoms, protozoa, and other microscopic beings. Tastes always accompany these odors and are usually, though not necessarily, similar in character. Industrial wastes are sometimes responsible for odors and tastes. Chemical treatment of water often produces tastes, and sometimes odors. Chlorin, used for disinfection, is a conspicuous example, an excess causing a chlorinous taste and odor, while smaller amounts often unite with organic constituents to produce an "iodoform" or medicated taste. The earthy odor of some ground waters is due to substances taken up during the passage of the water through the soil. When a well water becomes offensive it is evidence of stagnation at the bottom of the well or the presence of dead animal or plant life. In the case of deep wells, hydrogen sulphid and other inorganic compounds may impart odors to the water. The odors and tastes which develop in impounded reservoirs from stagnation and putrefaction of the organic matter in the depths have been discussed on page 944.

Determination of Odor.—The odor of the water should be observed both at room temperature and just below the boiling point. Odors should be determined at room temperature (20° C.) by shaking a sample violently in a gallon collecting bottle when it is half or two-thirds full; and by heating to a point just short of boiling about 150 c.c. in a 500 c.c. Erlenmeyer flask covered with a well-fitting watch glass. In either case care should be taken to observe the character of the odor the instant the receptacle is uncovered. The kind of odor observed may be described as vegetable, aromatic, grassy, fishy, earthy, mouldy, musty, disagreeable, peaty, sweetish, etc., and the intensity by such terms as very faint, distinct, decided, or very strong.

Odors Due to Microscopic Organisms.—The odors and tastes in water caused by microscopic organisms deserve special consideration, because they are common faults in water stored in open reservoirs of all kinds. Certain organisms can be distinguished by their odor, as, for example, the "fishy" odor of *Uroglena*, which is a protozoön and classed with the *Infusoria*; the "aromatic" or "rose-geranium" odor of *Asterionella*, which belongs to the *Diatomaceæ*; and the "pig-pen" odor of *Anabena*, which is one of the blue-

green algæ.² These microscopic organisms mostly grow near the surface and require sunlight for their development; hence, they never grow in covered reservoirs or in waters kept in the dark.

Calkins has shown that the odors caused by the undecomposed microscopic organisms are due to compounds of the nature of essential oils, and Whipple points out that the amount of such oil produced by an abundant growth of the organisms is quite sufficient to account for the effect observed. He notes for comparison that oil of peppermint can be recognized when diluted with water in the proportion of one part of oil to fifty million parts of water, and that when *Asterionella* is present to the extent of 50,000 organisms per c.c. the dilution of its oil is in the proportion of about one part to two million parts of water. Whipple further suggests that the flow of water through pipes may cause disintegration of organisms with liberation of the odor-producing oil, hence the odor at the tap may be greater than at the intake.

The microorganisms causing bad tastes and odors in water are mostly plant forms, known as *Algæ*, and do not depend upon organic matter or the bodies of other organisms for their food supply. They require for their growth only sunshine, carbonic acid and the nitrogen and mineral matters always present in the water and in the air. In other words, they have properties comparable to the

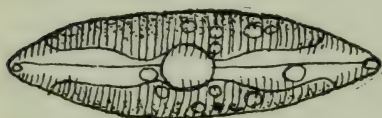


FIG. 88.—THE OIL DROPLETS IN ONE DIATOM.

higher orders of chlorophyll-containing vegetation.

There are very many kinds of algæ, and they differ greatly in their odor-producing powers. Most American impounded reservoir waters suffer from them, but some far more than others.

A certain degree of quiet and repose is necessary for the development of a large growth of algæ; that is why they never develop to great extent in rivers and flowing water. Wave action from wind also prevents growth, and this seems to be the only reason why large lakes and reservoirs are less troubled by them than smaller ones.

In most American impounded reservoirs the water is drawn from near the surface layer, so as to avoid the odors and tastes of putrefaction in the bottom water, but it sometimes happens that the surface water is the more objectionable because algæ grow there.

Prevention and Removal of Tastes and Odors.—The natural flow of water in the bed of a mountain stream over stones and ledges aerates it very well. This is nature's method of removing undesirable tastes and odors. Aëration may also be accomplished by bringing the water in contact with the air by devices such as fountains, waterfalls, etc. Such aëration always reduces, and sometimes removes, tastes and odors from the waters of reser-

² See also Whipple, *Microscopy of Drinking Water*, John Wiley & Sons, 1914.

voirs and small lakes, whether resulting from putrefaction in the stagnant bottom water, from growths of organisms in the surface water, or from gases held in solution.

In general it may be stated that filtration alone is not efficient in removing tastes and odors; however, slow sand filtration has considerable power of reducing, and in some cases of removing, tastes and odors, but it cannot be depended upon when the raw water is very bad.

It is practically impossible to prevent the seeding of reservoirs and ponds with algæ and other organisms responsible for the objectionable odors. The growth may be checked and the odors temporarily controlled by the use of copper sulphate. See page 1036.

If a well becomes stagnant at the bottom, and thus develops vile odors from putrefying organic matter, the trouble may be corrected by lowering the pump so as to prevent stagnation, or by filling up all unnecessary space with clean gravel and sand.

COLOR

Pure water, when viewed in small quantities, appears to be perfectly colorless, but, when viewed in bulk, as in the white-tiled baths at Buxton, and in certain Swiss lakes, it is seen to possess a beautiful greenish-blue tint. A very small amount of suspended or dissolved impurity is sufficient to modify or obscure this color.

Impure waters almost invariably exhibit a color varying from green to yellow and brown, when examined through a depth of several inches in suitable tubes. It does not, however, follow that the pollution which results in color carries with it infectious material.

Color in surface water is usually of vegetable origin; animal matter contributes but little color. The coloring matter is extracted from dead leaves, bark, and roots, from soil, and from peat. It is similar to the coloring matter of tea. It is certainly harmless, but makes water less pleasing in appearance, and great efforts have rightly been made to prevent and remove it. Water from swamps is usually highly colored, the degree of color depending upon the length of exposure and the character of the vegetation.

Ground waters are usually colorless. If the water contains iron it will be perfectly clear on coming from the ground, but will soon turn a rusty yellow color. This is caused by the oxidation of the soluble ferrous salts to insoluble ferric salts.

Color in water should be distinguished from turbidity. True color is due to colloidal and dissolved impurities; turbidity to substances in suspension. The "apparent color" is the color of the original sample, due to both dissolved and suspended matter.

Determination of Color.—The color of water is determined by matching a 50 c.c. sample in a Nessler tube with a standard solution of platinum and cobalt salts. The comparison is made by looking vertically downward through

the tubes upon a white surface placed at such an angle that light is reflected upward through the column of liquid. The amount of color is expressed by an arbitrary numerical standard; it may also be determined by comparison with the standard color disks of the U. S. Geological Survey.

Prevention and Removal of Color.—Excessive color in surface water can be partially controlled by preventive measures on the watershed. These principally consist of the drainage of swamps and the cutting of vegetation near the shore line. Thus, on the catchment areas of the various reservoirs supplying Boston, thousands of acres of swampy land have been ditched, with good results, to reduce the period of contact of water with dead vegetation. In the construction of artificial reservoirs heavy deposits of muck and organic matter are sometimes removed before the reservoir site is flooded. These measures are always very expensive.

Color may also be reduced by long storage in reservoirs. The action is the resultant of several forces. To some extent there is a bleaching action and oxidation due to exposure to sunlight and air, but it is probable that physical forces play the major rôle and operate to bring about a coagulation of colloidal color particles which gradually precipitate.

The best known artificial methods for color removal are: (1) slow sand filtration and (2) coagulation, followed by either slow or rapid sand filtration. Slow filtration will usually remove from 20 to 30 per cent of the color. Treatment with a coagulating chemical such as aluminum sulphate (filter alum) results in enmeshing the color particles in the precipitate. Sedimentation, followed by filtration, generally of the rapid type, will then remove most of the coloring matter and sometimes all of it. Such treatment is successfully carried out at Norfolk, Virginia, Charleston, South Carolina, Watertown, New York, Cambridge, Massachusetts, and dozens of other cities in the United States.

Ozone, peroxids and chlorin will destroy coloring matter in water, but the amounts required entail an excessive cost.

TURBIDITY

Practically turbidity is synonymous with muddiness. It is that property which interferes with the distinctness with which the outline of objects is seen through water. The turbidity of surface waters is usually due to mud, clay or silt, also to finely divided organic matter, microscopic organisms, and a great variety of matter. Turbidity represents the amount of foreign substances in suspension; it is frequently, though incorrectly, spoken of as color. In a general way turbid waters exist in those regions where color is not found; the former represents the washings of a readily eroded drainage basin, the latter is mostly extracted from the decaying vegetation of swamps.

Pure water is clear and sparkling, but brilliancy and clearness do not guarantee purity, nor, on the other hand, does turbidity necessarily indicate danger. A community may for years drink and seem satisfied with a

turbid water that is little less than liquid mud. This was formerly the case with Washington and the Potomac water, St. Louis and the Mississippi, and many other cities. When, however, the people of such a city once appreciate the beautiful appearance of a clean water, they complain if the turbidity reaches the point of a faint opalescence. The turbidity question is practically limited to river waters. Ground waters should seldom be turbid, and, if so, should at once excite suspicion. Some ground waters become more or less turbid through the precipitation of iron or through the failure of supporting strata which allows entrance of clay or soft rock material to the water.

All river waters are more or less turbid, but the differences are very great indeed. The amount of turbidity depends largely upon the character of the catchment areas. In general, rivers draining the large areas of our North and East, covered with glacial drift of a sandy character, are but little subject to turbidity. Thus, on an average, the Merrimac and Connecticut Rivers do not carry more than 10 parts per million of suspended matter. In that part of our country which is not glaciated, and this includes the lower Susquehanna basin, much of the Ohio basin, and the Missouri basin, and all to the south of them, turbidity is often present in large amounts, and consists largely of clay in extremely fine particles. The water often runs very turbid in these streams continuously for weeks and even months at a time. The Missouri River carries the largest amount of sediment of any of our rivers largely used to supply water. The annual average runs as high as 1,200 or 1,500 parts per million of suspended matter. In winter it falls to 200 parts or less, while in midsummer it rises for weeks and even months to 5,000 parts or more.

If the turbidity is sufficiently coarse-grained it may be removed by sand filtration without previous chemical treatment. Very turbid waters can be cleared, in part, in settling basins; this lightens the work of the filters and reduces the cost. Scrubbers, which are preliminary rough filters, may also be used to protect the sand filters. In many instances the individual particles of clay which make up the turbidity are much smaller than the bacteria. They will not settle out, even after prolonged storage, and they cannot always be removed by filtration alone. There is only one known way of removing such turbidity, and that is by coagulation or chemical precipitation followed by filtration as mentioned for removal of color. This is successfully done with very muddy supplies at New Orleans, Nashville, Richmond, St. Louis, and many other river towns. The substances most commonly used for this purpose are: aluminum sulphate (alum) and sulphate of iron. (See page 1033.)

With reference to the influence of suspended matter upon health we find some conflict of opinion. Kober states that water containing 50 parts per hundred thousand or 30 grains of solid matter per gallon is unfit for drinking purposes. The same effect has been ascribed to large numbers of *algæ*, especially the diatomaceæ, but little is known about these matters.

Ordinary turbidity appears to have no special sanitary significance, but is very undesirable.

The standard of turbidity adopted by the United States Geological Survey³ consists of a water which contains 100 parts of silica per million, in such a state of fineness that a bright platinum wire 1 millimeter in diameter can just be seen when the center of the wire is 100 millimeters below the surface of the water and the eye of the observer is 1.2 meters above the wire, the observations being made in the middle of the day in the open air, but not in sunlight, and in a vessel so large that the sides do not shut out the light so as to influence the results. The turbidity of such water is taken as 100, and all turbidity readings, no matter what method is used, should be based upon this method.

There are three methods by which the degree of turbidity may be determined: (1) the platinum-wire method as described above, (2) the comparison of a sample with standards in clear glass bottles, prepared by dilution of a standardized suspension of Pear's precipitated fuller's earth with distilled water, (3) the use of a turbidimeter, such as Jackson's, which employs a calibrated glass tube, illuminated from below by a standard candle and into which the sample is poured until the image of the candle flame is obscured.

The amount of suspended solids causing turbidity may be determined by passing a measured volume of sample through a previously weighed filter paper or Gooch filter. The increase in weight is due to particles causing turbidity. Where only an occasional analysis is made for general purposes it is sufficient to record the sediment and turbidity as very slight, distinct, or decided.

TOTAL SOLIDS

The total solids or residue on evaporation is obtained by evaporating a given quantity of water to dryness, when a grayish-white residue, composed of the mineral and organic matter which has been held by the water in suspension and in solution, will be obtained. The amount of this residue varies with the character of the water. If the total residue is ignited the "loss on ignition" gives a rough index of the total amount of organic substances present. Much can be learned upon ignition from the nature of the odors of the organic matter. The residue remaining after ignition consists of mineral matter and gives an index of the inorganic matter in the water. (See Interpretation, page 992.)

The amount of total solids in a water depends upon the character of the soil with which the water has been in contact, the length of exposure, and the amount of carbon dioxide in the water to favor the solution of inorganic salts. Some mineral springs contain very large amounts of total solids, de-

³ U. S. Geolog. Survey. Div. of Hydrography, Circular No. 8, 1902.

rived from deeply situated natural deposits; as, for example, the springs at Saratoga, Carlsbad, Kissingen, etc.

The permissible amount of solids as represented by the fixed residue, which consists of the mineral constituents, cannot be arbitrarily stated, but 500 parts per million are generally held as excessive. From a sanitary standpoint the problem is not alone quantitative, but also qualitative.

HARDNESS

Hard water is objectionable because it wastes soap and affects the skin unpleasantly; it is less satisfactory than soft water for cooking and washing; it produces scale in boilers and is objectionable in some industries, such as paper mills. The traveler is unpleasantly reminded of the effects of abruptly changing from soft to hard water, or vice versa, which are said to cause simple gastro-intestinal disturbances. Myers⁴ states that no evidence has been found proving hard water to be an etiological factor in urinary concretion formation. Upon experimented animals he showed that urinary output of calcium and phosphorus in rabbits was apparently not affected by the salts of hard water, and that in some instances animals developed better on hard than on distilled water.⁵ Opitz⁶ found that communities with very hard drinking water were much better off than those using soft water as regards the frequency of dental trouble and other particulars in school children, registrants for military service, and disability claimants. Those using soft water had less calcification of arteries as judged by those receiving invalidity allowances. Hardness in water, formerly regarded as an economic question, may have an influence upon health, but this has not yet been demonstrated. The problem needs careful study with critical analysis of control groups.

Hardness in water is due to the presence of the soluble salts of the alkaline earths—especially calcium and magnesium. These salts form a curd with soap instead of a lather, hence more or less soap must be wasted in decomposing the lime and magnesia compounds before a lather will form. Thus, one grain of calcium carbonate, for example, will use up 8 grains of soap before a lather can be produced; in this way hard water causes an enormous waste of soap. In this country hardness is expressed in parts per million of calcium carbonate, or in grains per gallon of calcium carbonate. In Europe, hardness is usually expressed in degrees. Each degree corresponds to one grain of carbonate of lime in a gallon of water or its equivalent in other lime or magnesium salts.

To translate parts per million to degrees of hardness, use the following table:

⁴*J. Infect. Dis.*, 1925, 36: 576.

⁵*J. Infect. Dis.*, 1925, 37: 131.

⁶*Deutsche med. Wchnschr.*, 1920, 46: 1391.

CONVERSION TABLE OF HARDNESS

Parts	Parts per Million	Clark Degrees	French Degrees	German Degrees
Parts per million	1.00	0.07	0.10	0.056
Clark degrees	14.3	1.00	1.43	0.80
French degrees	10.0	0.70	1.00	0.56
German degrees	17.9	1.24	1.78	1.00

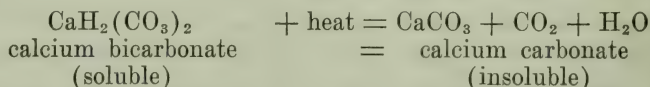
English degrees of hardness (Clark's scale) represent grains of calcium carbonate per imperial gallon.

French degrees of hardness represent parts per 100,000 of calcium carbonate.

German degrees of hardness represent parts per 100,000 of calcium oxid.

To convert hardness from one scale to another, multiply by the factor opposite the scale in which it is expressed and under the scale to which it is to be converted. Thus, to convert parts per million to Clark degrees, multiply by 0.07.

The hardness of water is called "temporary" or "permanent," depending upon the solubility of the salts it contains. *Temporary hardness* is due to calcium or magnesium carbonate held in solution as a bicarbonate by the dissolved carbon dioxid, CO_2 . The hardness is "temporary" because the carbon dioxid is driven off by boiling, and the soluble bicarbonates are precipitated as insoluble carbonates.⁷ The reaction is as follows:



Permanent hardness, on the other hand, is due mainly to sulphates and chlorids of calcium or magnesium. These salts are stable and, therefore, are not precipitated by boiling. Fifty parts per million of calcium sulphate and chlorid of magnesium is usually regarded as excessive.

Water under 4 degrees of hardness may be considered soft, those exceeding 12 degrees, very hard. In the lake cities, 100 parts per million is considered satisfactory, yet such a hardness in eastern Atlantic cities would be considered unsatisfactory. Boiler scale is usually due to deposits of sulphates and carbonates of calcium and magnesium.

Rain water is always soft; surface waters vary, but are usually not very hard; ground waters are apt to be hard.

Two conditions must be present to make a ground water hard: first, the material through which the water passes must contain lime or magnesia, and, second, the conditions must be favorable for dissolving it. The latter practically means that carbon dioxid must be present.

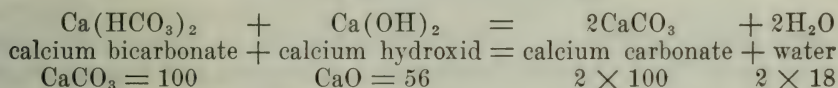
Waters drawn from limestone regions vary greatly in hardness. Rain water contains but little carbonic acid and, therefore, has little power of dissolving lime. The principal source of the carbonic acid in ground water is from the soil, coming from the decomposition of organic matter. The hardness of water, therefore, depends more upon the nature of the catch-

⁷ Calcium carbonate is very slightly soluble in cold water—13 parts per million; magnesium carbonate 106 parts per million.

ment area than upon the amount of lime in the various materials over which the water flows. Thus, the water supply of Vienna is comparatively soft, notwithstanding that it comes entirely from limestone rocks. The mountainous region which forms the catchment area is barren and sterile, and the water does not get the carbonic acid needed to dissolve the lime. The Winnipeg water drawn from limestone underlying the rich prairies is excessively hard. It is interesting to note that many deep well waters of eastern Massachusetts are comparatively soft, although they contain large amounts of carbonic acid.

The Clark Method of Softening Water.—Lime is added to the water in the form of milk of lime. The calcium hydroxid unites with the calcium bicarbonate in the water, forming two molecules of calcium carbonate, which is insoluble and precipitates. In the case of magnesium bicarbonate a molecule of calcium carbonate and one of magnesium carbonate are precipitated; or, if twice the amount of lime is added, two molecules of calcium carbonate and one of very insoluble magnesium hydroxid are thrown down. Sodium carbonate is used to reduce the permanent hardness of water due to sulphates, insoluble carbonate and soluble sodium sulphate being formed.

When bicarbonate of lime or magnesia is treated with lime water the following reaction takes place:



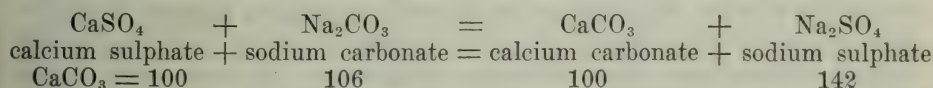
(Note: The hardness of water is always expressed in terms of CaCO_3 , therefore, all calcium and magnesium salts which cause hardness are reduced to terms of CaCO_3 .)

The amount of lime added is weighed as unslaked lime (CaO), therefore, the molecular weight of slaked lime (CaOH_2) is expressed in terms of CaO .)

One grain of CaO per U. S. gallon = 17.1 parts per million = 143 lbs. per million gallons. $17.1 \times \frac{100}{56} = 31$ parts per million of CaCO_3 .

Or, 100 parts per million CaCO_3 will require 3.2 grains of CaO per gallon = 458 pounds per million gallons.

When sulphates and chlorids of lime and magnesia are precipitated with soda ash, the reactions are:



1 grain of Na_2CO_3 per U. S. gallon = 17.1 parts per million = 143 pounds per million gallons.

$17.1 \times \frac{100}{106} = 16$ parts per million of CaCO_3 present as CaSO_4 will be precipitated by 1 grain of Na_2CO_3 per gallon.

Or, 100 parts per million will require 6.4 grains of Na_2CO_3 per gallon = 915 pounds per million gallons.

Softening Water with a Zeolite-Permutit.—This is an efficient and automatic method useful for softening water for household or other purposes. Zeolites have the property of exchanging the sodium base which they contain for other bases brought into contact with them. They exist naturally in soils, where they play the important rôle of holding potassium and other alkaline bases in the soil, thus preventing their being washed away with the rain water.

For the purpose of water softening, *synthetic exchange silicates*, such as Permutit, are now found upon the market. Synthetic exchange silicates are produced commercially by fusing felspar, kaolin, pearl ash and soda. The glass thus produced is crushed and lixiviated to remove the soluble silicates. In passing water through one of these “exchange silicates,” the calcium and magnesium are entirely removed from the water, and exchanged for the sodium of the zeolite. The zeolite can be rejuvenated with a 10 per cent solution of sodium chlorid. The Permutit process is used to soften water in homes, hotels, laundries, textile and other industries, and to prevent boiler scale.

ALKALINITY AND ACIDITY

Most natural waters have an alkaline reaction to ordinary indicators. This is due to the presence of bicarbonates of calcium and magnesium and occasionally to the carbonates of these metals. Some waters in the West also owe their alkalinity to the carbonates and bicarbonates of sodium and potassium. Alkalinity due to salts of calcium and magnesium forms a part of the total hardness, or soap-consuming power; that due to sodium and potassium salts is not a part of the hardness, for these compounds do not react with soap. On the other hand, they tend to make water soft by precipitation of the sulphates of calcium and magnesium in the form of carbonates.

Measured by their hydrogen-ion concentration, many so-called, alkaline waters are really acid to sensitive sulphonaphthalein indicators. This is likely to be the case with soft waters, the alkalinity of which is low and due to bicarbonates of calcium and magnesium. In order to maintain solution equilibrium and hold bicarbonates in solution, an excess of earthy dioxid is necessary. This forms with water the relatively weak carbonic acid. The excess found in low alkalinity waters is usually enough to increase the hydrogen-ion concentration beyond the point of true neutrality, and the reaction is acid, although some basic substances are present. In consequence of this acidity and the low alkalinity, such waters are corrosive to metallic surfaces. Along the Atlantic coastal plain the majority of surface and shallow ground water supplies are of this nature.

In mining regions waters are frequently acid from high quantities not only of carbon dioxid, but also of sulphuric acid and various sulphates—those of

iron and aluminum giving an acid reaction. Mine water is that which is constantly flowing from the coal and surrounding strata. It is collected in ditches at one side of the gangways and tunnels, and is allowed to flow to the lowest point in the mine or to the foot of the shaft, from which it is pumped to the surface. Large quantities of this and other water are used to wash the coal. This water is acid. The spent tan liquors from tanneries are also acid, as are the wastes from many other industries.

Rain water collected in the vicinity of towns has usually a slight acid reaction and acts upon lead. The free acid in rain water is apparently sulphuric, no doubt derived from the sulphur in the coal burned.

When the collection of an acid water cannot be avoided, arrangements should be made for adding lime or some other suitable alkaline substance capable of completely neutralizing the acid. Without some such arrangement there is serious corrosion of all metallic surfaces and the consumers of the water run the risk of lead poisoning, provided lead service pipes are used. A river water suddenly turning acid in reaction plays havoc with a slow sand filter. This has occurred in the Pittsburgh and Providence filters.

CHLORIDS

Chlorin as sodium chlorid or common salt is a normal constituent of all waters. Other chlorids, such as those of calcium and magnesium, are also apt to be present. Traces taken up from the air are found in rain water, especially near the seacoast. The rain water collected at Troy, New York, was found by Mason to average 1.64 parts per million of chlorin. The amounts varied from 0.75 part per million in April to 3 parts per million in October. The chlorid in surface and ground waters, generally speaking, comes from the mineral deposits in the earth; from the ocean vapors and spray carried inland by the wind; also from polluting materials like sewage and trades waste, both of which are apt to contain the common salt used in the household and in manufacturing. A comparison of the chlorin content of a water with that of other waters in the general vicinity known to be unpolluted frequently affords useful information as to its sanitary quality.

The amount of chlorids in water is determined by the well-established silver nitrate method, using potassium chromate as an indicator for the end-point.

Before the water analyst is able properly to interpret the significance of the chlorin content of a water it is necessary to know the normal amount of chlorin present in the waters of that locality. Thus, surface waters near Provincetown, on Cape Cod, contain from 23 to 24 parts of chlorin per million, while surface waters near Boston contain from 3 to 6 parts per million. Near the middle of the state of Massachusetts (Worcester) the surface waters contain only 1.2 to 1.9 parts per million, while in the western portion of the state, farthest from the sea, the surface waters contain but 0.7 to 0.9 part per million. The amount of normal chlorid in the waters

of Massachusetts has been carefully studied by the State Board of Health, and a map has been issued showing the isochlors, or normal chlorin lines.

In Massachusetts the whole of the surface of the country, with the exception of a very small portion, is non-calcareous, and the surface waters carry but little chlorin in composition, if unpolluted, the amount of chlorin decreasing continuously from the coast inland. In a report of the state water supplies, 1887-1890, the Commissioners state that "in a general way 4 families or 20 persons per square mile will add, on an average, 0.01 of a part per 100,000 of chlorid (0.1 part per million) to the water flowing from this area, and a much smaller population will have the same effect during seasons of low flow."

The amount of chlorid in a water of a district varies with several factors, such as the distance from the sea, the amount of rainfall, the amount of evaporation, and the direction of the winds. An increase over the normal is an indication of pollution, and comes mostly from urine. While the ammonia and the nitrites may have disappeared and the nitrates may have been largely taken up by growing vegetation, the chlorid salts, which are exceedingly stable, will be left to indicate remote or passed pollution.

The mixture of even a small proportion of sea-water renders water hard and salty and undesirable for domestic use. It also renders water unsuitable for use in boilers. Wells driven near the sea frequently become mixed with sea-water, particularly if sufficient water is withdrawn to allow sea-water to work back into the wells as an undercurrent. It then gradually mixes with the fresh water above it and sooner or later appears in the well water. When this happens it may be a slow and hard process to operate the well so as to avoid drawing sea-water. In wells near the sea it is important to draw no more fresh water than would otherwise flow to the ocean. This is often a difficult problem to arrange so as to get the maximum quantity of water obtainable. This sea-water question has been more thoroughly and scientifically studied in Holland than elsewhere.

IRON

Iron in water influences its quality from the standpoint of desirability rather than from the standpoint of health. After hardness there is no question of greater practical importance in considering the mineral quality of a water. All natural waters contain a certain amount of iron, and ground waters are apt to contain it in objectionable quantities. No water can be considered entirely satisfactory that contains more than about 0.5 part of total iron per million parts of water. More than this renders water unsuitable for domestic and technical purposes; it stains clothes in the laundry, clogs pipes, interferes with industrial processes and forms sludge in boilers. There is no objection to iron on hygienic grounds, in fact many mineral spring and well waters which are widely sought contain more than would be acceptable in public supplies.

When iron is present in water it supports a fungus (*Crenothrix cohn*), an organism which may grow in pipes in sufficient amounts to obstruct the flow of water or even completely choke them. This sometimes occurs in the pipes of driven wells. It is chiefly troublesome in ground waters containing organic matter and iron. *Crenothrix* was the cause of the complete obstruction of the water pipes in the New York Custom House in 1907.

Iron is very widely distributed and exists in practically all sands, gravels, soils, and rocks with which water comes in contact. The solution of the iron is partially brought about by the organic matter. The iron exists in the soil both as ferrous and as ferric compounds. The latter are reduced by the organic matter to ferrous salts, which are soluble in water containing carbonic acid. Iron is also a constituent of certain complex plant material and will be found in surface waters in organic combination. Trouble from iron is always to be expected when there is an excess of organic matter in the material through which the water passes. In a well-drained, porous soil the oxygen from the air circulates in the pores of the soil and furnishes what is required for the oxidation of the organic matter. Iron is not dissolved under these conditions, even in the presence of large amounts of organic matter, but if the air supply is cut off, as for instance in case the pores of the soil are filled with water, the solution of iron is sure to take place. The iron is dissolved in the form of ferrous salts, usually ferrous bicarbonate. When ground waters containing iron are first drawn they look clear, but the ferrous salts in solution are soon oxidized on contact with the air to insoluble ferric salts, which are precipitated as red hydroxids.

Iron Pipes.—Nearly all waters attack iron pipes, corroding them and forming tubercles on the inner surface. This is objectionable, because it reduces the carrying capacity of the pipe and also influences the quality of the water.

Tubercles are formed as follows: The organic matter in the water settles in the pipe and decomposes, forming carbon dioxid, which acts upon the iron, causing some of it to go into solution as ferrous bicarbonate. The soluble ferrous bicarbonate for the most part passes on in the flowing water, but some of it becomes oxidized by the oxygen in the flowing water and is precipitated as the insoluble ferric carbonate or hydroxid and remains at the surface of the deposit. The iron precipitated in this way acts as a coagulant upon the organic matter in the flowing water at the point where the iron is precipitated. It thus attracts the organic matter from the flowing water and binds it to that previously deposited into a firm, compact, but porous mass, and this mass is the beginning of a tubercle. The process is continuous, though slow. Many years may elapse before the tubercle reaches the height of an inch. Tuberculation starts more freely and progresses more rapidly in waters from rivers or reservoirs containing suspended organic matter. It is less troublesome with filtered waters, and with lake waters relatively free from such suspended matter. Tuberculation may be prevented by improving the quality of the water or by thoroughly coating the inside of

the pipes with asphaltum or tar. Cement-lined pipes are not subject to tuberculation, but have defects in other particulars. When the process has advanced far it may be corrected by pipe scrapers. They consist of appliances driven by the water pressure through the pipes, with blades to scrape off the tubercles. This temporarily increases the carrying capacity of the pipe, but the process must be repeated at intervals. It has the disadvantage of also scraping off a large part of the tar coating and leaving the iron of the pipe exposed to the action of water to a much greater extent. (Hazen.)

Water that passes through the iron water-back of the kitchen stove to the hot-water tank is particularly likely to collect iron, which accumulates at the bottom of the hot-water tank. This deposit may accumulate for days and even weeks until some unusual draught of water or other disturbance occurs—perhaps on washing day—causing a stirring up of the iron precipitate. This condition is very objectionable.

Removal of Iron.—The household filter is the most convenient and satisfactory means of removing iron deposits from water that is otherwise good. The removal of iron from a city's water supply is a process often combined with purification. In most cases iron may be removed by thoroughly aerating the water in order to drive off the excess of carbon dioxide and in order to introduce oxygen necessary to oxidize the iron from the soluble ferrous state, in which it exists, to the insoluble ferric state. The precipitated ferric salts can then be removed by sedimentation or, better, by filtration.

LEAD

The presence of lead may be discovered by chemical tests; more often it is surmised from the symptoms of lead poisoning among those who use the water. In the amounts present it does not affect either the appearance or taste of the water.

The sample of water used for testing lead should be the *first portion* (a pint or less) drawn after standing at least one hour in the pipes. Very often the sample examined will not represent the daily maximum.

A great many waters contain lead, though in small amounts. The private water companies in the United States place a limit of 0.5 milligram per liter, while certain towns in England allow 1 milligram per liter. The 1925 Report of the Advisory Committee on Drinking Water Standards, United States Public Health Service, states that the presence of more than 0.1 part per million of lead shall constitute ground for rejection of a supply. The question of lead poisoning and its relation to water is discussed on page 1050.

ORGANIC MATTER AND NITROGEN

Considerable information regarding the sanitary history of a water can be obtained from the determination of nitrogen in all its forms, organic and inorganic.

Organic matter in water originates from both plant and animal sources

and appears in a great variety of compounds, most of them too complex or too small in quantity to allow of direct determination. There is only one direct determination of organic constituents which is ordinarily made. That is the "loss on ignition" of total solids, which gives the weight of volatile and combustible organic constituents. Ignition is subject to the error of recording unavoidable loss of some mineral compounds. Nevertheless, a fair measure is obtained of the total weight of organic substances as indicated by the loss of combustible material.

Other determinations are made, indirect in character, which provide an index of organic pollution. The most valuable of these are the nitrogen determinations. All protein matter contains nitrogen, whether it be of plant or animal origin. The organic matter in water comes from both these kingdoms and the protein exists in both living and inert substance in all changes of the nitrogen cycle. The cells of bacteria and algæ, protozoa, and other microscopic forms contain nitrogen, and it is present in extracted vegetable matter and in animal refuse products. It is, therefore, present in suspended and colloidal particles and in smaller amount in dissolved substances. The total organic nitrogen may be determined by the well-known Kjeldahl method, and it may be divided by filtration of a sample before analysis into suspended organic nitrogen and dissolved and colloidal organic nitrogen. The former includes that from microscopic organisms, except the bacteria, and organic debris of every sort; the latter, that from extracted and dissociated material. The organic nitrogen determination does not differentiate between plant and animal nitrogen, but high values generally point to animal pollution because of the higher nitrogen content of animal tissue. High values also indicate fresh pollution—that is, that time enough has not elapsed for bacterial digestion of organic matter and oxidation of the nitrogen to more stable forms.

Albuminoid Nitrogen.—Ordinarily the total organic nitrogen is not determined because strong reagents, heat, and a considerable period of digestion are necessary. This is due to the difficulty with which some of the nitrogen is oxidized. Therefore, a simpler and quicker procedure, known as the "albuminoid nitrogen," or "albuminoid ammonia" test is used. This method oxidizes only that portion of the nitrogen which is not held in stable combination and yields it as ammonia. The albumins behave in this way, hence the term "albuminoid nitrogen." The method consists in distilling a sample of water with strong alkaline permanganate, and Nesslerizing the distillate for ammonia. The amount of albuminoid matter in the water is determined by the amount of ammonia in the distillate, expressed in terms of nitrogen.

The figure obtained is approximately one-half the total organic nitrogen which exists in ground waters or surface waters which have little pollution. In sewage and waste-polluted waters the albuminoid figure is variable and usually more than half the total nitrogen because nitrogenous matter of animal origin contains not only more nitrogen, but more nitrogen in the form of easily decomposable compounds, such as the albumins.

Like the Kjeldahl method this one also fails to distinguish between plant and animal nitrogen. Ordinarily high albuminoid nitrogen values are due to a large number of microscopic organisms or to sewage pollution. The determination, therefore, provides a fair index of the organic pollution in water. The organic matter itself is not dangerous to health, but is undesirable because it offers food for bacterial growth, defiles the water, and produces disagreeable tastes and odors. Any indication which can be obtained that the organic pollution is of animal origin has greater significance than an indication of plant origin because of the likelihood of disease organisms accompanying the former.

No arbitrary standard can be set as to the maximum amount of albuminoid nitrogen a good water may contain. It is not uncommon to find as high as 0.10 part per million in surface waters of acceptable quality, but collateral evidence should show this amount to be principally of plant origin. Ground waters of good quality contain less than this amount; in most cases practically none.

Free Ammonia.—Ammonia nitrogen is usually spoken of as “free ammonia” because it can be driven off or freed by boiling. The amount of free ammonia is determined by distilling a 500 c.c. sample and Nesslerizing the first 200 c.c. of the distillate in 50 c.c. portions. Nessler’s solution contains mercuric chlorid and potassium iodid and gives a brown color in the presence of ammonia, due to the formation of mercuric ammonium iodid. The amount of ammonia formed is expressed in terms of nitrogen.

Ammonia does not usually exist in water in its free state but in the form of its salts as ammonium carbonate and ammonium chlorid. The sources of ammonia in water are varied; usually it comes from the decomposition of organic matter, as a result of bacterial action. Ammonia is not the last stage in the mineralization of organic matter (see Nitrogen Cycle, page 913), for with the lapse of time ammonia is oxidized by bacterial action to unstable nitrites and finally to stable nitrates. Both the time and the processes necessary to effect nitrification bring about destruction of organic pollution and bacterial purification.

Free ammonia is also formed during the process of denitrification, by which nitrates and nitrites are reduced to ammonia. This action takes place only near the surface of soil and to a limited extent. In natural waters, therefore, this source is not significant.

The ammonia itself found in drinking water is harmless; its significance lies in the fact that it indicates the presence of decomposing organic matter which may be accompanied by germ-laden material inimical to health. The organic matter itself is only objectionable on æsthetic grounds. High ammonia values signify recent pollution, that is, within a period of time which has not allowed oxidation. If such pollution can be traced to animal origin, it carries an important warning of danger.

Not all the free ammonia in water originates from organic matter. Rain water takes free ammonia from the atmosphere. Angus Smith and Boussin-

gault place the average amount of ammonia in the rain of temperate climates as 0.5 part per million. The amount of ammonia in rain water was studied by Filhol. He found that in the city of Toulouse the rain contained 6.60 parts per million, while the rain water collected outside the city contained only from 0.44 to 0.77 part per million. These figures show the marked difference between city and country rain. The presence of ammonia in clean and properly stored rain water has little significance. It registers atmospheric pollution with gases.

Nitrites.—Nitrites never accumulate to any extent in water because they are soon oxidized to nitrates, the final and stable form of mineralization of organic nitrogen. Nitrites represent the transitorial stage of decomposition between ammonia and nitrates. "The state of change is a state of danger." Further, nitrites are a sign that complete oxidation of protein matter has not been accomplished, and therefore indicate that there may yet remain bacterial pollution of dangerous character. Nitrites, when high, suggest bacterial activity. Nitrites are an index, just as the colon bacillus is an index. Their significance varies with the amount, the source, and the relation to other constituents in the water.

Nitrites have less significance when ammonia nitrogen is low and nitrate nitrogen is high, and greater significance when the reverse is the case. A low ammonia content is evidence of little protein material in a state of decomposition.

In unpolluted ground waters nitrogen will not be found as nitrites, except as there may have been reduction of nitrates by ferrous compounds or other reducing agents. When this happens the amount of nitrites is likely to be high and can be disregarded in the light of other tests which register an absence of pollution.

Concerning the amount of nitrites in water it must be remembered that the colorimetric test for nitrites with sulphanilic acid and α -naphthylamin acetate is one of the delicate tests in chemistry. With this method we are able to detect quantities of nitrogen as small as one part in a thousand million. When, therefore, a water analyst reports a trace of nitrites it means an exceedingly minute quantity.

Nitrites are poisonous compounds, but the minute amounts found in water can scarcely have a pharmacological effect.

Nitrates.—Nitrates represent the final stage in the mineralization or oxidation of nitrogen originally present in organic compounds. Nitrates are used as food by plants for the upbuilding of new protein, which important step completes the circle that nitrogen passes through in its continuous circulation in nature. (See Nitrogen Cycle, page 914.)

The detection of nitrates in water depends upon the yellowish color produced by phenoldisulphonic acid, and the amount can be determined by comparing with standard solutions.

In the same sense that nitrites may suggest danger, nitrates may indicate safety. Nitrates being the final stage of protein decomposition are a

sign that decomposition of protein is complete, provided organic nitrogen and nitrites are absent. The presence of nitrates is not of itself evidence of safe quality in drinking water, for nitrates must be interpreted in the light of the bacteriologic findings and other contributing data. When the nitrates are high but other forms of nitrogen are absent or present only in traces, it means that the water was polluted, but purification has taken place. Under these circumstances nitrates tell the chemical story of the past history of the water. An illustration of this is given on page 1000.

Nearly all surface waters of safe quality contain nitrogen as nitrates in amounts varying from 0.10 to 1.0 part per million. There is considerable seasonal fluctuation due to use of nitrates for food by algæ and other forms of plant life. Nitrates in surface waters come largely from the soil where there is constant mineralization of nitrogenous organic matter. When surface water has been heavily polluted at some distant point with either soil washings or liquid wastes the nitrate content may be several parts per million of nitrogen.

Shallow ground waters often show large amounts of nitrates, from 1.0 to several parts per million. This may be due to oxidation of soil nitrogen or of wastes thrown upon or in the ground. If the latter, there is a considerable element of danger shown by the nitrate content, unless organic matter and other forms of nitrogen are practically absent.

High nitrates in deep ground waters usually have their origin in underground mineral deposits and are without sanitary significance. Young⁸ has shown that the ground waters of Kansas sometimes contain large amounts of nitrates—as much as 500 parts per million. The medicinal dose of potassium nitrate is 0.3 gram. Less than a liter of such water would, therefore, contain sufficient nitrates to produce effects such as irritation of the mucous membrane of the stomach, and also diuresis, with irritation of the mucous membrane of the bladder.

Oxygen Consumed.—One of the oldest tests for organic matter in water is that with potassium permanganate and acid. Unstable organic compounds are oxidized by the permanganate, which in turn is reduced and decolorized. The test has a quantitative application. The amount of permanganate used up may be expressed in terms of oxygen, and this is the “oxygen consumed” by the oxidizable matter present.

There are several facts which operate to impair the usefulness of this test. In the first place, the oxygen values will vary with the temperature and time of digestion. It is, therefore, important to record the conditions of the test, or results will not be comparable with the results of others. Again, the determination tells nothing as to the source or kind of organic matter. It was thought at one time that carbonaceous organic matter was oxidized in this way in contrast to nitrogenous material by the albuminoid nitrogen test, and that the results accordingly registered pollution by plant material.

⁸ *J. Am. M. Ass.*, 1911, 56: 1881.

This is only partially true, and the test in no way can be used to distinguish between organic matter of plant and animal origin. Neither does it record the oxygen necessary to stabilize all the organic matter present, for the test is not carried that far; nor does it give much idea of the ease with which the organic material is oxidized—that is, how unstable it may be. However, rapid absorption of permanganate at the beginning of the experiment indicates a large amount of easily oxidized matter.

Certain chemical substances of mineral nature, such as ferrous salts, nitrites, and sulphids, give false values as they are capable of absorbing oxygen from potassium permanganate.

Oxygen-consumed values when used alone are not of great significance but give corroborative information in interpreting the results of other determinations, such as color, loss on ignition, organic nitrogen and albuminoid nitrogen. They are also useful in comparing periodical fluctuations in oxidizable matter.

Good ground waters show practically no oxygen consumed, for they contain very little organic matter. In surface waters free from waste pollution the oxygen consumed will usually vary as the color, ranging from 1.0 to 10 parts per million.

DISSOLVED OXYGEN

Water in contact with the air becomes saturated with oxygen. Being held in solution, as a gas in a liquid, the oxygen is termed “dissolved oxygen.” This determination has no relation to “oxygen consumed.” The actual amount of gas present will depend upon the partial pressure of oxygen in the atmosphere and also upon the barometric pressure and the temperature of the water. In most cases temperature is the controlling factor, for the amount of oxygen dissolved varies greatly with temperature. Thus, distilled water at 0° C. and 760 mm. pressure holds, when saturated, 14.62 parts per million of oxygen; at 20° it holds 9.17 parts. Without oxygen water has a flat, insipid taste.

There are many processes which go on in water that influence the dissolved oxygen content. Clean surface waters are ordinarily saturated, but in the presence of rapidly growing plant life, particularly algæ, supersaturation often occurs due to liberation of oxygen by the plant cells. At the same time there is a reduction of the carbon dioxid content which is used as food for plant growth. The relation of dissolved gases to microscopic growth in water is close and important.

When water is polluted with any oxidizable material such as the extracted matter from dead vegetation, sewage, or other wastes the oxygen content will be diminished, but may be replenished by absorption from the atmosphere if the pollution is not heavy and decomposition not too rapid. When the demand of bacterial activity for oxygen exceeds the rate at which oxygen can be dissolved from the air the value in the water will fall and as it approaches zero putrescible conditions arise due to anaërobic bacterial processes. This

results in the production of partially oxidized compounds which possess obnoxious physical properties. In surface waters receiving sewage or industrial wastes this condition may establish a nuisance.

Dissolved oxygen is a most important determination in the study of pollution. The amount of oxygen found is an indication of the load which is being put upon natural processes of purification. The amount found in the water of a running stream at different points, for instance, furnishes valuable information as to the rapidity with which self-purification is taking place below a given point of pollution.

Oxygen bears an important relation to fish life. Most fish will live when the oxygen content is as low as 50 to 60 per cent of saturation. Below this point suffocation takes place, the critical value depending upon the variety of fish.

Nearly all lakes and impounded water supplies show a decrease in oxygen content with increasing depth. Frequently none is found near the bottom during the summer months. This results from the temperature stratification of the water which creates a zone of stagnation at the bottom. Due to its greater density the water here does not reach the surface for aëration, and bacterial decomposition at the same time is more active than at the surface, due to concentration of organic impurities from natural sedimentation. Water in the zone of stagnation is impregnated with foul products of decomposition and should be avoided for general domestic use (see page 954).

Ground waters are usually partially or wholly depleted in oxygen content, due to percolation of the surface water through the soil where oxidation of organic matter and, also, of mineral deposits is in progress.

CHAPTER III

MICROSCOPICAL EXAMINATION OF WATER

The chief object of the microscopic examination of water is the determination of the presence or absence of those microorganisms which produce objectionable tastes and odors. In certain cases the determination is also of value as an index of pollution or as a guide to the identity of the water. The microscopical organisms for this purpose comprise the Diatomaceæ, Chlorophyceæ, Cyanophyceæ, Fungi, Protozoa, Rotifera, Crustacea and other organisms minute in size, but not including the bacteria. Fragments of organic matter, broken-down organisms, zoöglea, etc., should be termed amorphous matter. Clay, silt, óxid of iron, and mineral matter in general are not included under amorphous matter and are not measured by microscopic examination.

The term "microorganisms" as used by the water analyst includes all organisms, whether plant or animal, that are invisible or barely visible to the naked eye, other than bacteria. The bacteria are set apart, inasmuch as their significance and the method of studying them are different from all other microscopic organisms. As Whipple aptly phrases it, "Bacteria make a water unsafe, microorganisms make it unsavory."

The sanitary quality of water cannot be definitely shown from a microscopical examination. Surface waters are usually rich in microscopic life, while ground waters are comparatively free. However, as soon as ground waters stand in pipes or are exposed to the light, microscopic organisms develop.

"Plankton" is the general name given to the microscopic aggregation which is investigated in any given sample of water. The term as used embraces plants and animals that float about in the free state, also larvæ, egg masses, etc., of higher animals. It includes diatoms, algæ, fungi, protozoa, etc.¹

The Sedgwick-Rafter Method.—This is the standard method for counting the number of microscopic organisms in water. It consists in collecting the microscopic particles suspended in a known quantity of water, and counting them in a cell of known capacity under the microscope. The microscopic particles are collected upon sand by filtration. Full details of the procedure may be found in *Standard Methods of Water Analysis*, page 103.

There are many sources of error in a quantitative determination of the microscopic organisms in water. Some of the organisms stick to the sides

¹ For a full discussion of this subject see *The Microscopy of Drinking Water*, by George C. Whipple, Wiley & Sons, New York, 1914.

of the funnel; some pass through the sand; some are so heavy that they settle in the sample, especially if it has been allowed to stand; some are so fragile that they disintegrate readily, and further error may be due to unequal distribution of the organisms in the counting cell. The errors are to some extent compensating, but accuracy is not to be expected within less than 10 per cent deviation from the true results, and comparative results may be obtained only by careful standardization of methods.

Significance of the Examination.—The microscopical examination of water is of great value in supplementing the chemical and bacterial analyses. It may explain the cause of odors and tastes in a water; it may explain certain chemical determinations, as albuminoid ammonia, dissolved oxygen, oxygen consumed, carbon dioxid, etc.; it may indicate sewage contamination; it may suggest the state of self-purification of a polluted water; it may identify the source of the water.

Several of the microscopic organisms, when present in sufficient quantities, give rise to objectionable odors and tastes, either when in a vegetative state or upon decomposition. The natural odors of organisms are due to oils analogous to the essential oils, as in peppermint. In general, the diatoms have an aromatic odor, increasing to that of a geranium leaf, and even to an intensity that is fishy. The cyanophyceæ, or blue-green algæ, have a grassy or musty odor. The chlorophyceæ have little odor, although some of the motile forms give rise to faintly fishy odors. The ciliated protozoa have in general no odor. Uroglena, synura, dinobryon, and peridinium may and often do give rise to fishy odors. Of the other microorganisms, the rotifera and crustacea, no forms have been recorded as giving rise to objectionable odors. These forms are present only when there are lower forms upon which to feed. They are scavengers and as such may be considered as desirable elements in water. Their presence, however, calls for an investigation of the nature of their food supply, as it is often furnished by pollution. This does not necessarily hold true in all cases.

Besides these animal and plant forms there may be present also sponges, mosses, yeasts, and molds, the significance of which is varied and dependent upon local conditions.

THE BACTERIOLOGICAL EXAMINATION

Practically all natural waters contain bacteria. This is true of rain water, ground water, and the waters of lakes, rivers, and oceans. The number and variety of the bacteria vary greatly in different places and under different conditions. The bacteria are washed into the water from the air, from the soil, and from almost every conceivable object. The intestinal contents of animals pollute waters with enormous numbers of microorganisms, but it is the infection with certain species from man that makes water most dangerous when consumed by his fellowmen.

A final judgment of the potability of a water should never be based upon

the bacteriological examination alone, but should be combined with a sanitary survey and other laboratory tests. A sanitary survey may discover possibilities of danger, even though the sample examined contains few bacteria and no *Bact. coli*. On the other hand, a water may contain large numbers and miscellaneous kinds of harmless bacteria and a sanitary survey will confirm absence of human pollution. The chemical and microscopical examinations assist in the interpretation of bacteriological findings.

NUMBER OF BACTERIA IN WATER

The number of bacteria is not as important as the kind, yet much may be learned from a simple enumeration of the bacteria. Roughly speaking, the number of bacteria in water corresponds to the amount of organic pollution. No known method furnishes a complete census of the bacterial population of a given sample of water. Methods based upon the direct microscopic count of the bacteria do not distinguish between the live and the dead ones; further, only those that may readily be seen by simple methods are thus visible. Many bacteria, especially those pathogenic for man, do not vegetate at 20° C., so that the usual counts upon gelatin may vary greatly from those obtained upon agar at 37° C. Some varieties require acid, others alkaline, media; some are aërobic, others anaërobic; some will not grow unless the medium contains blood or other suitable pabulum, and so on through a wide gamut of conditions.

Although it is not possible to determine the total number, inferences of importance may be drawn from the counts obtained by standard methods. Significance also is attached to the differences in the numbers of bacteria in a given water obtained by different methods. Thus a water containing great numbers of bacteria, developing on gelatin at 20° C., and but few colonies upon agar at 37° C., has little sanitary significance, whereas a water containing few bacteria, but most of them varieties that grow upon agar at 37° C., with relatively few at 20° C., must be looked upon with suspicion. The distinction between contaminated waters and waters of good quality is more sharply marked by counts at 37° C. than is the case with counts at 20° C. Another advantage of growing the plates at a higher temperature is that the results are available in a much shorter time.

The number of bacteria which grow at 40° C. may be of special interest, since this class includes the typhoid bacillus and other water-borne pathogens, but excludes the common water bacteria which have little sanitary importance.

From Germany we have the arbitrary standard based upon the dictum of Koch that a good water should not contain over 100 bacteria per c.c. This is a good working rule, but should not be taken too literally. Thus, water may contain great numbers of the common aquatic bacteria which vegetate at room temperature and which are not harmful to man. Surface waters contain the greatest numbers on account of exposure to pollution to which

they are liable; rain waters contain comparatively few, excepting the first shower through a very dusty atmosphere; ground waters from the depths are practically sterile. Unpolluted shallow well waters are also exceptionally free. The number and significance of the bacteria, therefore, vary with the source of the water. For example, a hundred bacteria, including a few colon bacilli, of the fecal type, in a ground water would be regarded with great suspicion, whereas a hundred or more bacteria, with an occasional colon bacillus, in a river water draining an uninhabited watershed would be normal.

The number of bacteria in water depends somewhat upon the manner in which it is stored. Thus a water containing a few organisms placed in a closed bottle and kept at room temperature may, at the end of twenty-four hours, contain hundreds or thousands per c.c. I once examined a deep well water that was practically sterile as it came out of the earth, but when stored in a cistern gave over a thousand organisms per c.c. These came from the multiplication of the many ordinary and harmless bacteria that entered the water from the air, dust, leaves, and other sources. On the other hand, water stored in impounded reservoirs shows a marked diminution in the number of bacteria.

The numerical determination of bacteria in water is of very great value when studying surface waters, such as lakes and rivers. As a rule, the number of bacteria is proportional to the pollution of a river—not necessarily fecal pollution, but pollution from dead organic matter of one kind or another. The bacterial content of a river water varies sharply from time to time. Contrary to the usual opinion, the highest counts are not obtained in the warmest months, but rather at other seasons when freshets and heavy scour wash in large amounts of suspended impurities. The turbidity so contributed is laden with bacterial life. An exception to this general rule is found in streams which are grossly polluted with sewage, like the Merrimac. They show the highest bacterial content at times of low flow, and this is diluted by flood waters. It is an interesting fact that in the Potomac and other rivers the bacterial curve does not correspond to the typhoid fever curve. Typhoid in Washington is highest in summer, but the bacteria are most numerous in winter. Sudden variations in the number of bacteria have a ready explanation in the case of turbid and torrential rivers; in the case of lakes, and especially in a ground water, variation in numbers indicates nearby sources of pollution and is a danger signal. For shallow wells the interpretation of numbers is not so easy. Wells which are poorly protected at the top will always show an unusually large number and variety of bacteria, but these may be harmless soil forms and have no more significance than they do in surface waters.

Numerical determination is also of importance in tracing imperfections and leaks in a water supply. Thus Shuttleworth, of Toronto, was able through this means to suspect a broken water main, and upon examination it was found that a whole section of the conduit had dropped out of place,

so that the supply was being taken from the lake near the shore instead of some distance away where the intake was located.

The great value of the numerical estimate of bacteria is well known in determining the efficiency of water purification and sewage treatment processes.

The Standard Methods of the American Public Health Association for the bacteriological examination of water give concordant results and should be closely followed in all details, in order to have results that are comparable with others.

KINDS OF BACTERIA IN WATER

Water analysis is in its infancy so far as methods for determining the kinds of bacteria are concerned. It is comparatively easy to isolate colon bacilli and to determine their approximate number in a water. It is also comparatively easy to isolate the cholera vibrio. Methods for determining whether a water does or does not contain typhoid, dysentery, and other pathogenic parasites are tedious, difficult, and often impossible in the present state of our knowledge.

A certain amount of information may be gleaned from the presence and number of organisms belonging to certain groups, such as chromogenic, liquefying, and fermenting types. Chromogenic organisms exist everywhere in surface waters. They should be practically absent from ground waters. The same is true of organisms that are able to liquefy gelatin and ferment sugars. The chromogenic, proteolytic and fermenting types are widespread in nature and exist almost everywhere in the air, the soil, and in surface waters. Their presence in a ground water signifies contamination or pollution, often from the surface.

The significance of the various types of bacteria that grow at different temperatures has already been discussed.

THE COLON BACILLUS

Bacterium coli belongs to a group that is very widely distributed in nature. The normal habitat of most members of this group is in the intestines of man and many other animals. The colon bacillus is usually taken as an index of pollution. The sanitary significance of colon bacilli in water varies with their number and, further, with their source. While the colon bacillus indicates contamination, it does not necessarily signify immediate danger, that is, infection.

The coli-aërogenes group includes all Gram-negative, non-spore-forming bacilli which ferment lactose with gas formation and grow aërobically on standard solid media.

By common consent a ground water should be condemned if it contains even a few colon bacilli of fecal type, for these organisms have no business in a soil-filtered and properly protected well or spring water. Surface waters are not regarded as particularly suspicious, provided they have not more than

one colon bacillus per 50 c.c., especially if the surface water is known to drain an uninhabited or controlled catchment area. Many of the colon bacilli in a surface water come from the droppings of wild and domestic animals and birds and, therefore, are infinitely less indicative than those that come from the intestinal tract of man. Tests for the colon bacilli in water must, therefore, be qualitative and quantitative.

The most important work of recent years in the field of water bacteriology has been that of the differentiation of various species of the coli-aërogenes group and the establishment of their habitat. It has led to a clearer understanding of the significance of these forms in water. For a discussion of this work and details of the methods for identification the references given in the footnote should be consulted.²

Types of Colon Group Organisms.—Two types are now recognized: (1) fecal or intestinal and (2) non-fecal or aërogenes.

The former have their normal habitat in mammals and birds, and even in some cold-blooded animals, and therefore are well named fecal bacteria. Colon bacilli of the non-fecal type are found in soil, seeds, fruit, leaves and very many other places in nature. The two types are fairly well differentiated, although feces of man and other animals as a rule contain a small percentage of the non-fecal type of organisms.

The difference between the two types depends upon the following reactions: (1) the methyl red test, (2) the Voges-Proskauer reaction, (3) uric acid broth, (4) sodium citrate broth; to which was formerly added (5) the gas ratio.

The Methyl Red Test.—The fecal type of *Bact. coli* produces a relatively high concentration of hydrogen ions in dextrose broth, and this correlates very well with low gas ratio. The non-fecal type, in addition to having a high gas ratio, is characterized by a relatively low hydrogen-ion concentration. This may be determined by the addition of methyl red to cultures of dextrose broth. The former (fecal type) will give a red color—acid or methyl-red-positive, and the latter (non-fecal type), yellow—alkaline or methyl-red-negative.

The Voges-Proskauer reaction is also useful in differentiating between the various members of the coli-aërogenes group. To 5 c.c. of dextrose broth culture add 5 c.c. of a 10 per cent solution of potassium hydroxid. Allow this to stand twelve to eighteen hours. A positive test is indicated by an eosin pink color, and is seldom given by a positive-methyl-red organism. This production of pink color with a fluorescence is probably due to action of acetyl-methyl-carbinol (one of the by-products in decomposition of dextrose by bacteria) on peptone in the presence of alkali.

Uric Acid Test.—*Bact. coli*, or the fecal type, will not grow in the presence of uric acid, 0.5 gram per liter, while aërogenes will, because *Bact. coli*

² Prescott and Winslow, *Elements of Water Bacteriology*, Wiley & Sons, New York, 1924, 4th Ed. "Bacteria Fermenting Lactose and Their Significance in Water Analysis." Max Levine, 1921. Bulletin No. 62, Iowa State College.

cannot obtain nitrogen from uric acid and other members of the purin group which it is unable to break down.

Sodium Citrate Test.—*Bact. coli* does not grow in broth containing 2.5 to 3 grams sodium citrate in one liter, while the non-fecal type, *aërogenes*, does.

The Gas Ratio.—The earlier workers in this subject defined *Bact. coli* as an organism producing gas from dextrose, consisting of one volume of carbon dioxid to two volumes of hydrogen. More recent work indicates that the fecal coli usually produce carbon dioxid and hydrogen in about equal volumes, and are thus low gas ratio organisms. Non-fecal (*aërogenes*) organisms usually differ from the fecal type in that they produce carbon dioxid and hydrogen in a ratio of 2:1 or 3:1 (high ratio type).

Presumptive Test for *Bact. Coli*.—Presumptive or partial tests for the presence of *Bact. coli* are fairly reliable and afford useful information. The test consists in inoculating a 10 c.c. portion of sample into each of five fermentation tubes containing from 30 to 40 c.c. of lactose broth. In order to obtain a sufficiently low dilution, so that no fermentation will result in one tube in the series 1 c.c., 0.1 c.c., 0.01 c.c., or even 0.001 c.c. (if heavy pollution is suspected) portions of the sample may be inoculated into lactose fermentation tubes. When such small quantities of water are tested, less than 30 c.c. portions of media should be used. The fermentation tubes described above should be incubated forty-eight hours at 37° C. The presence of 10 per cent or more of gas constitutes a presumptive test, since the majority of bacteria which give such a reaction belong to the group.

If in such a series fermentation with gas production occurs in the tubes containing 1 c.c. or more, but does not take place in the tubes containing the smaller portions, it may then be stated that the water contains at least one colon bacillus per cubic centimeter, *provided* the isolation tests show that the fermentation was caused by this organism.

It may be presumed that in the absence of fermentation colon bacilli are absent, and that fermentation with gas production suggests their presence. Both these conclusions may be misleading. Grossly polluted waters containing many colon bacilli may be slow in fermenting sugars with the production of gas on account of the preponderance of other more active species. On the other hand, many organisms other than the colon bacillus often found in water ferment sugars with gas production. It is therefore necessary to isolate the suspected organism in pure culture and pass it through the well-known tests before it is labeled *Bact. coli*.

Confirmation Tests for *Bact. Coli*.—Cultures should be made on Endo's or eosin-methylene-blue-agar plates from the tube showing gas with the smallest portion of sample, and incubated twenty-four to forty-eight hours. If typical colon-like colonies form, at least two from each sample should be inoculated on agar slants and in tubes of lactose fermentation medium for further confirmation. If, after twenty-four hours, no typical colon colonies develop on the Endo's or eosin-methylene-blue-agar plates, two colonies

appearing nearest like coli are treated as above. From the agar slant, examinations may be made to demonstrate that the organism in question is a short, Gram-negative, non-spore-forming rod.

The number of colon bacilli may also be determined approximately by planting the water directly upon the surface of lactose litmus agar or Endo's medium. The red colonies should then be studied to determine how many of them are *Bact. coli*, and the number may thus be approximated per cubic centimeter.

Significance of *Bact. Coli*.—The absence of colon bacilli in water proves its harmlessness so far as bacteriology can prove it. It is fair to assume that typhoid bacilli, dysentery bacilli, and other intestinal parasites would not be likely to be present in a water in the absence of the colon bacillus. It is possible to conceive that in rare instances a water may be polluted with urine alone containing typhoid bacilli, but no colon bacilli.

The colon bacillus, then, is a danger signal; its absence a sign of safety. Final judgment, however, should not be passed on the presence or absence of *Bact. coli* alone. Their number and type are significant, and furthermore, the findings should be interpreted in the light of other information obtained in the laboratory and in the field.

INTERPRETATION OF SANITARY WATER ANALYSES

The interpretation of a sanitary water analysis is much more difficult than the analysis itself, where everything may be carried out in accordance with standard procedures. Single or occasional determinations of either the chemical or bacterial properties of water are of limited value. A single water analysis may even be misleading, especially in surface waters, which vary greatly from time to time. A river water may require repeated examinations extending over long periods of time correlated with conditions of rainfall, stream flow, wind, temperature, sewage pollution, and other factors in order to secure helpful information.

There has been much conflict and useless discussion between chemists and bacteriologists concerning the relative advantages of their methods. The chemists were first in the field, but the limitations of chemical methods were strongly emphasized when it was shown that chemistry can only indicate pollution but cannot discover infection. Much was hoped from bacteriology, but it is rather exceptional that bacteriologists are able to isolate pathogenic microorganisms from a sample of water. For the most part, the routine bacteriological examination of water does nothing more than the chemical examination, that is, it shows contamination but does not prove infection. Both chemical and bacterial analyses of water have, therefore, distinct limitations; they do not antagonize, but supplement each other. From the chemical side we learn much of the past history of a water; the bacteriology tells us more of its present state. Chemical methods reign supreme when we desire to discover the presence of lead or other inorganic poisons; also in deter-

mining the hardness, mineral and organic constituents, etc. From the number and character of the bacteria in water we obtain a fair index of the presence and degree of pollution. Occasionally bacteriologists may determine whether a water contains certain specific agents, such as cholera vibrio. It must, however, be admitted that the ordinary routine chemical and bacterial examination of water affords but meager information, especially when only one analysis has been made. Fortunately, the inferences drawn from a sanitary water analysis are on the safe side, and many good waters are condemned; in fact, it is very difficult for an unsafe water to pass the muster of a complete sanitary analysis.

At most, the information furnished is only of present conditions and is not a guarantee of future safety. A surface water or a ground water may to-day be exceptionally free from chemical impurities and practically sterile bacteriologically, whereas to-morrow it may contain typhoid, dysentery, cholera, or other water-borne infection; these may come from sources that would at once be evident from a sanitary survey of the watershed.

A sanitary survey of the catchment area is frequently of much greater practical importance than all the information furnished by the laboratory. It needs neither chemists nor bacteriologists to tell us that the water from a creek with an overhanging privy a short distance above will some day carry infection; or that the water from a shallow well in limestone or coarse gravel very near a leaking cesspool must be a source of danger. A sanitary survey is able to discover the sources of contamination, the kinds of pollution, and the degree, often with greater precision than combined chemical or bacteriological tests. No sanitary analysis of a water can therefore be considered complete unless it includes an examination of the watershed and a study of the geology and topography of the catchment area.

From a sanitary standpoint the principal determinations to be considered in a chemical analysis are those dealing with organic matter, free ammonia, nitrites, nitrates, chlorids, and in some cases hardness and alkalinity. These are the only tests ordinarily made in routine chemical analyses.

The various tests for organic matter are difficult of interpretation, unless they register marked differences from values known to be normal for the source of the sample, or unless they are used in conjunction with other tests. Perhaps more can be learned about the kind and condition of organic matter by use of the odor test than in any other way, but this requires a well-trained sense of smell. Odors of vegetable decay, such as "peaty" and "vegetable," indicate organic matter from dead vegetation or the presence of a few microscopic organisms. Grassy, aromatic and fishy odors point to heavier growths of more objectionable plankton. Musty, moldy and disagreeable odors like those accompanying putrefactive processes indicate the possible presence of sewage or other organic wastes which may carry contamination. Tarry, oily and chemical odors serve to detect certain trades waste. Organic matter registered by the albuminoid ammonia should be practically absent in good ground waters and also in surface waters which have very little vegetable

matter, as shown by the color test. High albuminoid ammonia is due to vegetable coloring matter, algæ and similar organisms, or to nitrogenous waste, usually sewage. Increased colloidal turbidity will accompany the latter if more than traces are present.

All the nitrogen tests, if used together, give testimony as to sanitary conditions. High free ammonia figures are suspicious in any sample of water, except in the case of deep wells where ammonia may be derived from mineral sources. Ordinary ground waters contain only traces, if any. Surface waters not subject to contamination will have little ammonia except in the presence of algal growths which are in a state of disintegration. Ammonia registers proximity to a source of pollution. Nitrites, too, furnish a danger signal, for the reason that they show incomplete oxidation of nitrogenous waste. Tests for organic matter under such conditions may show only small amounts remaining. Nitrites in the presence of nitrates are also an indication of incomplete purification. Nitrates (in the absence of nitrites) indicate complete mineralization of organic matter and therefore are evidence of distant or past pollution, but are not alone proof of a reliable protective barrier. Chlorids above the normal for the region come mainly from urine. The source may be near or distant, for the chlorids are stable.

Hardness and alkalinity are usually derived from mineral sources, but in soft water regions sewage adds appreciable quantities of lime, magnesium and sodium salts which affect these determinations. Hardness and alkalinity may, therefore, furnish contributing evidence of sewage pollution although, like chlorids, they afford no measure of the proximity of pollution.

Ground waters should contain fewer bacteria than surface waters, and should be free from organisms of the *Bacterium coli* group in 50 c.c. Artesian waters should be practically sterile. Surface waters to be acceptable should not contain more than a few hundred bacteria per c.c. developing at 20° C. and less than 100 per c.c. developing at 37° C. Colon bacilli should not be present in 1 c.c. of sample, and usually not in 10 c.c.

The interpretation of the chemical constituents of water is helped very much by an understanding of the nitrogen cycle which is described in detail on page 911.

Waters that vary in composition from time to time without evident cause must be regarded as unsafe. This applies particularly to ground waters. Surface waters vary greatly as the result of freshets, etc., but a ground water should show no sudden variations.

These general statements may be quite misleading when interpreting the analysis of a specific case. Therefore several selected analyses and interpretations are given at the close of this chapter.

Bacteriological Limits of Pollution.—In dealing with the bacterial content of drinking waters it has been possible to define limits of pollution more closely than in dealing with the chemical constituents.

In 1914, after an intensive study by experts, the U. S. Treasury Depart-

ment standard was adopted by the United States Public Health Service.³ The standard was made operative for the quality of drinking water, including bottled waters, supplied by common carriers in interstate commerce. It was not intended to define standards of purity but rather to establish maximum limits of bacterial pollution allowable under the prescribed conditions. Neither was it expected that general use of the standard would be made as a measuring stick for the quality of public water supplies in general. However, its adoption officially and otherwise became fairly general. One instance was that of the Water Analysis Laboratories of the American Expeditionary Forces in France.

The requirements were that the total number of bacteria should not exceed 100 per c.c. when grown on standard agar plates and counted after

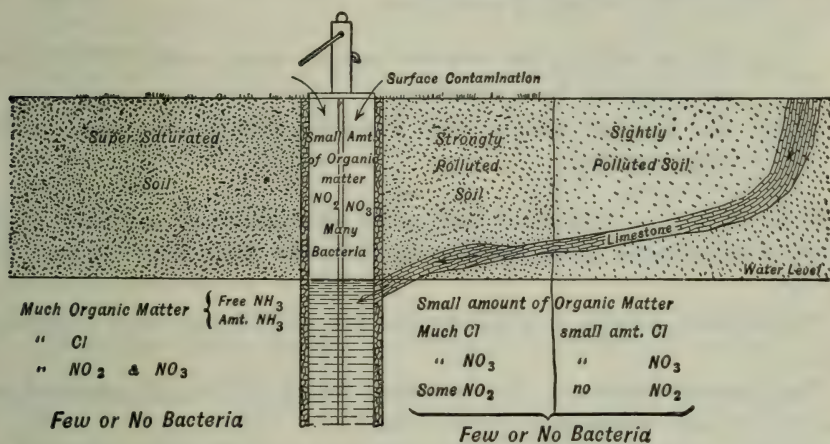


FIG. 89.—DIAGRAM ILLUSTRATING THE CHARACTER OF THE GROUND WATER IN RELATION TO SOIL POLLUTION, TO ASSIST IN THE INTERPRETATION OF A SANITARY ANALYSIS.

(See also Nitrogen Cycle, page 911.)

twenty-four hours' incubation at 37° C. Further, that not more than one out of five 10 c.c. portions of any sample examined should show the presence of organisms of the *Bacterium coli* group.

Rigorous as the demands of this standard seemed to some at the time it was promulgated, experience has shown the possibility of bringing many water supplies to a quality which surpasses these requirements. The tendency to adopt it widely was due to the fact that it was the only standard which had received government sanction. After ten years of trial the Public Health Service decided that revision of the existing limits would better suit the purposes of the standard. A committee, broad in its personnel, considered the matter and made the recommendations given below which appeared in the Public Health Reports, April 10, 1925, Vol. 40, No. 15, under the title

³ U. S. Pub. Health Rep., 1914, 29: 2959.

"Report of the Advisory Committee on Official Water Standards." The recommendations were adopted by the Treasury Department June 20, 1925, and promulgated as standards for drinking and culinary water supplied by interstate carriers.

The committee report dropped the provision relating to the total number of bacteria and based the standard entirely upon the occurrence of organisms of the coli group as follows:

1. Of all the standard (10 c.c.) *portions* examined in accordance with the procedure specified, not more than 10 per cent shall show the presence of organisms of the *Bact. coli* group.

2. Occasionally three or more of the 5 equal (10 c.c.) *portions* constituting a single standard *sample* may show the presence of *Bact. coli*. This shall not be allowable if it occurs in more than

- (a) five per cent of the standard samples when twenty (20) or more samples have been examined.
- (b) one standard sample when less than twenty (20) samples have been examined.

Note: It is to be understood that in the examination of any water supply the series of samples must conform to *both the above requirements*, (1) and (2).

The *standard portion* of water for this test shall be ten cubic centimeters (10 c.c.).

The *standard sample* for this test shall consist of five (5) standard *portions* of ten cubic centimeters (10 c.c.) each.

For a better understanding, a number of sanitary analyses of water are given with an interpretation. The student is advised first to study the analyses, draw his conclusions, and then compare them with the interpretation given.

EXAMPLES OF ANALYSES AND INTERPRETATIONS

ANALYSIS No. 1—*Gross Pollution*

Free ammonia	0.214	part per million
Albuminoid ammonia	0.810	part per million
Nitrogen as nitrites	0.005	part per million
Nitrogen as nitrates	21.0	parts per million
Chlorin as chlorids.....	47.0	parts per million
Total residue	412.0	parts per million
Volatile residue	279.0	parts per million
<hr/>		
Fixed residue	133.0	parts per million
Bacteria per c.c. upon gelatin at 20° C.....	65,000	
Bacteria per c.c. upon agar at 37° C.....	120,000	

Many liquefying colonies. Many chromogens per c.c. Fermentation in lactose bouillon in 0.001 c.c. *Bact. coli* present in 0.01 c.c.

This represents a grossly polluted water and should unhesitatingly be condemned, no matter what its source.

The following analysis of the Hamburg public supply from the Elbe River during the cholera epidemic of 1892 is given in *Chemical News*, 46: 144:

ANALYSIS No. 2—*Elbe River during Cholera Epidemic*

Appearance	Turbid and very yellow
Taste	Slightly unpleasant
Odor	Extremely little
Deposit	Small and dirty-looking
Chlorin as chlorids.....	472.0 parts per million
Free ammonia	1.065 parts per million
Albuminoid ammonia	0.293 part per million
Nitrates	26.43 parts per million
Oxygen consumed (15 minutes).....	0.928 part per million
Oxygen consumed (4 hours).....	3.428 parts per million
Total solids	1,160.7 parts per million

This is given simply as an instance of a grossly polluted river (Elbe) water, known to be infected.

ANALYSIS No. 3—*Boston Tap, Typical* (not averaged results)

Free ammonia	0.010 part per million
Albuminoid ammonia	0.114 part per million
Nitrogen as nitrites.....	0.000 part per million
Nitrogen as nitrates.....	0.02 part per million
Chlorin as chlorids.....	2.7 parts per million
Total residue	27.0 parts per million
Volatile residue	10.0 parts per million
Fixed residue	17.0 parts per million
Hardness	13.0 parts per million
Bacteria per c.c. upon gelatin at 20° C.....	77
<i>Bact. coli</i>	None

This is a surface water, collected in impounded reservoirs and stored about thirty days before it reaches the consumer. The watershed is fairly well protected. The chemical analysis shows little organic pollution; the ammonias are moderate in amount, nitrites absent; nitrates low; chlorids normal; bacteria indicate nothing suspicious. The water is of good sanitary quality, judged by chemical and bacterial analysis.

ANALYSIS No. 4—*A Suspicious Well Water*

Free ammonia	0.018 part per million
Albuminoid ammonia	0.020 part per million

Nitrogen as nitrites.....	0.007	part	per million
Nitrogen as nitrates.....	1.5	parts	per million
Chlorin as chlorids.....	19.3	parts	per million
Total residue	106.0	parts	per million
Volatile residue	37.0	parts	per million
<hr/>			
Fixed residue	69.0	parts	per million
Hardness	33.8	parts	per million

(The residue did not char and gave no odor upon ignition.)

Bacteria per c.c. upon gelatin at 20° C.....	60
Bacteria per c.c. upon agar at 37° C.....	45

No liquefying colonies. One chromogen per c.c. No fermentation in lactose bouillon in 10 c.c. No *Bact. coli*.

This water came from a driven well at Wenham, Massachusetts. Upon inspection it was found that the well was 400 feet from a stable, 200 feet from a cesspool, and 250 feet from the house.

The first thing that strikes our attention in this analysis is the high chlorids. This, however, lacks sanitary significance, as it is normal for the ground waters of this neighborhood. The hardness of the water is due to the mineral deposits in the surrounding soil through which the water percolates. The carbonic acid taken up by the water from the decomposing organic matter dissolves the lime in the soil. The organic matter as represented by the ammonias is low. The nitrates are high and indicate that the water has dissolved this end product of the oxidation of organic matter in its passage through the soil and perhaps in seepage from the cesspool. The noticeable quantity of nitrites indicates that all the organic matter has not been consumed and that the mineralization is not complete. The small number of bacteria present shows that the filtering action of this soil through which the water passes is effective in keeping out sewage contamination either from the surface or from the cesspool. This conclusion is strengthened by the absence of fermenting organisms and especially the absence of *Bact. coli*. The absence of liquefying bacteria and the presence of an occasional chromogenic organism indicate that there is little or no pollution from the surface and, in fact, upon inspection the platform covering the well was found to be tight and well constructed.

This particular sample of well water shows nothing injurious to health, and if subsequent analyses are equally satisfactory the water may be used without fear for drinking purposes. It is plain, however, that this well needs watching, for it is evident that the soil is already surcharged with organic matter, some of which appears in the water, and a further loading of the soil or a break in the cesspool might readily contaminate the well.

ANALYSIS No. 5—*Surface Pollution of a Well*

Free ammonia	0.022	part	per million
Albuminoid ammonia	0.035	part	per million
Nitrogen as nitrites.....	0.007	part	per million
Nitrogen as nitrates.....	1.0	part	per million
Chlorin as chlorids.....	19.0	parts	per million
Total residue	356.0	parts	per million
Volatile residue	151.0	parts	per million
Fixed residue	205.0	parts	per million

(Residue charred upon ignition with disagreeable odor.)

Bacteria per c.c. upon gelatin at 20° C.....	9
Bacteria per c.c. upon agar at 37° C.....	275

Many liquefying colonies. Several chromogens per c.c. Fermentation in lactose bouillon in 0.1 c.c. *Bact. coli* present in 1 c.c.

This is a shallow well in Washington, D. C., 28 feet deep, the water standing 4 feet in the well. There is a sewer 60 feet from the well and a privy within a block. The pump is old and of wood and the cover to the well is rotten at the base.

Although this water contains only a moderate amount of nitrogenous organic matter, as indicated by the ammonias, every other factor indicates serious contamination. The nitrates and nitrites are high; the chlorids are excessive. It is important to notice that this water has only 9 bacteria per c.c. when judged by the colonies that grow upon gelatin at 20° C. Nevertheless, it contains 275 bacteria per c.c. growing at 37° C., colon bacilli in 1 c.c., other fermenting organisms, and liquefying and chromogenic colonies. It is probable that most of the contamination in this case came from the surface, as the well had a very poor and leaky platform. This water should not be used for domestic purposes, and if it does not materially improve after the correction of the platform it should be condemned.

ANALYSIS No. 6—*Well Water, Surface Pollution*

Free ammonia	0.007	part	per million
Albuminoid ammonia	0.018	part	per million
Nitrogen as nitrites.....	0.0005	part	per million
Nitrogen as nitrates.....	2.5	parts	per million
Chlorin as chlorids.....	14.0	parts	per million
Total residue	62.0	parts	per million
Volatile residue	32.0	parts	per million
Fixed residue	30.0	parts	per million

(Residue charred upon ignition and gave disagreeable odor.)

Bacteria per c.c. upon gelatin at 20° C.....	820
Bacteria per c.c. upon agar at 37° C.....	640

Many liquefying colonies. Many chromogens per c.c. Fermentation in lactose bouillon in 1 c.c. *Bact. coli* in 10 c.c.

This water came from a shallow well in Washington, D. C., 18 feet deep, the water standing 3 feet from the bottom. The high nitrates and chlorids in this case represent past pollution. The small amount of organic matter with a trace of nitrites plus the number and character of the bacteria indicate surface pollution. This view is strengthened by the fact that repeated examinations of the water from this well showed marked variations in the number of bacteria. Upon inspection the pump and covering to the well were found in very bad condition and leaky; surface drainage was toward the well.

ANALYSIS No. 7—Illustrating Remote Pollution

Free ammonia	0.006	part	per million
Albuminoid ammonia	0.011	part	per million
Nitrogen as nitrites.....		trace	
Nitrogen as nitrates.....	20.0	parts	per million
Chlorin as chlorids.....	89.0	parts	per million
Total residue	430.0	parts	per million
Volatile residue	113.0	parts	per million
Fixed residue	317.0	parts	per million

(No charring upon ignition.)

Bacteria per c.c. upon gelatin at 20° C.....	92
Bacteria per c.c. upon agar at 37° C.....	16

No liquefying colonies. No chromogens per c.c. No fermentation in lactose bouillon in 10 c.c. *Bact. coli* absent.

This is a ground water from a shallow well in Washington, D. C. The well is 29 feet deep and the water stands 4 feet from the bottom. Top is well protected, waste water drains to sewer nearby. There are two privy vaults within two blocks of the well.

The analysis shows high chlorids and nitrates; otherwise nothing suspicious. This means remote pollution. The organic matter has been completely mineralized and the bacteria held back by the soil. The high result for volatile matter was due to volatilization of mineral constituents.

ANALYSIS No. 8—High Chlorids

Free ammonia	0.016	part	per million
Albuminoid ammonia	0.015	part	per million
Nitrogen as nitrites.....	0.000	part	per million
Nitrogen as nitrates.....	0.14	part	per million
Chlorin as chlorids.....	11.20	parts	per million

Bacteria per c.c. upon gelatin at 20° C.....	48
Bacteria per c.c. upon agar at 37° C.....	12

No liquefying colonies. No chromogens per c.c. No fermentation in lactose bouillon in 10 c.c. *Bact. coli* absent.

This water is from a driven well at Beverley, Mass. The analysis shows nothing suspicious, excepting the high chlorin, which is not excessive for this neighborhood, near salt water, and therefore lacks sanitary significance.

ANALYSIS No. 9—*High Free Ammonia; Deep Well*

Free ammonia	0.170	part	per million
Albuminoid ammonia	0.000	part	per million
Nitrogen as nitrites.....		trace	
Nitrogen as nitrates.....	0.0	part	per million
Chlorin as chlorids.....	3.1	parts	per million
Total residue	115.0	parts	per million
Volatile residue	45.0	parts	per million
Fixed residue	70.0	parts	per million

No bacteria per c.c. upon gelatin at 20° C.

No bacteria per c.c. upon agar at 37° C.

No fermentation in lactose bouillon.

This water is from a driven well in Washington, D. C., 96 feet deep; water stands 81 feet from the bottom. Good platform and drain, and pump in first-class condition.

It is exceptionally pure, both chemically and bacteriologically, excepting the large amount of free ammonia. This supposedly comes from the reduction of nitrates.

It is not uncommon to find water from deep wells to be high in free ammonia, and it is assumed that this comes from a chemical reduction under high pressure, and perhaps temperature, of the nitrogenous mineral matter in coal and alluvial deposits.

ANALYSIS No. 10—*Rain Water Stored and Polluted*

Free ammonia	1.050	parts	per million
Albuminoid ammonia	0.175	part	per million
Chlorin as chlorids.....	2.0	parts	per million
Nitrogen as nitrites.....		distinct	trace
Nitrogen as nitrates.....	0.0	part	per million
Oxygen consumed	2.25	parts	per million
Total residue	20.0	parts	per million

Bacteria per c.c. upon gelatin at 20° C..... 625

No fermenting organisms. No *Bact. coli*.

This is rain water from a dirty cistern. In appearance the water was clear and good. The analysis shows that the water is dirty and polluted with organic matter. The bacteriological results indicate absence of fecal pollution. The water is undesirable, but not dangerous, as far as the possibility of infection is concerned.

ANALYSIS No. 11—*Artesian Well Water, Showing the Effects of Storage*
(The figures are in parts per million.)

	Water Directly from the Well	Same Water from the Storage Cistern
Free ammonia	0.052	0.062
Albuminoid ammonia	0.003	0.016
Nitrogen as nitrites	0.000	0.0007
Nitrogen as nitrates	0.01	0.01
Chlorin as chlorids	10.4	10.2
Dissolved oxygen	10.65	10.69
Oxygen consumed	0.10	0.15
Total residue	111.0	97.0
Volatile residue	40.0	30.0
Fixed residue (mineral matter)	71.0	67.0
Bacteria per c.c. upon gelatin at 20° C.	6.0	6500.0
Fermentation in lactose bouillon	none	in 0.1 c. c.
<i>Bact. coli</i>	absent	absent

This water is from eight artesian wells at the Government Hospital for the Insane at Anacostia, D. C., 375 feet deep. The water is forced out by compressed air and flows by gravity to the storage cistern, which is of brick and cement, and has a capacity of 80,000 gallons.

It will be observed that this water is fairly low in total solids and is almost free of organic matter as represented by the ammonias, nitrites, nitrates, and oxygen consumed. The water is clear as it flows from the ground, but soon turns slightly yellowish on account of a small amount of iron in the ferrous state that is oxidized to the ferric salt, which is insoluble and is precipitated upon contact with the air. The amount of chlorids is somewhat large, but has no sanitary significance in this case. The principal point in this analysis is the bacteriology, which shows the water to be practically sterile as it flows from the ground, but which contains over 6,000 bacteria per c.c. in the storage cistern. These come from the air and contaminating surfaces, and illustrate the great growth of the common water bacteria in water stored under these circumstances. The slight increase of the ammonias and nitrites in the cistern water, as compared with the water direct from the well, indicates organic pollution and bacterial activity. The diminution in the residue results largely from separation of the iron. This water is pure and wholesome, despite the fact that it contains many more bacteria than that usually allowed. It has been used for some years by about 3,000 persons, who have been singularly free from typhoid fever and other water-borne diseases.

ANALYSIS No. 12—*Chemical and Bacteriological Changes in Potomac River Water as the Result of Storage and Filtration*

(The figures are the averages of fourteen representative analyses.)

	<i>Dalecarlia</i> Inlet Raw Water Entering Storage Reservoir	<i>Dalecarlia</i> Outlet Raw Water after About 3 Days' Storage	<i>Georgetown</i> Reservoir Second Storage Reservoir (Water Remains Here About a Day)	<i>Washington</i> Reservoir 3rd Storage Basin Water Applied to Filter	Filtered Water from Filtered Water Reservoir
Free ammonia	0.024	0.027	0.022	0.017	0.015
Albuminoid ammonia ..	0.161	0.131	0.117	0.096	0.054
Nitrogen as nitrites ..	0.0031	0.0051	0.0065	0.0056	0.0003
Nitrogen as nitrates ..	0.61	0.57	0.6	0.61	0.67
Chlorin as chlorids ...	2.6	2.61	2.61	2.47	2.53
Total residue	203.0	163.0	160.0	141.0	127.0
Volatile residue	47.1	48.0	49.0	41.0	39.0
Fixed residue	156.0	115.0	111.0	100.0	88.0
Bacteria per c.c. upon gelatin at 20° C...	526	381	306	235	36
Per cent samples with <i>Bact. coli</i> in 1 c.c...	42	40	33	16	4.7
Per cent samples with <i>Bact. coli</i> in 10 c.c...	28	40	40	41	9.5
Total per cent show- ing <i>Bact. coli</i>	71	80	73	52	14.2

Analysis No. 12 is a good illustration of the bacteriological and chemical character of a river water, and illustrates the changes that occur during short storage (three to five days) and after filtration through a slow sand filter.

It will be seen from this table that there is a gradual diminution in the amount of free ammonia and a more marked diminution in the amount of albuminoid ammonia. The amount of organic matter as represented by the ammonia is diminished just one-third. The nitrites show an increase during storage of the water, indicating active oxidation, but a marked decrease after it is filtered, showing the rapid completion of the oxidation of the organic matter in the filter. The nitrates show a tendency to increase in amount, which would be expected as the nitrites diminish. It is evident that storage and filtration have little effect upon the chlorin content of the water. The total residue diminishes as the result of storage, sedimentation, and filtration. It will be noted, however, that this diminution is more marked with the fixed residue than with the volatile residue. This is due to elimination of turbidity present in the form of clay.

The number of bacteria decreases as the result of storage, but the most marked decrease occurs as the result of filtration. Likewise, the effect of the few days' storage does not very materially affect the number of *Bact. coli*, but there is a marked diminution in their number following filtration.

The analyses of surface waters, shown in Analysis No. 13, on page 1005, with diagram (Fig. 90) of the locations from which samples were obtained, will repay careful study. This table and diagram were furnished through the kindness of Professor Whipple.

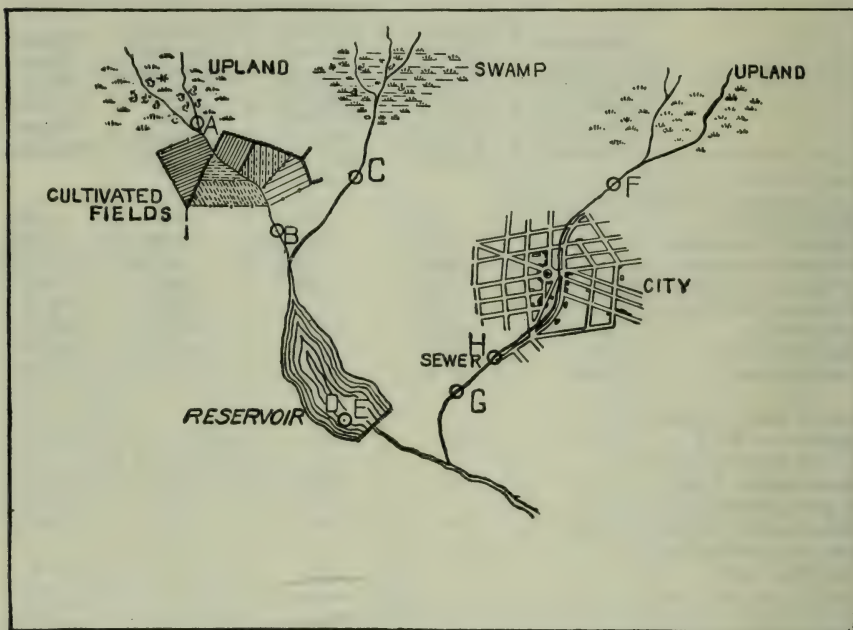


FIG. 90.—DIAGRAM SHOWING LOCATION OF SAMPLES.
(Analysis No. 13, page 1005.)

CHAPTER IV

THE PURIFICATION OF WATER

The ways in which water may be purified for practical purposes are not many. It is worth noting that most of the advances in water purification come from the development of old empiric processes. It is only at long intervals that a new method or principle of treatment is discovered that is important enough to find a permanent place in the art of water purification.

The principal methods at present serviceable for the purification of water upon a large scale are: (1) storage, (2) filtration, (3) chemical treatment (with sulphate of aluminum or iron, chlorin, hypochlorite of lime, or ozone), and (4) application of ultraviolet rays.

No method of purifying water can be considered to approach a satisfactory hygienic standard that does not first of all practically eliminate water-borne diseases. The process must also reduce the turbidity and color to inappreciable amounts and remove something like 99 per cent of the bacteria when these organisms result from sewage pollution and are fairly numerous. There is perhaps no final reason for the bacterial standard. It has been adopted by consent because it represents a purification that is satisfactory and that can be accomplished at reasonable cost. With the further awakening of the sanitary conscience of the community the standards will inevitably tend higher, and it is probable that in time our standards will approach an ideal that is now not regarded as necessary. At present there is no evidence that the few microörganisms left in the water after a satisfactory method of purification, such as slow sand filtration, are injurious. Certainly, if injurious influence is exercised, it is too small to be determined or measured by any methods now at our disposal.

NATURE'S METHODS OF PURIFYING WATER

In nature, water is purified by various methods, the chief of which are: (a) evaporation and condensation, which makes rain water the purest of natural waters; (b) the self-purification of running streams, which is a variable and uncertain quantity; (c) storage in lakes and ponds, which clarifies water and in time eliminates danger; and (d) the physical, chemical, and biologic action of the soil upon water that filters through the soil into the earth, one of nature's greatest purifying agencies.

Evaporation and Condensation.—The purifying action of the distilling and condensing process through which all meteoric water passes is one of

nature's beneficent processes. Enormous quantities of sea water, marsh water, and polluted water of all kinds are thus returned to us suitable for domestic use. Somerville estimates that "186,240 cubic miles of water are annually raised from the surface of the globe in the form of vapor chiefly in the inter-tropical seas." Water is thus constantly being purified in nature. The ocean has been compared to a boiler, the sun to a furnace, and the atmosphere to a vast still. The cooler air of the higher atmosphere and colder zones acts as a condenser, causing the precipitation of the distilled water as rain. About three-fourths of the earth's surface (145,000,000 square miles) is covered with water, much of which is in the tropical belt.

Self-Purification of Streams.—Streams become purer during the course of their flow. Of this there can be no doubt. This half-truth based upon chemical data has in the past suffered sanitarians to permit the use of water that now we know was responsible for much sickness and many deaths. Streams become purer, but not pure. Some impurities always remain, that is, the process is not complete and final. All surface supplies, except those from uninhabited catchment areas, are now regarded with suspicion and are stored, filtered, or otherwise purified before they are used by educated communities.

It was formerly said that a stream purifies itself in seven miles. Such a generalization is absurd. We now know that it is not the distance so much as the time and opportunity for the various factors of purification to become effective. Thus, Buffalo's sewage flows to Niagara's intake, a distance of about sixteen miles, in a few hours. There is little chance for self-purification to take place, and despite the high dilution the danger from the raw water is very great. Niagara's average typhoid rate for ten years, from 1899 to 1908, was 132.9 per 100,000, the highest in the country. Now that the water is filtered and chlorinated, water-borne typhoid is controlled.

A brisk flow brings the microorganisms of disease alive and virulent to the intake of the waterworks below; sluggish flow or stagnation corresponds to storage and results ultimately in the destruction of the bacteria which cause water-borne infections.

A good instance of self-purification of streams was found in the studies of the Potomac River and its relation to typhoid fever in the District of Columbia. The Potomac River drains an area of about 11,400 square miles which, in 1900, contained a population estimated to be about half a million, or about 44 per square mile. The velocity of the flow of the Potomac is extremely variable. It takes from four to seven days for the water to travel from Cumberland to Great Falls (where the Washington intake is located), a distance of about 176 miles. The waters of the Potomac are directly polluted by sewage at numerous points. The direct pollution is contributed by about 45,000 individuals, or 9.1 per cent of the total population on the watershed. Of this pollution about 80 per cent enters the river at points 176 or more miles from the intake at Great Falls, about 15 per cent enters it at points

between 50 and 170 miles above Great Falls, and 5 per cent is contributed by about 2,200 of the population and enters the river at points between 19 and 50 miles above the intake. There is practically no direct pollution of the Potomac within 19 miles of the intake. Here we have an instance of a stream draining an extensive and populous area and receiving industrial and human wastes from many thousand persons. Nevertheless, self-purification has occurred to such an extent that little, if any, of the typhoid fever occurring in Washington could be attributed to the use of this water. Slow sand filtration greatly improved the appearance, bacteriology and other qualities of Washington's water, but had no appreciable effect upon typhoid fever in that city.

The Mississippi River is perhaps one of the best examples of the self-purification of a stream, for, after draining almost the entire continental United States in a flow of over 3,000 miles, it is exceptionally free of intestinal bacteria at New Orleans, judged by the comparative absence of colon bacilli.

The principal factors concerned in the self-purification of water are varied and interesting. They are: (1) *chemical*, the oxidation of nitrogenous organic matter, resulting in its reduction or mineralization; (2) *biologic*, the death of microorganisms through antibiosis, time, and various means; and (3) *physiocal*, such as dilution, sedimentation, sunlight, etc.

Oxidation.—Organic matter is gradually oxidized, thus diminishing the amount of food for bacteria. The activity of the oxidation is favored by falls, rapids, and a turbulent flow. It is mainly the aërobic bacteria in running streams which have an active proteolytic action, and are thus able to digest and destroy organic matter. During the course of flow the complex nitrogenous substances are thus mineralized. Chemical analyses show a rapid decrease in the amount of organic matter and an increase of nitrates, and diminution of nitrites. It was these facts that led chemists to conclude that flowing rivers soon purified themselves.

Biological Factors.—Minute animals, such as infusoria, amebæ, water-worms, water-fleas, etc., which exist in countless numbers in certain waters, feed upon the organic matter and bacteria, and are a considerable factor in the self-purification of water.

Time and antibiosis play an important rôle in self-purification of streams, as they do elsewhere. Pathogenic bacteria die in water because it is an unfavorable environment. They succumb more quickly in a polluted water than in a pure water. It is probable that symbiosis and antibiosis also play a part. The saprophytic bacteria somehow help to kill off the dangerous varieties in the struggle for existence. Pettenkofer believed that the greater part of self-purification is due to the growth of algæ and other low forms of vegetation which clear the water of its impurities in the same way that the higher plants utilize the decomposing manure on cultivated fields. This view is endorsed by Bokorny, Emerisch, and Brünner and others who have studied the question. It is proved that these plants not only take up the simpler inorganic compounds, but all manner of organic substances, including volatile fatty acids, amino-acids, glucose, and urea.

On the other hand, it is an observed fact that such low forms of plant life do not flourish until purification is partially accomplished by the bacteria and protozoa, and that they grow luxuriantly in the absence of organic impurities if there is a supply of such end-products of organic decomposition as carbon dioxid and nitrates.

Dilution and Sedimentation.—Dilution is one of nature's real sanitary blessings. The abundance of water quickly dilutes the impurities under ordinary conditions so as to render them harmless. A small amount of infection in a great volume of river or lake water soon becomes so diluted as literally to become lost. It is true that one germ may cause disease just as a spark may start a forest fire, but the conditions must be fortuitous. It is fortunate for us that a single typhoid, cholera, or dysentery bacillus, especially when attenuated, may not, as a rule, induce disease. It is further clear that the chances of receiving a single bacillus in the few glasses of water one drinks are mathematically very small when the dilution is very great. Owing to these facts and to the further fact that pathogenic spore-free bacteria soon become attenuated and die in water, dilution becomes one of our sanitary safeguards.

Sedimentation is favored by a slow-moving stream containing insoluble inorganic particles such as clay. In muddy streams, such as the Mississippi and Potomac Rivers, the water is purified in very much the same way that the snow clears the air. The particles, constantly settling, wash the water by enmeshing the bacteria, which are thus carried to the bottom, where they are imprisoned and die. It is almost a filtration process. The water is swept or scoured many times by the innumerable fine particles in a turbid stream. The coagulation and sedimentation of colloidal organic matter likewise carry down bacteria and remove organic food from the water. This is the principle used to clarify and purify water with chemical coagulants such as sulphate of alumina.

Sunlight.—The germicidal influence of sunlight exerts its power upon all surface waters. The depth of penetration is slight, and varies with the turbidity and color of the water, the strength and direction of the sun's rays, and other factors.

Storage in Lakes and Ponds.—Nature makes use of the purifying power of time in storing water in lakes and ponds and other surface collections. Very few parasites pathogenic for man multiply in water under natural conditions. In time they all die out. Hence a stored water is reasonably safe. In addition, the organic matter undergoes decay and returns to its simple mineral constituents. Hence a stored water will in time free itself not only of harmful parasites, but also of most of its organic pollution. Storage is a natural and economic method of improving the quality of the water. The stagnation of stored water has been described on page 984.

The purifying power of the soil has been fully discussed in connection with the nitrogen cycle (page 911).

DISTILLED WATER

The distillation of water is the best method known for rendering it pure in a chemical sense. From a hygienic standpoint it is ideal; from a practical and economic standpoint it has several objections. It is used especially on naval and other vessels and in some industries.

In the distillation of water the first portion of vapor contains a disproportionate amount of volatile impurities, if such are present. If the distillation is continued to dryness or nearly so the concentrated solution of mineral and organic matters suffers reactions by which more volatile matter is formed and the distillate is again contaminated. For these reasons standard distilled water usually includes only what is technically termed the "middle run of the still," some of the first portion being rejected and the distillation stopped before all the water passes over.

Distilled water, even when obtained with precautions, is not always acceptable for drinking purposes. The taste is flat and suggestive of scorched organic matter. This is often ascribed to the want of aëration, but in many cases the sample is not improved by thorough aëration. Even when so improved, the additional operation adds expense, and unless purified air is used it adds organic matters living and dead. Leffmann believes that the disagreeable taste of distilled water is often due to volatile matter.

Statements are occasionally made that distilled water is too pure and hence not well adapted for drinking purposes, but these statements are not based upon physiological principles or clinical experience. Distilled water should be ideal, but has proven disappointing.

BOILED WATER

Boiling renders water safe so far as water-borne infections are concerned. It also destroys the labile toxins and probably renders harmless most poisonous substances of organic origin that may be in the water. Water containing lead and other stable chemical substances injurious to health would not, of course, be rendered safe by boiling.

For the traveler, the camper, and others who must use water of various sources, the character of which cannot be readily ascertained, the only safe procedure is to have his own tea kettle and little alcohol lamp. Enough water may be boiled in a few minutes in the morning or evening to last twenty-four hours or more for personal use. Chlorinated lime may also be used for this purpose. (See page 1362.)

Boiling drives off the dissolved gases, which gives to boiled water a flat taste. This may be corrected by shaking the water in a bottle or stirring with an egg-beater, or simply exposing it to the air over night, care being

taken not to recontaminate it. The disagreeable taste of boiled water is partly due to changes in the organic matter which take place at 100° C. As a matter of fact, it is not necessary actually to boil water to render it safe so far as typhoid, cholera, dysentery, and other non-spore-bearing infections are concerned. A temperature of 60° C. for twenty minutes or 70° C. or 80° C. for a few moments is sufficient. However, in the kitchen, where thermometers and scientific care are not expected, it is better to require the water actually to boil to insure safety, especially during epidemics or in waters known to be infected. Boiled water may be kept in covered pails or conveniently placed in well stoppered bottles, in which case it may be iced without the risk of contamination.

FILTERS

Slow Sand Filters.—Slow sand filters are simply beds through which the water percolates. They are also called English filter-beds, consist of large, shallow, tight reservoirs suitably underdrained and containing some four or five feet of stratified filtering material of progressive degrees of fineness, beginning at the bottom with broken stone or gravel and ending with an upper layer of fine sand. The water is passed through such a filter very slowly, from above downward. The cleansing of such a filter is done by removing the surface layer of dirty sand.

Slowly passing water in this way through sand purifies it biologically, physically, and chemically; nearly all of the objectionable bacteria as well as other microorganisms are removed and many of the particles in suspension are strained out and much of the organic matter is oxidized.

This process is called "slow" sand filtration to distinguish it from the rapid process known as mechanical filtration. The slow sand filters are spoken of as the English method, or as English filter-beds, because it was in England that they originated;¹ whereas the mechanical filters are spoken of as the American method, because this process was developed in this country to meet our special needs. The student should have a clear comprehension of the differences between these two methods.

The water in the slow sand filter passes very deliberately through a layer of sand; the filter chokes by the clogging of the superficial layer of sand, and the cleansing of this type of filter is done by removing this surface layer or *Schmutzdecke*, as it is called. Rapid sand filtration, on the other hand, consists in first adding a coagulant such as sulphate of alumina, then settling in basins, and finally passing the water rapidly through a layer of sand.

¹The first recorded attempt to filter water through sand was in 1829 when the one-acre slow sand filter was built by James Simpson for the East Chelsea Water Co., at London, England. In 1872 a plant was built at Poughkeepsie, N. Y., in accordance with plans prepared by James P. Kirkwood, which was the first practical attempt at purification of a municipal water supply in America.

Rapid sand filters are also called mechanical filters because the sand is sometimes cleansed by mechanical stirring and back-washing with water. Compressed air is also used, but generally water only.

The slow filtration of water through sand originated as an empiric process imitating nature's method of purifying water as it slowly passes through the soil. It was used before the chemistry or bacteriology of the process was understood. In fact, the intimate processes concerned in slow sand filtration are not yet part of our philosophy. We know that the spaces between the sand are enormous when compared with the size of bacteria; nevertheless, about 99 per cent of the bacteria are held in the superficial layers of the sand. Nitrification and oxidation of organic matter also take place. The process is not entirely a simple straining. It is aided by "vital" reactions in which bacterial activity plays a very large part. The bacteria and other micro-organisms in the upper layers of the sand grow and form a zoögleal mass; each grain of sand becomes coated with a gelatinous and adhesive film. A continuous sticky layer forms upon the surface a carpet-like mass constituting the *Schmutzdecke*, which effectively holds back the bacteria in the water.



FIG. 91.—THE ARRANGEMENT OF A SLOW SAND FILTER.

The removal of the bacteria then is largely due to the action of bacteria which become established in the filter. A visible *Schmutzdecke* is not, however, essential for successful sand filtration. In Hamburg, Lawrence, and other cities a greenish or brownish, slimy *Schmutzdecke* is formed upon the surface of the sand, and gradually becomes so thick and dense as to offer much resistance to the passage of the water itself. The *Schmutzdecke* is then removed. This can readily be done by scraping or shoveling, and then washing the surface sand in special apparatus. Where a visible *Schmutzdecke* is not formed, as in Washington sand filters, it is probable that the microörganisms which form a zoögleal mass do not find favorable conditions for growth. Nevertheless, in this case the surface layer of the sand becomes clogged in the usual manner and the underlying sand is quite clean. The bacteria that escape the surface action are caught upon and stick to the mucilaginous coating of the sand particles below, where they perish as in a trap. The experiments of the Massachusetts Board of Health at Lawrence show that filtration may be as effective from a bacteriological standpoint without the visible *Schmutzdecke* as with it.

Construction and Operation.—In view of the importance of the subject

the student should be familiar with the general principles and some of the details concerning the construction and method of operating a slow sand filter.

It is advisable to let the water settle, if it is turbid, before it is applied to the sand, for the reason that this prevents undue choking or clogging of the filters and thus effects a great economy. One of the main items in the cost of maintaining a slow sand filter is the scraping of the surface layer and the washing of the dirty sand. There are several preliminary methods of treating the water before it is applied to the filter. These methods differ with the character of the water, and consist in the main of screening, scrubbing, or coagulation. These processes are discussed more in detail upon another page.

A slow sand filter requires an extensive tract of land, for it should be recalled that only two and one-half to five million gallons of water should be filtered per acre per day. The filter should be conveniently located near the community it is to serve, and the high price of urban property is an important economic consideration. Thus, in Washington it requires 21 acres alone for the filter-beds to furnish 63,000,000 gallons of water daily at a 3-million-gallon rate per acre. The settling basins, storage basins for the filtered water, sand-washing apparatus, pumping station, laboratory, and other accessories require considerably more land.

The entire filtering surface is divided into units known as filter-beds. The size of each filter-bed has grown with the development of the art. In the filters recently constructed each bed occupies about one acre. Each bed must be an independent unit, so that the rate of filtration, the cleaning and all other operations may be carried on without disturbing the other beds. The pipes carrying the effluent from each filter-bed must be so arranged that the water may be wasted or utilized. Where the climate is cold, filters should be covered to prevent freezing.

In construction a filter-bed is built very much like an ordinary reinforced concrete reservoir. The bottom and sides must be water-tight, for failure in this regard may be annoying and dangerous for the reason that there may be considerable loss of filtered water or entrance of pollution from the outside if the pressure is reversed. The sides of the bed are usually vertical. Formerly it was thought best to slant them.

The sand may be obtained from a river bed or from sand banks; the grains should be sharp, hard silicates. If the sand contains clay this should be removed by washing before it is used. It is also important that the filtering sand should be free from lime, which has a tendency to make the water hard and to cause disintegration of the filtering material. The effective size of the sand best suited usually varies from 0.2 to 0.3 millimeter.

The sand used for filtration contains particles of various sizes; the water is forced around the larger particles and through the finer interstices which

occupy the intervening spaces, so that it is the finest portion which mainly determines the efficiency of the sand for filtration. According to Hazen, a provisional basis which best accounts for the known facts considers the size of grain such that 10 per cent by weight of the particles are smaller and

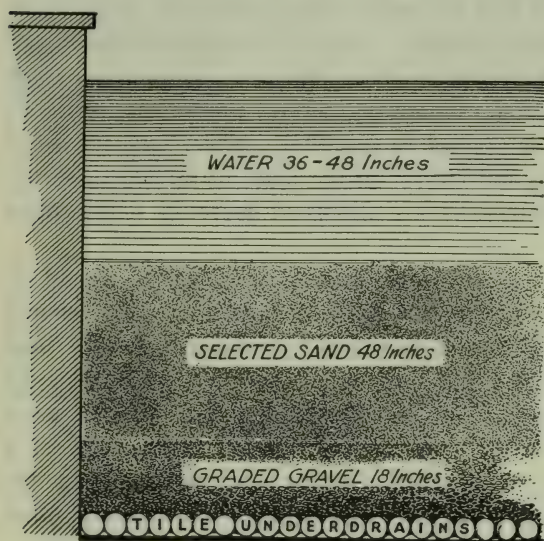


FIG. 92.—SECTION OF A SLOW SAND FILTER.

90 per cent larger than itself. This is considered the *effective size*, and is determined by sifting a weighed amount of the sand through a series of sieves. Another important point in regard to the sand is its degree of uniformity; that is, whether the particles are mainly of the same size or whether there is a great range in their diameters. This is shown by the *uniformity coefficient*, a term used to designate the ratio of the size of grain than which 60 per cent of the sample is

finer to the size than which 10 per cent is finer.

The usual thickness of the sand layer varies from 12 to 48 inches. The Imperial Board of Health of Germany has fixed 12 inches as the limit below which the sand should never be scraped. The higher limit is advisable wherever practicable. In this country the usual depth of the sand layer is 3 to 4 feet, and this is reduced by successive scrapings for the purpose of cleaning until it approaches 24 inches, when the sand is replaced. A thick sand layer has a steadying action upon the water on account of the increased friction, and thus aids in preventing irregularities in the rate of filtration and in the quality of the effluent.

The sand rests upon a stratified layer of rock and gravel laid in graded sizes which supports the sand so that it does not work its way down into the underdrains.

The size, position, and nature of the underdrains are a very essential part of the construction of a slow sand filter. The underdrains must be set so that the rate of filtration will be the same in all parts of the filter. If this part of the apparatus is not properly designed in a filter-bed having the broad expanse of an acre the water may pass through the sand in certain portions at the rate of ten or more million gallons while it may be found that at other portions there is practically no flow at all. This is a practical problem in hydrodynamics.

The depth of the water above the sand is usually 3 feet. In European filters the depth varies from 3 feet to 52 inches. It is comparatively easy through simple mechanical devices to regulate the flow of the applied water so that the depth of the water above the sand will remain uniform.

Probably the most important factor in the operation of a slow sand filter is the rate of filtration. In this country slow sand filters are usually run at a rate of 2,500,000 to 4,000,000 gallons per acre per day. Four million gallons is the maximum rate commonly allowed. During times of stress, however, or for other reasons, the rate is sometimes speeded up to five or six million gallons per acre daily. In Hamburg the filters are not allowed to run faster than 1,600,000 gallons, and in Berlin 2,500,000 gallons. Water passed through sand at the

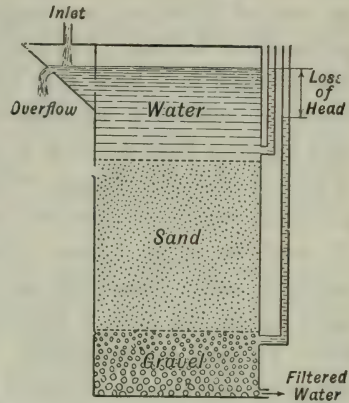


FIG. 93.—DIAGRAM ILLUSTRATING "LOSS OF HEAD."

rate of 4,800,000 per acre daily has a vertical movement of 3.94 inches in an hour. When the rate is 2,400,000 gallons the vertical motion is 1.97 inches per hour, and when the rate is slower the vertical movement is correspondingly diminished. It will thus be seen that this process is well named in that the water passes very slowly through the filter. This is of fundamental importance because the hour or more during which the water rests upon the surface of the sand and passes through the superficial layer is the critical time when the bacteria are enmeshed in the *Schmutzdecke* or adhere to the particles of sand, and when the other biological and chemical processes take place. If the raw water is reasonably clean or if hypochlorite or chlorin is added to the filtered water there may be no objection to speeding up the rate of the filter. The rate of filtration may be governed by automatic devices or may be controlled by hand by simply regulating the valve which governs the pipe carrying the effluent from each filter-bed. The friction of the sand layer varies from time to time, so that careful attention is required in order to maintain a steady flow and a constant rate, which is essential, for sudden variations in rate are fatal to the successful purification of water by the slow sand process.

The friction of the sand is measured by the *loss of head*. The loss of head is the difference between the level of the water above and below the sand layer measured in water gauges. This loss represents the friction or resistance of the sand layer. It greatly increases as the filter clogs. When a filter is new or perfectly clean the loss of head is usually 0.2 to 0.3 foot; when it exceeds 4 feet the rate of filtration cannot be maintained and the filters must be cleaned.

The length of time a filter may run before the loss of head becomes so great that it becomes unprofitable and requires cleaning varies from a few days to many months. The time depends upon the character of the water, the rate of filtration, and temperature, the density of the *Schmutzdecke* and many other factors. In cleaning a filter it is sufficient to scrape off only enough sand to a layer that appears clean. As a rule the sand immediately below the surface is not apparently soiled, and usually it is not necessary to take off more than an inch or so of the surface layer. This sand is removed to special cleaning devices, where it is thoroughly washed with filtered water and then stored in bins and replaced when the sand layer reaches a depth of about 24 to 30 inches. Practice varies in this matter, less sand being removed before resanding in the case of heavily contaminated waters. In some plants the sand is washed in place by machines which travel over the filter, no sand being ejected. The *Schmutzdecke* and the surface layers of the sand are usually removed by hand with broad shovels. There are also mechanical devices which accomplish the same purpose. After cleansing, the effluent from a filter-bed should be wasted until the bacteriological examination shows that the filter is again performing efficient work. This may require several days, the time varying with the temperature, the character of the water, the sand, and other conditions.

Efficiency and Control of Slow Sand Filters.—The efficiency of a slow sand filter is mainly measured by a comparison of the number of bacteria in the raw and filtered water. A good filter should eliminate 99 per cent of the bacteria, provided the applied water carries a load of a few thousand bacteria per c.c. In any event the filtered water should not contain over 100 bacteria per c.c. and colon bacilli should be absent in 10 c.c. It is to be noted that all the bacteria in the filtered water do not represent those that actually pass through the sand. Some of them grow in the underdrains and gravel layer and are, so far as known, harmless varieties.

In Germany the rate of filtration and other factors are minutely regulated and controlled by official ordinances. In many states in this country close observation, and sometimes control, of water purification plants is maintained by state departments of health, but in general a competent engineer, or operator, is allowed freedom of action if his methods do not imperil the health of consumers. New Jersey (August, 1918) and other states have passed laws requiring the examining and licensing of operators of water and sewage treatment plants under the direct control of the State Department of Health.

A slow sand filter plant cannot be effectively operated without skilled superintendence of an engineer expert in the art of water purification. It also requires a small laboratory with a competent analyst, who must make daily observations of the applied water and the effluent from each filter. The effluent from a filter not giving good results should be wasted. The water

from a new filter, or one just scraped, should not be used until the bacterial results show that it is accomplishing effective purification. The best results require skillful and scientific control.

There are many ways by which a better effluent may be secured, such as the use of lower rates of filtration, finer grained filtering materials, and more complete preliminary treatment, such as settling basins, storage, or chemical coagulation. The filtered water may be further purified with chlorin, hypochlorite of lime, ultraviolet rays, or ozone. It requires a surprisingly small amount of chlorin practically to sterilize a filtered water. Doses of 0.1 to 0.25 part per million are commonly used.

Because slow sand filtration has achieved such marked success with some waters and greatly reduced the amount of typhoid is no reason why it should be universally recommended under all circumstances. To recommend slow sand filtration in all cases would be as irrational as to recommend the use of antitoxin in every case of sore throat. A correct diagnosis is essential. Every water cannot be successfully or economically treated by this process alone. Thus, the very turbid waters of our South and West contain particles of clay so fine that some of them pass a sand filter. No amount of sand filtration will satisfactorily clarify such waters. The Potomac water in times of high turbidity may be passed through a sand filter three or four times without removing the residual turbidity due to these microscopic particles. To apply a very turbid water to a sand filter soon chokes it and adds unnecessarily to the difficulty and expense of the process. The particles may be so fine that they will not all settle even when the water is given long storage. There are several ways of solving this problem, which is of first magnitude for the purification of the surface water of a large part of our country. One of the best ways is to provide large storage reservoirs, so that the water may be settled or taken from the river only at favorable times, rejecting the flow during periods of high turbidity. Another is to use preliminary coagulation with aluminum sulphate and provide for sedimentation before applying the water to the sand. In some cases coagulation is supplemented by rapid filtration through mechanical filters, the water finally being passed through slow sand filters. This is done at Poughkeepsie, New York. Scrubbers are less frequently used. These are filters of gravel, or other coarse-grained material, through which the water is passed at a rapid rate. Partial removal of turbidity is accomplished in this way. No general rule can be set down. Waters differ radically, and the same stream varies from time to time. Each problem must be studied and solved in relation to its own special condition. Whether the filtered water should be further purified depends upon circumstances.

Results of Slow Sand Filtration.—The good results of purifying water by the slow sand method have been abundantly demonstrated in Altona, near Hamburg, in 1892, during the cholera epidemic; also in Hamburg

since 1893 and in Lawrence, Mass., also since 1893; further in Albany, Philadelphia, Pittsburgh, Berlin, Paris, and many English cities. It should be noted that at Albany the typhoid rate did not come down immediately after filtration. It sometimes requires one or two years to reach the residual or "normal" rate. In a few instances, such as Washington, D. C., and in Youngstown, Ohio, filtration of the water was not followed by a noticeable diminution in the typhoid rates, presumably due to the fact that little of the typhoid fever in these places was water-borne. Forty-seven cities in the United States had, in 1925, slow sand filters with a capacity of one million gallons per day or more. The population served was 5,054,000.

The best results in water filtration, as measured by the improvement in the health and reduction of the death rate among those who use the water, have been obtained with slow sand filters. Hazen believes that this is probably because the method is an old one, has been long and carefully studied, and has been applied on a large scale in well-perfected forms for many years, rather than to any natural superiority of the method.

The purification of water through slow sand filtration not only diminishes the amount of typhoid and other water-borne intestinal infections, but is believed also to reduce the general death rate. This assumption, known as the Mills-Reincke phenomenon, is discussed on page 1040.

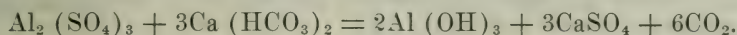
Mechanical Filters.—The essential and characteristic features of mechanical filtration are: (1) The addition of a chemical precipitant or coagulant to the water, followed by sedimentation. (2) Passing the water rapidly through a layer of sand. (3) Washing the sand when dirty by reversing the flow of water and in some cases using pneumatic or mechanical means of agitating the sand. The filters are either the gravity or the pressure type. The filtering sand is contained in a large wooden, iron, or concrete tank.² These filters are well named, not only because the filtering sand is sometimes washed mechanically, but because the action is more strictly a mechanical straining, whereas biological processes are important features in the purification of water passing through a slow sand filter.

Washing the Filters.—The filters are washed by a reversed flow of water. The sand may be agitated by compressed air; originally revolving rakes were used in small installations. When a "high velocity wash" is provided, as is usually the case, fifteen to twenty gallons of filtered water per square foot of filter per minute are forced in a reversed direction through the sand. This rate provides sufficient force to lift the sand and cause the grains to circulate and rub against one another, thus ridding them of coagulum, dirt and bacteria. Low velocity washing occasionally used in conjunction with

² Mechanical filters date from 1884, when the process was patented by J. W. Hyatt and Albert R. Leeds. They were first used municipally at Somerville, N. J., in 1885. The Hyatt patent expired in 1901, and since then numerous improvements in details have been made and patented, considerably improving the art of cleansing water by this process.

compressed air may save filtered water. The washing process requires from 2 to 5 per cent of the total water filtered.

Coagulants.—The *coagulants* used are sulphate of aluminum, commonly called “filter alum,” and occasionally sulphate of iron. The alkaline carbonates present in the water decompose the aluminum sulphate with the formation of aluminum hydroxid, which is thrown out of the solution as a flocculent, colloidal, jelly-like precipitate. The reaction is as follows:



Calcium bicarbonate or other alkaline substance is necessary to decompose and precipitate the alum, and if not normally present in the water some lime or soda must be added. The reactions and methods of estimating the quantities are given in detail on page 1033. The precipitated aluminum hydroxid clears the water very much as white of egg clears coffee. Suspended matter, including bacteria and inorganic particles, are enmeshed and deposited on the surface of the sand. Thus it will be seen that an artificial inorganic *Schmutzdecke* is quickly formed upon mechanical filters instead of the natural organic *Schmutzdecke* of the slow sand filter-bed. When this deposit becomes abundant enough to clog the filter the filter is washed.

It is advisable to provide coagulating basins to hold the water for a few hours after it has received the coagulant, in order to allow the chemical reaction resulting from the treatment to take place. Such basins also serve to remove by sedimentation much of the precipitate, and the filters therefore do not clog so readily, and cleansing is not required so frequently.

The rate at which water is passed through mechanical filters is very great when compared with slow sand filters. Rates varying from 100,000,000 to 175,000,000 gallons per acre per day are used.

One hundred and twenty-five million gallons per acre daily may be taken as a fair average of what is to be expected of them. On account of the rapid rate of filtration there is great economy of space. However, while the mechanical filters are cheaper when first cost is considered, the advantage is with slow sand filters as far as cost of maintenance and operation are concerned.

The proper amount of coagulant in solution is added to the water by means of a calibrated orifice, or in dry form by means of some type of “dry feed” apparatus. It requires, as a rule, from one to three grains of alum for each gallon of water to be treated, that is, from 143 to 429 pounds per million gallons. Larger doses are sometimes required.

The amount of alum added to the water must be varied from time to time in order to maintain effective coagulation and precipitation. Aluminum hydroxid is produced in colloidal suspension, and like all colloids its stability is affected by many things, that is, the iso-electric point, or point at which surface charges are neutralized and precipitation is optimum, will be in-

fluenced by physical and chemical conditions surrounding the colloidal particles. The amount of alum required for good coagulation will depend to some extent upon the amount of turbidity and color to be removed, but more particularly will it be governed by temperature, alkalinity, carbon dioxid content and the nature of organic colloids in the water. So it is that every natural water is a law unto itself in the quantity of coagulant needed to treat it. Less than a given minimum produces negligible results; more than the optimum is a waste of chemicals and may even reverse the efficiency of the process.

It is also true that many waters, especially river waters, change in character from day to day, and even from hour to hour, thereby necessitating frequent laboratory examination and close control of plant operation. Mechanical filters require intelligent and constant supervision in order to insure satisfactory results.

Mechanical filtration meets with special favor in this country because it affords a comparatively cheap method of supplying clean water from a very turbid or highly colored source. The process is particularly applicable to the muddy streams of our South and West and the highly colored waters of the Atlantic seaboard. In fact, it is the only known method of rendering some of these waters quite free of turbidity and color.

Mechanical filters, when properly designed and operated, will take out most (from 95 to 99 per cent) of the bacteria contained in the raw water. The bacterial purification is not as constant and uniformly high as that obtained by slow sand filtration. Mechanical filters have not had the same dramatic chance to prove their health value as slow sand filters. This is because slow sand filters were first in the field, and at a time when water-borne typhoid was excessive. Rapid sand filters came later and did not have a similar opportunity. There is reason, however, to suppose that if properly designed and well run, they could have made a good showing.

For the complete list of mechanical filters see *The Filtration Plant Census, Journal of the American Water Works Association*, August 25, 1925, Vol. 14, No. 2.

In 1925 there were 587 cities in the United States having rapid sand filter plants with a capacity of one million gallons per day or more. The population supplied was 18,600,000.

THE DIFFERENCES BETWEEN SLOW SAND AND MECHANICAL FILTRATION

<i>Slow Sand Filtration</i>	<i>Mechanical Filtration</i>
English system or English filter-beds— originated in England.	American system—developed in America to meet our special needs.
Has been long in use.	Comparatively recent (since 1884).
Preliminary treatment not an essential part of the process, though sometimes desirable.	A coagulant is first added to the water— sulphate of aluminum, or sulphate of iron.

<i>Slow Sand Filtration</i>	<i>Mechanical Filtration</i>
Water passes slowly through a layer of sand, in large, shallow, tight reservoirs.	Water passes rapidly through a layer of sand in small wooden, concrete or iron tanks.
Usual rates from 2,500,000 to 4,000,000 gallons per acre per day.	Usual rates 40 to 50 times as rapid—100,000,000 to 175,000,000 gallons per acre daily.
Cleaned by scraping surface layer of sand— <i>Schmutzdecke</i> .	Cleaned by reversed flow of water and mechanical agitation of the sand—hence the name “mechanical” filtration.
The process is both biological and a mechanical straining. Duplicates nature's process of purifying water.	The process is mainly a mechanical straining. An artificial imitation of nature's process.
Partial oxidation of nitrogen compounds.	Chemical indices of pollution not affected.
First cost is large; maintenance comparatively small.	First cost is comparatively small; maintenance large.
Especially serviceable for water having little turbidity and color.	Especially suitable for turbid waters, containing silt and clay, and for colored waters.
Removes about 30 per cent of the coloring matter.	Takes out nearly all of coloring matter.
Removes about 99 per cent of the bacteria; action is uniform.	When properly operated removes from 95 to 99 per cent of bacteria—less uniform.

In 1900, according to Hazen, 1,860,000 people, or 6.3 per cent of the urban population of the United States, were being supplied with filtered water. In 1904 the number of people so supplied had increased to 3,160,000, or 9.7 per cent of the urban population of the country. Since that time many large cities have installed filter plants, until (1925) about 23,000,000 people were being served with filtered water. Most of this filtered water is now also treated with chlorin or with bleaching powder.

Household Filters.—The domestic filter as ordinarily used in the household has limited sanitary value. The purification of water, even by so simple a method as straining, requires a degree of care, knowledge, and experience that is not found in the kitchen. If a water is contaminated, reliance should not be placed upon any household filter operated in the usual way. It is possible in the laboratory by the use of special precautions to pass water through a Pasteur-Chamberland or a Berkefeld filter so as to obtain a sterile filtrate. This requires skilled bacteriological manipulation of a kind that cannot be attained in ordinary service in the house. I have seen janitors “clean” a filter in such a way as to actually contaminate the water.

There are two main types of household filters: one made of unglazed porcelain (kaolin), known as the Pasteur-Chamberland, and the other made of diatomaceous earth, the Berkefeld, or Mandler. Sandstone, charcoal, asbestos, and a great variety of filtering substances are used. Even in the closest grained unglazed porcelain filter the pores of the filter are larger than the bacteria. The bacteria cannot get through the tortuous passages; they

adhere to the particles that make up the filtering substance. But if conditions are favorable, bacteria, such as typhoid, may soon work their way through its walls. The Berkefeld or Mandler filters of diatomaceous earth are more porous than the Pasteur-Chamberland filters.

When a water is not contaminated, but turbid, household filters are serviceable in rendering it clear. They are specially useful when the turbidity is due to clay or to iron, or other inorganic particles that may readily be removed by simple straining. They also remove many forms of algæ.

The sanitarian places no reliance upon the filtration of water in the household, and for drinking purposes such water, if suspected, should be boiled, even though filtered. The boiling should be the last process.

STORAGE

The storage of water is one of the simplest and best means of purifying it. The first cost may be large, but the cost of maintenance is comparatively trifling. Harmful bacteria soon die in a stored water, the solid particles settle out, the organic matter is largely oxidized, the color is gradually bleached, and other improvements take place. Storage takes advantage of many of nature's methods of purifying water, viz., time, sunlight, dilution, sedimentation, oxidation, and antibiosis.

A stored water may deteriorate in quality owing to the growth of algæ and the decomposition of organic matter. Algæ grow in stored water exposed to sunlight, particularly in warm weather. While these organisms are not harmful, they impart disagreeable tastes and odors to the water. (See page 966.) The decomposition of the organic matter in a stored water may also cause unpleasant tastes and odors, especially at the spring and fall overturn. (See page 944.) Waters stored in a closed reservoir keep without deterioration, and the advantage is therefore manifest. Filtered water should always be stored in covered reservoirs, not only to protect it from strong light, but also to prevent contamination from dust and other sources.

Sedimentation.—Sedimentation is one of the factors in purification by storage. It has great influence in this connection, as may be observed from the depth of mineral and organic deposits built up in reservoirs, ponds and lakes. Such deposits are, likewise, found in the beds of streams which carry a heavy load of suspended solids. The silting up of stream courses and the formation of sludge banks are manifestations of one of nature's attempts at purification. With these solids are found numbers of bacteria of all kinds. If slow flow or long time can exert their effects colloidal particles as well as coarse ones are thrown out by virtue of the agglutination of these into masses large enough to settle. This action enmeshes bacteria which are carried to the bottom along with sedimented material. The purging action of the particles as they fall is most effective in turbid waters.

Sedimentation on an artificial scale is used to improve the sanitary quality of water, but it has distinct limitations. It is the cheapest way to get rid

of turbidity due to relatively large particles that settle in a moderately short time. Sedimentation basins are, therefore, frequently used as a preliminary process in water purification. They are especially useful to protect filters by reducing the load of solid matter that tends to clog them. Sedimentation basins also help to equalize the physical, chemical and bacterial qualities of water from sources subject to great fluctuation. They may be large enough to take the water only at favorable times. The water as a rule does not remain in sedimentation basins long enough to enjoy the full effects of storage.

CHEMICAL METHODS OF PURIFYING WATER

The chemicals which are successful in practical use for the purification of drinking water are ozone, chlorin, calcium hypochlorite (chlorinated lime), potassium permanganate, aluminum sulphate, ferrous sulphate, and copper sulphate. The removal of hardness by means of lime, soda ash and the zeolites has been discussed in a previous chapter (page 973).

OZONE

Ozone, discovered by Schönbein in 1840, is one of the most satisfactory substances for purifying water from a sanitary standpoint. When conditions are favorable, its application as a germicide is the most effective of all the methods used except boiling. An ozonized water is practically sterile. The limitations of the ozone process are that it does not act effectively in the presence of turbidity or vegetable stain, and does not remove these constituents. From a practical standpoint the expense of producing ozone in sufficient concentration is disproportionately large.

It is not desirable to add ozone to a colored or turbid raw water. It is better first to clarify the water by some method before applying the ozone. The quantity of ozone required for effective bacterial action depends upon the amount of organic impurities contained in water. Much of the ozone is used up by the organic impurities, and this may happen so rapidly that it will not have a chance to act upon the bacteria.

An impure water containing much organic pollution, when treated with ozone, may give disappointing results from the fact that unpleasant tastes may be developed. These are doubtless due to the partial oxidation of the decomposing organic matter with the production of nitrogenous compounds not well understood.

For the purification of water, ozone is produced by electrical discharges in the atmosphere, and this ozonized air is then brought into intimate contact with the water. To produce the ozone requires a silent brush discharge and the air must be cold and free of moisture. If sparking occurs between terminals, oxids of nitrogen are formed which are corrosive and prevent the formation of ozone. The ozonizing apparatus therefore must be carefully designed, and its operation needs skilled supervision.

The molecule of ozone (O_3) readily gives up one atom of this gas in a nascent condition. It therefore has a very strong oxidizing action upon organic matter, and has a very powerful germicidal action. In this respect the action of ozone corresponds chemically to potassium permanganate, the hypochlorites, and chlorin, all of which are used in water purification.

It is necessary to get any excess of ozone out of the water in order to avoid the corrosion of pipes. This may be done by aëration, by means of

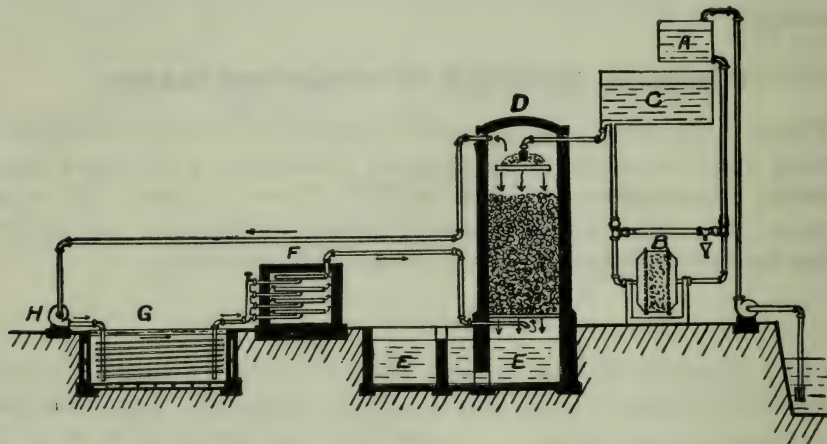


FIG. 94.—AN INSTALLATION FOR TREATING WATER WITH OZONE.

- A. Settling tank. B. Filter. C. Storage reservoir for filtered water. D. Tower, water enters above and ozone below. E. Pure water reservoir. F. Ozonizer. G. Dryer, to remove moisture from air before it passes into ozonizer. H. Fan.

fountains or cascades. On account of the insolubility of the ozone, it soon disappears. The fact that ozone is largely insoluble in water makes it necessary to bring it into intimate contact with all portions of the water to be treated. This is usually accomplished by allowing the water to trickle downward through tall cylinders filled with coke, lava, or other similar substances while the ozone is admitted to the bottom of the cylinder. The water flows downward, the ozonized air works its way upward, and in that way the desired contact is obtained between the ozone and every portion of the water.

A very small amount of ozone is effective for the purification of water. It only requires from 1 to 3 milligrams per liter. The modern machines produce concentrations as high as 10 grams and more of ozone per cubic meter of air. The ozone not taken up by the water may be used over and over again. This is accomplished in some of the ozonizing processes by conducting the air that leaves the upper part of the water cylinder back to the ozonizer.

In general, it may be said that, owing to the expense and the electrical and engineering difficulties involved, the ozonizing process is not at present applicable to the purification of water upon a small scale. It has been

applied with success upon a large scale in a number of places abroad, but not to any extent in this country. The first ozonizing apparatus for the purification of water on a large scale was installed by Siemens-Halske at Lille, France. Other ozonizing plants for purification of drinking water have been installed at Wiesbaden and Paderborn in Germany; Cosne, Chartres, Nice, Denard in France; Ginnekin in Holland; Sulina on the Black Sea, and Petrograd in Russia; Lindsay, Ontario; and Paris, in part, 24,000,000 gallons per day from the River Marne. In France there are 26 large municipal plants where ozone is used. At Lindsay the ozone treatment failed because the ozone and the water were not properly mingled. At Wiesbaden much trouble was caused by the oxidation of the iron. Experiments at Ogdensburg, New York, failed to remove the color of the water.

One objection to the treatment of water by ozone is that the electric apparatus is delicate and complicated and requires skilled attendance. The ozone processes are not yet standardized; at present it is difficult to determine what waters may best be treated with it. Where waterpower may be obtained for the generation of the electricity necessary to produce the ozone the cost is very much lessened. The principal systems at present used for ozonizing water are the Siemens-Halske, the Gerhard, Tindal, De Frise, Otto, Abraham Marmier, Vosmaer, Bridge, Stynis, and others.

CHLORINATION

Chlorinated Lime--Bleaching Powder or "Chlorid of Lime."—Chlorinated lime, popularly miscalled "chlorid of lime," and often spoken of as "bleach," has for years been used to disinfect sewage, outhouses, cellars, and for miscellaneous purposes. Its use in the disinfection of water as a practical process in the United States dates from 1908, when Mr. G. A. Johnson was called to solve the serious and difficult problem in water purification at the Chicago stock yards, the discharges from which entered Bubbly Creek. Filtration of the water of Bubbly Creek was not satisfactory, and all methods failed to abate the nuisance until Mr. Johnson treated the water with chlorinated lime. The method was given further widespread attention when the Jersey City Water Company essayed to comply with its contract to furnish pure water to Jersey City by simply adding a little bleaching powder. Other municipalities soon took it up in order to render their public water supply safe, until it came to be used throughout the country.

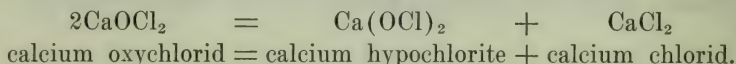
Properties.—Chlorinated lime is made by saturating slaked lime with chlorin at ordinary temperatures. It is a white or whitish powder occurring also in friable lumps; dry or slightly damp, with a feeble odor of chlorin and a disagreeable, bitter, and saline taste. It has an alkaline reaction, but finally bleaches litmus paper. The medicinal dose administered by the mouth is from one to five grains (0.06 —0.3 gram). As a mouth wash a 1 per cent solution may be used. The physiological action of chlorinated lime resembles that of chlorin with the superadded causticity derived from the

lime in its composition. Externally it is an active irritant and sometimes moderately caustic.

A 6 per cent solution in water may be made. However, all the constituents of bleaching powder are not soluble. Chlorinated lime contains a large amount of calcium hydroxid ($\text{Ca}(\text{OH})_2$) which is largely insoluble, hence the milky appearance of the solution, and also the precipitate known as "sludge," which settles rapidly. Calcium hypochlorite, the active principle, is readily soluble in water.

Upon exposure to the air the hypochlorites deteriorate rapidly to the more stable and inert carbonates. Care must therefore be taken to keep the substance in air-tight containers and to know the correct amount of available chlorin in each lot of the bleach at the time it is used.

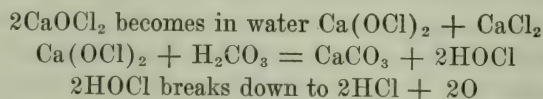
Composition.—The precise chemical constitution of chlorinated lime is not definitely known, although the matter has been frequently investigated. It seems quite certain that neither chlorid of lime, CaCl_2 , nor hypochlorite of lime, $\text{Ca}(\text{OCl})_2$, exists as such in dry bleaching powder, but is formed on dissolving it in water. Calcium oxychlorid, CaOCl_2 , is now generally accepted to be the essential agent of *dry* bleaching powder, and calcium hypochlorite, $\text{Ca}(\text{OCl})_2$, to be the active germicidal principle of the solution. Thus:



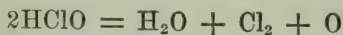
Calcium chlorid which is thus formed, or calcium carbonate, which forms when bleaching powder is exposed to carbon dioxid, are both inert so far as germicidal value is concerned.

Modes of Action.—When bleaching powder is added to water, the reactions taking place are complex. The germicidal action is due at least to three factors: (1) the nascent oxygen formed; (2) the free and liberated chlorin; and (3) chloramins which are formed from organic matter in the water. The hypochlorites are themselves directly toxic.

1. *Nascent oxygen* is a very powerful germicide; it is liberated from the hypochlorous acid.



2. *Chlorin* is a potent germicide. There is always some free chlorin in bleaching powder. Chlorin is also liberated to some extent when the hypochlorous acid decomposes, thus:



The intimate nature of the chemical processes is somewhat involved, but it is plain that hypochlorous acid and its salt, calcium hypochlorite, both liberate free chlorin, especially in acid solutions.

3. *Chloramins.* Hypochlorites in contact with ammonia form chloramin, NH_2Cl . Hypochlorites in contact with amino-acids also form chloramins, most of which are germicidal, some of which have extraordinary potency. Dakin and Cohen examined a number of chloramins, and found dichloramin-T to be particularly active.

Rideal added chlorin to water and found that when all the chlorin is consumed, there remains a strong germicidal action. It was also found that ammonia added to water with bleaching powder increases the activity of the hypochlorite. Dakin attributed the persistent action to the formation of chloramins, by reaction of the hypochlorites on protein or amino-acids. Dakin and Dunham studied the disinfection of water and found that adding citric, tartaric, acetic and similar organic acids increases the activity of chloramin-T. The compounds thus formed are unstable but benzoic acid produces a relatively stable compound with chloramin-T which is sold as *Halozone* (p-sulphon-dichloramin-benzoic-acid). It destroys typhoid bacilli in thirty minutes in dilutions of 1 part to 300,000 parts of water. For further discussion of chloramins, see page 1363.

It will thus be seen that chlorin itself, nascent oxygen and also chloramins take part in the disinfecting action of bleaching powder. The relative part played will vary with the amount of organic matter contained in the water. It is theoretically conceivable that the liberated chlorin will directly kill a typhoid bacillus by combining with the protein of the germ, and the chloramin thus formed may kill another typhoid bacillus. It is generally conceded that hypochlorites in water owe their action principally to the release of nascent oxygen and that the disinfecting action is one of oxidation.

Strength.—The amount of hypochlorites added is usually expressed in terms of "available chlorin," although in reality this represents the available oxygen liberated by the chlorin. Thus a good bleaching powder will average 35 per cent of available chlorin, which is the equivalent of about 7.9 per cent of available oxygen. By available chlorin is understood the chlorin readily liberated from its combination as determined by the usual thiosulphate titration.

How to Dissolve Chlorinated Lime.—Chlorinated lime is soluble in about twenty times its weight of water, leaving an insoluble residue consisting mostly of calcium hydroxid, $\text{Ca}(\text{OH})_2$. Half a pound of chlorinated lime may be dissolved in a gallon of water. Such a solution contains approximately 6 per cent by weight of chlorinated lime, representing about 2 per cent of available chlorin.

To obtain a clear solution of chlorinated lime it is important to remember that the available chlorin is readily soluble, even in fairly cold water, and the undissolved sludge of calcium hydroxid, silica, etc., settles readily. Settling, at least, takes place readily if a few simple rules are observed. (1) Do not mix too stiff a paste, otherwise a gelatinizing action takes place and greater difficulty in settling is encountered. (2) Never mix a paste with less than one-half a gallon of water for one pound of chlorinated lime. (3) It

is not necessary or desirable to grind up or break up the lumps too thoroughly; nearly all the available chlorin dissolves readily, and too much agitation is detrimental to prompt settling. With these points in view a stock solution containing approximately 2 per cent of available chlorin may be made as follows:

Three hundred pounds commercial chlorinated lime (35 per cent available chlorin) = 105 pounds of available chlorin. Assuming a recovery of 100 pounds of this free from sludge, these 100 pounds must be contained in 600 gallons to give a clear 2 per cent solution. Due allowance must be made for proper washing of the sludge, for it contains, in addition to the suspended lime and silica, a solution of equal strength to that of the clear liquid. The amount of sludge is equivalent to about one gallon for each five pounds of chlorinated lime used.

Method of Dosing.—While the chlorinated lime treatment of water supplies is essentially simple, yet it requires able professional supervision; disappointing results will come from haphazard work. The great essential is a uniform dosing of a standard solution.

Economical working makes it desirable to use two tanks, each equipped with agitators and a looped chain as a drag along the bottom. These tanks should be made of concrete, or at least lined with cement, and adjustable means provided for drawing off the clear liquor from above, as well as an outlet for removing the sludge at the bottom.

The standard stock solution thus prepared will contain available chlorin equal to $\frac{1}{2}$ pound of chlorinated lime per gallon, or about 2 per cent available chlorin, or 6 per cent of chlorinated lime by weight. Hence an average clear water requiring 8 pounds of chlorinated lime per million gallons will require 16 gallons of this standard solution per million gallons of water. This is a trifle less than 1 drop of this solution containing 2 per cent of available chlorin in a gallon of water.

The table on the following page covers the range of chlorinated lime ordinarily used in water purification, and may be found useful for comparison.

Amount Used in Water Purification.—The remarkable germicidal power of chlorinated lime is better understood when it is known that 3 grains of a practically harmless substance will kill myriads of bacteria contained in a barrel of water. Ordinarily the amounts used are from 0.25 to 1.0 part "available chlorin" per million parts of water. This approximates 2 to 8 pounds of chlorin gas per million gallons of water, or 6 to 24 pounds of chlorinated lime per million gallons.

The amount of chlorinated lime necessary to add to a water in order to accomplish satisfactory results varies with the composition of the water. In general, the more organic matter the water contains the more bleaching powder is necessary. This is for the reason that some of the bleaching powder is used to oxidize the organic matter before it can produce its germicidal action. A bacterial reduction of 99 per cent may be obtained in a water containing little organic matter with 1 part per million or less, whereas

it requires up to 40 parts per million in sewage to affect a similar bacterial reduction; and still more for feces in a bed pan or cesspool.

Pounds Chlorinated Lime per 1,000,000 Gallons of Water	Parts Chlorinated Lime per 1,000,000 Parts Water	Parts Chlorin per 1,000,000 Parts Water	Grains Chlorinated Lime per Gallon of Water	Grains Available Chlorin per Gallon of Water	Drops Chlorinated Lime Solution, 2 Per Cent Chlorin or $\frac{1}{2}$ Pound Chlor- inated Lime (per Gallon), Used per Gallon Water
2.....	.24	.08	.014	.005	.25
4.....	.48	.16	.028	.009	.50
6.....	.72	.24	.042	.014	.75
8.....	.96	.32	.056	.019	1.00
10.....	1.20	.40	.070	.023	1.25
12.....	1.44	.48	.084	.028	1.50
14.....	1.68	.56	.098	.033	1.75
16.....	1.92	.64	.112	.037	2.00
18.....	2.16	.72	.126	.042	2.25
20.....	2.40	.80	.140	.047	2.50
22.....	2.64	.88	.154	.051	2.75
24.....	2.88	.96	.168	.056	3.00
26.....	3.12	1.04	.182	.061	3.25
28.....	3.36	1.12	.196	.065	3.50
30.....	3.60	1.20	.210	.070	3.75

Clark and Gage found that 0.1 part of available chlorin per 100,000 effected a satisfactory purification of the Merrimac River water; that is, results were obtained equal to slow sand filtration. *Bact. coli* was entirely eliminated. They discovered the interesting fact that the hypochlorite is a differential germicide, that it destroys some bacteria more readily than others. When small quantities are employed certain species growing at body temperature are only slightly affected.

In Pittsburgh it was found that 0.13 part of chlorinated lime, measured in terms of available chlorin per 1,000,000 parts of water, was sufficient to practically sterilize the Allegheny River water after it had passed the sand filters. It required as much as 1 part per 1,000,000 to accomplish the same results in the raw water. In Minneapolis from 2 to 4 parts per 1,000,000 were used. In the Jersey City case, already referred to, 5 pounds of bleaching powder, containing 35 per cent of available chlorin, were added to each million gallons of water treated. The raw water in this case was not highly polluted, ranging as low as 30 bacteria per cubic centimeter, and rarely going over 15,000. The number of bacteria in the treated water averaged only 15 bacteria per cubic centimeter, and *Bact. coli* was practically absent. It was found only once out of 455 samples.

Emergency Use.—The hypochlorite treatment has a very useful field of application in the disinfection of water on a small scale, as for military use, camps, tourists, explorers, and others. For *tourists* and *campers* a solution may be prepared by adding one-half a teaspoonful of chlorinated lime to one

pint of water. Use one teaspoonful of this to 10 gallons; 36 drops to 1 gallon; or 9 drops to 1 quart. Let stand at least 15 minutes.

Tablets of "Halazone"³ and other hypochlorites have been prepared for this purpose; they are effective only when fresh, for they are not stable.

Other Uses.—Bleaching powder is also used in the disinfection of the water of swimming pools, for street sprinkling and flushing, for the disinfection of feces and sputum; and to a certain extent, for the disinfection of glassware, fabrics, brushes, and combs. It is one of the best substances we have for the general disinfection of rough places, such as slaughterhouses, bakehouses, dairies, outhouses, cellars, and the like.

Summary.—The purification of water by means of a little bleaching powder is cheap, reliable, efficient, and so far as known, harmless and easy of application, all of which make it an attractive method. When added in proper quantities it leaves no undesirable chemical substance in the water. It must, however, be remembered that bleaching powder in no sense clarifies a water. In fact, turbidity interferes with its action to a certain extent. It cannot, therefore, render a turbid supply satisfactory. Furthermore, while chlorinated lime in such small quantities will kill bacteria it will not destroy organic matter nor remove discoloration, nor take away the unpleasant smells which raw waters often contain. It is, in fact, least effective when water is most polluted with organic matter. It is especially useful in a clean water; hence, polluted waters should have a preliminary process.

Chlorinated lime has a slight tendency to add to the hardness, while chlorinated soda renders the water correspondingly soft. The latter, however, is more expensive than the former.

Impure waters containing organic matter of any kind may, when treated with hypochlorites, give rise to unpleasant tastes. This is particularly true in the presence of phenol and some of its derivatives, when the obnoxious "iodoform" or "medicated" taste develops. Industrial wastes are usually the source of the phenol compounds, although they may be precipitated from a smoke-laden atmosphere. Experiments have shown that one part of phenol in 1,000 million parts of water will produce a noticeable taste after chlorination. The exact composition of the compounds giving rise to taste in the presence of chlorin is not known. The disinfection of impure waters is attended with uncertainty in the matter of taste production. Preliminary treatment for removal of impurities usually, but not always, gives better results.

In surgery chlorinated soda is used, the action of which is entirely analogous to chlorinated lime.

In spite of the enormous improvement effected in the safety of public water supplies in this country by chlorination, the danger of water-borne typhoid is by no means a thing of the past. Epidemics are occurring from time to time as a result of mismanagement, neglect and other causes.

³ Dakin and Dunham, *Brit. M. J.*, May 26, 1917.

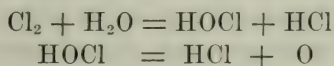
Chlorin.—Liquid chlorin may be used in place of chlorinated lime. In fact, chlorin has largely displaced bleaching powder. It is just as efficient and acts in practically the same manner. Its first use for the disinfection of a city water supply dates from December, 1912, at Niagara Falls, where the water power from the falls makes the chlorin that, in turn, disinfects the drinking water. By the end of 1918 there were about 2,500 liquid chlorin municipal plants in the United States. In 1925 over 7,000 were in operation, disinfecting over 75 per cent of the drinking water supplied to cities in North America.

Chlorin gas is formed by the electrolytic decomposition of salt solutions. The moist gas evolved from the electrolytic cells is dried and then compressed into a liquid in steel cylinders of about 100 pounds capacity. The gas is released, controlled and measured by special apparatus and introduced into the water (or sewage) in the proper proportions to effect disinfection.

There are two general types of apparatus: one by which the chlorin gas is introduced directly into the water; this is known as *dry feed*; another by which the gas is first dissolved in a small quantity of water and the resulting chlorin solution is piped to the point of application—*wet or solution feed*.

The gaseous chlorin process is covered by general process patents and the mechanical devices necessary for its application are likewise controlled by a few companies engaged in installing plants.

The action of chlorin gas in water is just like the action of the hypochlorites. Chlorin gas reacts with water to give the unstable hypochlorous acid which is quickly decomposed with liberation of nascent oxygen, thus:



The HCl is neutralized by the alkalinity of the water to form CaCl_2 .

The advantages of chlorin gas are such that it has rapidly replaced bleaching powder for the purification of water supplies. The gas can be obtained in a pure state, the dosage can be accurately controlled, it does not deteriorate on keeping, the apparatus is compact, and the results are uniform. The cost of treatment is usually less than for bleaching powder.

The quantities added are usually expressed in parts of chlorin per million parts of water by weight; 8.3 pounds of chlorin to 1,000,000 gallons of water is the equivalent of one part per million, since a gallon of water weighs 8.3 pounds. The amounts used to disinfect the water are the same as stated under chlorinated lime, and vary in practice from 0.25 to 1.0 part per million, depending upon the amount of organic matter present in the water.

Good results on the typhoid rates have followed the chlorination of the water in Chicago, Baltimore, Jersey City, Milwaukee and elsewhere. Chlorination plants should always be installed in duplicate, to provide against accident or emergency. The use of a polluted water supply seems to give a

communal tolerance or immunity not evidenced in cities using a pure or purified source of supply. Thus, it is often noted that typhoid and diarrheal outbreaks are especially severe and explosive in case of a breakdown or failure of the chlorinating process.

Chlorin is used to disinfect the water supply of Buffalo, New York City, Philadelphia, Chicago, Richmond, Baltimore, Detroit, Louisville, New Haven and Stamford, Connecticut; New Brunswick, New Jersey, and very many other cities. It was also used both in the United States and abroad in many government camps, military establishments and field units during the World War.

Many state boards of health have provided traveling emergency chlorin outfits for use in epidemics. Louisiana was the first state to use a railroad coach as a laboratory for water analysis, and a garage for housing machines used in collecting samples and for fighting water-borne epidemics. New Jersey uses a small automobile for both purposes, while other states merely keep hand-operated chlorin apparatus for emergency work. Uruguay, because of distances, has equipped several railroad coaches with emergency chlorin outfits.

The rapidly extending use of the process of disinfection by means of chlorin and its compounds has been the most striking tendency of the past decade in the art of water purification. The practice has included the treatment of both raw and filtered waters and has been a great factor in the elimination of water-borne diseases. Experience has shown the general efficacy of the treatment, but it is also true that blind reliance has been placed at times solely upon the use of chlorin when the dictates of sanitary science would have called for the inclusion of other protective measures.

Grossly contaminated water and that which is charged with large amounts of turbidity and organic coloring matter should first have the sources of objectionable constituents removed, or else be rendered reasonably clean by long storage or filtration. Chlorination will then give the fullest measure of protection because it will not have to bear the entire burden of purification, and will not have its active agent neutralized by substances it is not designed to remove. In the rôle of a "finishing" process, chlorin finds its greatest and most logical use, and here provides the final protective barrier.

PERMANGANATE OF POTASH

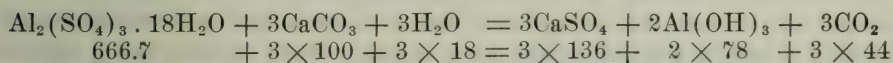
Permanganate of potash was much used in India, particularly in wells during cholera epidemics; also in water tanks on board ships, and other places. Enough permanganate is added to secure a faint pink tinge, which indicates a slight excess. The permanganate acts as an oxidizing agent precisely as ozone, or similar to the hypochlorites. It is a powerful germicide, but not sufficiently so in the strength used to depend upon it. If too much is added to wells, springs, etc., so as to kill the fish, frogs, and turtles, the water may be spoiled by putrefaction of their dead bodies. Like all chemical methods, the action is not continuous; the agent expends itself in

oxidizing organic matters before attacking the bacteria, and the amount necessary for the purification of a water depends, therefore, upon the amount of organic impurities in the water.

Experiments by Clark and Gage show that complete sterilization is not obtained by the use of permanganate of potash. Over 98 per cent of the bacteria were eliminated by treating water with 0.5 part to 100,000 in from four to six hours. Larger amounts of potassium permanganate or longer time gave no better results. Potassium permanganate has a comparatively low efficiency with a relatively high cost, which will always limit its usefulness. Further, the method is difficult of practical application, being rather slow. It also increases the color of the water on account of the reduction of permanganate by organic matter, resulting in the formation of brown colloidal manganese compounds. Permanganate has found an important application in the London water supply for the prevention and removal of chlorin after-tastes.

COAGULANTS

Alum or Sulphate of Aluminum.—The single and double sulphates of aluminum have long been used to clarify turbid waters. In the amounts used they have no direct germicidal action. The action is entirely an indirect one, and depends upon the fact that the alkaline carbonates react upon the alum to form aluminum hydroxid. This salt has a large colloidal molecule, and, being insoluble, is thrown out of solution as a flocculent precipitate which entangles much of the suspended matter and bacteria as it forms and falls. In a sense the purification of water with alum corresponds very much to the clearing of coffee with the white of egg. Some of the aluminum hydroxid may also combine directly with the organic matter to form undetermined compounds. The reactions are as follows:



1 grain of alum per gallon = 143 lbs. per million gallons.

1 " " " " " = 17.1 parts per million parts of water.

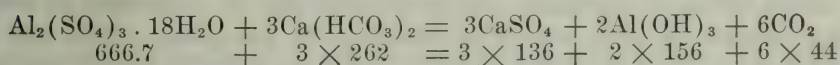
1 " " " " " requires therefore

$$17.1 \times \frac{300}{666.7} = 7.7 \text{ parts per million of alkalinity expressed as CaCO}_3.$$

100 lbs. of alum per million gallons = 5.5 parts per million of alkalinity as CaCO₃.

$$7 - 7 \times \frac{100}{143} = 5.5$$

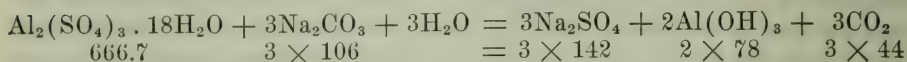
In water, however, the reaction is with calcium bicarbonate:



Therefore 1 grain of alum per gallon liberates 6.8 parts per million CO₂.

Also 1 grain of alum per gallon converts 7.7 parts per million bicarbonate alkalinity to 7.7 parts sulphates or incrustants, all expressed in terms of CaCO_3 .

Reactions using soda ash with alum:



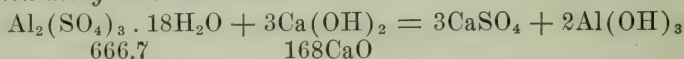
1 grain per gallon alum requires $\frac{318}{666.7}$ of 17.1 = 8.2 p. p. m. Na_2CO_3 .

1 p. p. m. = 8.3 lbs. per million gallons.

1 p. p. m. alkalinity as $\text{Na}_2\text{CO}_3 = 8.3 \times \frac{106}{100} = 8.8$ lbs. Na_2CO_3 per m. g.

1 grain per gallon alum liberates $\frac{132}{666.7}$ of 17.1 = 3.4 p. p. m. CO_2 .

Reaction using lime with alum:



1 grain per gallon alum requires $\frac{168}{666.7}$ of 17.1 = 4.3 p. p. m. $\text{CaO} = 36$ lbs. per m. g. CaO .

This liberates no CO_2 .

1 grain per gallon alum with lime increases hardness 7.7 p. p. m.

It will be seen that if alum is added in quantities which do not combine with all the alkalinity present there is left in the water no undesirable constituent. This is important, for there is a popular prejudice against the addition of a chemical, especially alum, to drinking water. It has already been pointed out that there are many turbid waters in our country which contain silt in such fine subdivision that even prolonged sedimentation and repeated filtration will not render them entirely clear; likewise many colored waters cannot be made attractive without the use of a coagulant.

In the use of alum good results depend upon adding it in just the right amount. The quantity will vary with the turbidity and color, the amount of calcium carbonate contained in the water and the hydrogen-ion concentration. The usual amount of alum added to water is from 1 to 3 grains per gallon. This should be carefully determined from time to time, for if not enough alum is added the result is incomplete, and if too much is added it remains in the water as such. The process therefore needs constant supervision, for turbid waters usually come from turbulent streams, which are subject to sudden variations. If the process is left to automatic devices or placed in incompetent hands it is sure to give disappointing results.

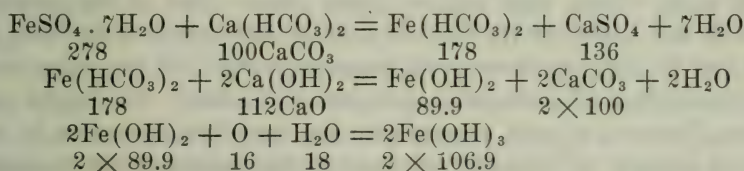
Alum or other coagulants are never used for the treatment of water unless followed by some method of removing the precipitate. This may be sedimentation, filtration (either slow or rapid), or, as is the general practice, by both. So it is that the addition of a coagulant is not a process sufficient

in itself for the purification of water, for it does not destroy bacteria or dispose of inert impurities. Sedimentation following the addition of alum may be for a period of a few hours to a few days depending upon the treatment to follow. With rapid sand filters, sedimentation of a few hours is customarily employed after the coagulating chemical is added.

Most plants have been dependent upon the open market for alum. Hoover of Columbus devised in 1916 a practical method for making alum at the plant. Two parts of commercial sulphuric acid are mixed with one part of low grade bauxite ore and poured into flat boxes. In a few hours alum cake forms in the pans, and is used without further treatment. The product is considerably cheaper than commercial aluminum sulphate.

Sulphate of Iron and Lime.—This combination is used in many places. At St. Louis it was introduced as an emergency installation to clarify the muddy waters of the Mississippi, in order to make a good impression during the Louisiana Purchase Exposition in 1904. It gave such satisfactory results that it was decided to continue its use.

Reaction using lime and iron (ferrous sulphate) when the iron is added before the lime:



1 grain per gallon $\text{FeSO}_4 \cdot 7\text{H}_2\text{O} = 17.1 \times \frac{100}{278} = 6.2$ p. p. m. alkalinity
and 100 lbs. per m. g. = 4.5 p. p. m. alkalinity.

1 grain per gallon FeSO_4 converts 6.2 p. p. m. alkalinity to 6.2 p. p. m. incrustants.

To precipitate the ferrous bicarbonate first formed requires $17.1 \times \frac{112}{278} = 6.9$ p. p. m. $\text{CaO} = 57$ lbs. per m. g. for each grain per gallon FeSO_4 .

If the lime contains 85 per cent CaO then $57 \times \frac{100}{85} = 67$ lbs. per m. g. will be required for 1 gr. per gal. FeSO_4 .

The dissolved oxygen in the water completes the reaction, forming brown $\text{Fe}(\text{OH})_3$.

Lime and iron are cheaper commodities than sulphate of aluminum. Their application is much more difficult to control adequately, and it should never be undertaken except with the assistance of a competent resident chemist and good appliances for maintaining a required chemical feed. At St. Louis the water is subject to the iron and lime treatment,⁴ followed by

⁴For the year 1923-24 an average of 5.96 grains per gallon of lime, 1.44 grains of ferrous sulphate and 1.09 grains of alum was used. To the filtered water an average of 3.22 pounds per million gallons of chlorin was applied.

subsidence in large basins in which the bulk of the precipitate settles. This clarified water is then treated with alum, filtered and chlorinated. New Orleans uses a similar method, but does not employ alum after lime and iron treatment.

COPPER SULPHATE

The use of copper sulphate in drinking waters was proposed by George T. Moore of the United States Department of Agriculture in 1904. The original claim was that copper sulphate in minute amounts would poison algæ which produced objectionable tastes and odors, and the further claim was made that it was also capable of destroying typhoid and other pathogenic microorganisms. We know now that copper sulphate in great dilution is a specific poison for many algæ and other microscopic organisms, but that it has little or no effect upon typhoid, cholera, or dysentery bacilli in the amounts used. The specific action of copper sulphate changes the biologic balance of the microscopic growth in water, which may be favorable or unfavorable, depending upon conditions.

Copper sulphate is used in the proportion of 0.1 to 0.25 part per 1,000,000 parts of water. Some algæ require larger doses. Most of the copper combines with the bodies of the microorganisms and settles with them to the bottom and in this way is removed from the water. If the water is afterwards filtered most of the remaining copper is removed. The copper remaining in the water is in such minute amounts that there seems to be no real danger in using it in this way or even in its occasional use in somewhat larger doses where the water is very bad.

The method of applying the copper is to place weighed quantities of the copper sulphate in loose cloth bags and to tow them back and forth with boats through the water of the reservoir until the material is dissolved. It should be remembered that, while the copper kills some species of organisms in the amounts used, it has little effect upon others. In fact, it permits the growth of certain species by removing the retarding symbionts, thus clearing the way for stronger growths of the forms that are not directly affected. Copper sulphate may therefore entirely change the flora in a reservoir. This change is frequently accompanied by a great improvement in odors and tastes. On the other hand, the destruction or suppression of one species may be followed by an overgrowth of an equally objectionable and more hardy form. Therefore the results from the use of copper sulphate for the correction of odors and tastes in water vary from complete successes to utter failure.

It is clearly established that copper sulphate in the amounts mentioned above does not prevent or even materially reduce putrefaction and the tastes and odors resulting from it. According to Hazen, the method of treating water with copper sulphate is easily and quickly applied, and considerable good has come from it. The correction is only partial, however, and is not permanent. It is not therefore to be relied upon in all cases.

ULTRAVIOLET RAYS⁵

Recently the well-known germicidal power of the ultraviolet rays has been put to practical use in the sterilization of water and other substances. These rays, of short wave length, may be obtained from the Cooper-Hewitt mercury vapor lamp, which is very rich in ultraviolet rays.⁶ Nagier conceived the idea that this lamp might be used for the sterilization of water, and the experiments made in France, England, and elsewhere show this assumption to be correct. As glass is opaque to ultraviolet rays, it is necessary to use quartz or lamps made of fused silica. The apparatus used in the experiments of Thresh and Beal consisted of an aluminum cylinder about 12 inches long by 6 inches in diameter containing a Cooper-Hewitt quartz lamp with an internal diaphragm, which causes the water entering at one end to travel along the cylinder in close proximity to the lamp. By an ingenious arrangement the moment the light goes out the flow of water is stopped. This small apparatus is capable of sterilizing 50 to 200 gallons of water per hour, depending upon the character of the water. In clear water many of the bacteria are killed in from 5 to 20 seconds. The resisting spores succumb in from 30 to 60 seconds, *Bact. coli* in from 15 to 20 seconds, *B. typhosus* from 10 to 20 seconds, cholera vibrio 10 to 15 seconds. The current used in these experiments was 6 amperes and 130 volts. The presence of colloidal material or turbidity retards the action of the rays. The results show that a fairly clear and bright water may be practically sterilized by exposure to ultraviolet rays for a brief time. The bacteria are killed by exposure to the direct action of the ultraviolet rays themselves. The action is not influenced by temperature (0°-55° C.) and oxygen content of the water. The formation of H₂O₂ is not necessary to produce sterilization. The process does not in any way clarify the water.

Ultraviolet rays should preferably be applied to water free of turbidity and color or filtered. Overdosing gives no detrimental effects other than increased cost. The water is not changed in any way chemically. Turbidity of the water or accumulations on the lamps greatly impair the efficiency of the rays. Direct current is now used at 110 to 500 volts. A continuous good effluent requires constant voltage and that the lamp be in good condition.

Marseilles adopted the ultraviolet ray process to purify its water supply. There were preliminary roughing filters, and the water passed the quartz tube mercury arc lamp three times. No *Bact. coli* were found in the treated water, and the total bacterial reduction was 98.3 per cent. Many plants have been built in this country. In 1915 there were 320, comprising twenty for swimming pools, but none have been installed in large cities. At Henderson, Kentucky, the process was adopted for disinfecting water previously passed

⁵ See also page 1336.

⁶ Literature on the subject is abstracted in the *U. S. Pub. Health Rep.*, 1919, 133: 2821.

through rapid sand filters. Good bacterial results were reported. At Berea, Ohio, a small municipal supply is treated with ultraviolet rays.

The general experience has been in the United States that the ultraviolet process is not capable of maintaining high efficiency and giving proper protection at times when the physical qualities of surface waters suffer impairment. Even the effluents of filter plants are subject to short periods when the water is slightly turbid or colored. As stated above, the rays do not effectively penetrate such water, which is in greater need of disinfection than water of normal quality. This process has not been able to compete with chlorin because the process on a large scale is expensive.

Installations are often found in houses, hotels, steamships and swimming-pools which are using a physically clean supply, and satisfactory results are obtained. Ease of operation and the absence of chemical and physical effects on the water commend the use of the process.

CHAPTER V

WATER AND ITS RELATION TO DISEASE

Water is a vehicle for certain infections such as cholera, typhoid fever, dysentery, and other diseases having their primary seat in the digestive tract. It may carry inorganic poisons such as lead. It is responsible for a large group of nutritional and dietetic disorders less well understood. It may lack qualities which bring about derangements of metabolism resulting in such conditions as goiter; further, it may be the medium for carrying infections now not generally regarded as water-borne, or it may lower resistance so as to favor infections not water-borne. It is also occasionally responsible for conveying animal parasites, amebæ, worms, etc. From time to time new troubles are disclosed—recently epidemics of infectious jaundice have been traced to water.

While water has an established place among the carriers of certain infections, it has not a supreme or exclusive place, and this should be kept carefully before us. The tendency to exaggerate the importance of water as a bearer of disease and death has sometimes led to overstatement. The facts are bad enough and do not require extravagant language to emphasize their importance. The greatest danger in water is pollution from human sources. All the discharges from the body: urine, feces, expectoration, secretions from the nose, and washings from the skin, find their way sooner or later into our streams, especially where modern water-carriage systems are installed for the disposal of wastes. All sewage-polluted water must be regarded as dangerous, whether there are any known cases of typhoid fever on the watershed or not. It is highly probable that the sewage of large communities always contains typhoid bacilli in larger or smaller numbers, because in large communities typhoid fever does not die out completely at any time, and carriers and missed cases are always liable to be present.

Water differs in several essential particulars from any other article of diet. Above all, it is partaken of raw, while perhaps 90 per cent of all our other food is disinfected by cooking before it is used. Again, it is a vehicle which comes in contact with many objects spread over broad acres, and it is the natural vehicle for the removal of wastes from these areas. Its great solvent and erosive powers favor this action.

The relation of water supply to sickness and death has been shown with force in many cities, notably at Lowell and Lawrence, Massachusetts; in Albany, New York; at Jersey City and Newark, New Jersey; at Philadelphia and Pittsburgh, Pennsylvania; at Chicago, Illinois; and abroad at London, Paris, Hamburg, Altona, Berlin, and many other cities.

THE MILLS-REINCKE PHENOMENON

Following the filtration of the water supply at Lawrence, Massachusetts, in September, 1893, Mr. Hiram F. Mills, a member of the State Board of Health of Massachusetts, noted that a marked decrease in the general death rate of the city, and not merely in the death rate from typhoid fever, was taking place. About the same time (May, 1893) filtered Elbe River water was furnished the city of Hamburg, and Dr. J. J. Reincke, health officer of that city, in his successive annual reports, noticed that the general death rate was declining more rapidly than could possibly be accounted for by the deaths from typhoid fever alone. To this phenomenon Sedgwick and MacNutt have given the name of the "Mills-Reincke phenomenon."¹ In 1904 Mr. Allen Hazen, a sanitary engineer, formulated a numerical expression for the comparative effect of water purification upon typhoid fever mortality and total mortality. He said that, "where one death from typhoid fever has been avoided by the use of a better water, a certain number of deaths, probably two or three, from other causes have been avoided." Sedgwick and MacNutt examined the vital statistics of the cities of Lawrence, Massachusetts, and Hamburg, Germany, and also of Lowell, Massachusetts, Albany, Binghamton, and Watertown, New York. They found evidence of the life-saving power of a purified water upon many diseases other than typhoid fever in the cities studied, except Watertown. The method of purification used at Watertown, and also at Binghamton, was rapid or mechanical filtration.

One of the most surprising results of these studies is the disclosure of the remarkable relation existing between polluted water and infant mortality. This was emphasized especially by Dr. Reincke at Hamburg. Closely associated with infant mortality stand diarrhea and gastro-intestinal disorders in relation to polluted water.

McLaughlin² has also noted the relation of a sewage-polluted water to infant mortality, and concludes that it is certain that in practically every instance, in addition to a lessened number of deaths from typhoid fever, the substitution of a safe for a polluted water supply results in the saving of many lives from diseases which are not reported as typhoid fever. Hazen's theorem has also been studied by Arthur Lederer,³ who finds a large number of affirmative statistical results.

More recent evidence from Providence, Cincinnati, Columbus, Pittsburgh, and Philadelphia does not tend to bear out the Mills-Reincke theory, except perhaps in regard to diarrheal diseases in Pittsburgh. The proposition is not demonstrated and it would be exceedingly unwise to promise a marked lowering of the general death rate as a result of the purification of water supplies alone.

¹ W. T. Sedgwick, J. S. MacNutt, *J. Infect. Dis.*, 1910, 7: 489.

² *U. S. Pub. Health Rep.*, 1912, 26: 579.

³ *Am. J. Pub. Hyg.*, 1910, 304.

NON-SPECIFIC DISEASES DUE TO WATER

Impure water is responsible for disorders other than the specific gastrointestinal infections, but these disorders are often obscure or overlooked. It is not always plain just what quality or what impurity in the water is responsible for these non-specific disorders, and the diseases themselves may present a vague and ill-defined clinical picture. The relationship has been worked out in only a few instances.

A turbid or malodorous water may not in itself be particularly injurious to health, but, on account of its unattractive appearance or repulsive condition, less may be taken than is necessary for the maintenance of good health. In this way water may be indirectly responsible for much harm. The drinking of too little water is a very common dietetic error.

While a polluted water may not carry specific germs, it is conceivable that it may so undermine health or lower resistance as to favor infections not usually associated with the digestive tract. This may bear hardest on the young, the old, or the enfeebled.

From the nature of the case the effects of an impure water cannot always be measured by gross results, but the cumulative or separate action of small effects often repeated may result in deranged digestion, altered metabolism, irritation of delicate membranes or sensitive organs and structures, which may lead to or hasten the course of chronic diseases.

The *organic matter* in the quantities usually contained in a natural water is not of itself harmful. This organic matter, however, does not stay in its native state, but soon decays and it is suspected that some of the intermediate products of decomposition may have toxic potency. Ordinarily these toxic substances are in minute quantities, or at least in great dilution, but under certain circumstances they may accumulate in noticeable concentration. Further, while persons habitually taking such toxic substances may soon become immune, the newcomer will not be so fortunate. The case of organic matter in water is not a clear one, and sanitarians have ever erred on the safe side in condemning waters containing much organic matter. It is well known that if the organic matter is not derived from sewage it is probably harmless. Thus, in the case of organic matter of vegetable origin, Mason has been able to find but few cases of illness traceable to peaty waters. In such instances the patients suffered from a mild and transient form of diarrhea, the cause of which is uncertain.

As far as the *inorganic impurities* usually found in water are concerned, the chlorids, carbonates, sulphates, and silicates, and lime, magnesia, and aluminum can scarcely be harmful in the amounts ordinarily found. In fact, most of the mineral matter carried by water is needed by the body, especially lime. The trace of iodine in water is our main source of this element in the diet. It is commonly stated that water containing 500 parts per million, or 30 grains per gallon, of clay and silt is unfit for drinking purposes,

on account of its irritating effects upon the gastro-intestinal tract; but beyond this probability, turbidity is of no special sanitary significance, unless the water also contains metallic poisons or objectionable qualities.

An attempt has frequently been made to correlate the formation of concretions such as urinary and biliary calculi with the inorganic salts in hard water. We now know that biliary calculi usually form about a colon bacillus or a typhoid bacillus or about some pathological particle as a nucleus, and that urinary calculi probably have a similar pathogenesis. There is no known relation between these concretions in the body and the inorganic salts in water, even those in a very hard water. It is stated that a change from a soft to a hard water causes diarrhea.

GOITER

Goiter (from guttur, throat) is a chronic enlargement of the thyroid gland, and may be due to a variety of causes.

The functions of the thyroid gland manifest themselves through its internal secretion, which contains a specific hormone rich in iodine. The thyroid gland controls metabolism, and the rate of metabolism is therefore an index of the activity of this gland; through its action on other endocrine glands, it influences reproductive and digestive functions; and exerts some control on the functions of other organs. One of the chief functions of the thyroid is to stimulate and maintain the energy utilization of the body at a normal level, in other words to regulate metabolism. Hypersecretion raises the rate of metabolism of all foodstuffs, so that protein, carbohydrates and fats are consumed at an increased rate. The thyroid also exerts some control of the glycogenic content of the liver, for in hyperthyroidism the liver is not capable of the normal storing of glycogen, although the body is still able to metabolize carbohydrates in usual amounts. In deficient secretion (hyposecretion) there is a diminished and retarded growth of tissues and a lowered metabolism.

The thyroid has some relationship with the sex organs,⁴ especially in the female, the nature of which is at present but poorly understood. There often is increase in size of the thyroid at puberty or during menstruation, pregnancy and lactation. There is also some evidence that it has a detoxicating action on certain poisonous substances, but our knowledge here is meager.

There are three diseases due to disturbed function of the thyroid gland: (1) *hypothyroidism*, associated with atrophy of the gland and diminished functional activity; (2) *hyperthyroidism*, associated with enlargement of the gland and increased functional activity; (3) *simple goiter*, probably compensatory, associated with enlargement of the gland, but with little or no constitutional manifestations.

Hypothyroidism.—*Cretinism* occurs congenitally or develops in early years of childhood. There is marked retardation of growth, the sex glands remain

⁴In the king scorpion the thyroid is a reproductive gland.

in an infantile condition, the skin shows defective development, and the mind remains in the condition of feeble-mindedness or idiocy. Cretinism may be prevented by giving the mother sufficient iodine during pregnancy, and the condition may be materially helped by the use of thyroid extract. The improvement is often extraordinary, but develops only to a certain point, where it stops. The condition reverts if the extract is discontinued. *Myxedema* of adults, or Gull's disease, occurs mostly in women between the ages of thirty and fifty years. It is characterized by lowered metabolism, loss of expression and memory, thickening of the skin, and subsequent infiltration with a peculiar mucoid edema.

Hyperthyroidism.—Exophthalmic goiter (also known as Graves', Basedow's or Parry's disease), is an enlargement of the thyroid gland with increased functional activity. The chief symptoms are rapid heart beat, exophthalmos or protrusion of the eyeballs, goiter, tremor and nervousness, high blood-pressure and increased rate of metabolism. The etiology is obscure.

ENDEMIC OR SIMPLE GOITER

Simple Goiter.—*Simple goiter* is of special interest to the sanitarian because it is frequent, widespread and readily preventable, even curable if not too far advanced. Simple goiter is a deficiency disease, due to lack of iodine in water, food, or both.

Simple goiter includes all cases of enlargement of the thyroid, except toxic goiter, exophthalmic goiter, thyroiditis, and true neoplasms of the thyroid. Simple goiter embraces types formerly called endemic, epidemic and sporadic goiter; adolescent goiter and goiter of pregnancy; also non-toxic, parenchymatous goiter, colloid goiter, and cystic goiter. It is also called struma or bronchocele.

Simple goiter is a deficiency disease characterized by a non-inflammatory enlargement of the thyroid gland, without marked functional disturbances. The enlargement is compensatory, that is, a defensive reaction to iodine starvation. The goiter is a matter of concern to the patient, because (1) it is disfiguring;⁵ (2) it may cause pressure symptoms on the trachea or surrounding structures; (3) it may be responsible for constitutional disturbances such as apathy, chilliness, constipation, asthenia and mental apprehension.

Simple goiter is rarely congenital. It usually starts at or about puberty, and the tendency diminishes after the twentieth year. Females are more susceptible than males, the ratio varying with age. The sex predisposition probably finds its explanation in the fact that the thyroid gland is somehow associated with the physiology of the female generative functions. There are three critical age and sex periods: (1) during fetal life, (2) during adolescence, (3) during pregnancy and lactation. Simple goiter is endemic,

⁵The full goitrous throat is so common in some localities that it is regarded as a type of beauty. See pictures of Burne-Jones and many other artists.

but sometimes occurs sporadically. Epidemics have been noted, but usually in endemic localities.

Prevalence.—Goiter is prevalent in those regions where there is a deficiency of iodine in the water and soil. Iodine is especially lacking in glaciated regions away from the sea. The classic home of simple or endemic goiter is in the Swiss Alps. The disease is prevalent in all of Switzerland, northern France, northern Italy and the Balkan states. The traditional seat of goiter in England is in Derbyshire ("Derbyshire neck"), while Sussex and Hampshire have also been affected. There are many endemic centers in the Himalayan Mountain regions of eastern and southeastern Asia, the Gilgit district of northern India, and the plateau regions of western China and Mongolia. In South America, it is found in the Andes Mountain regions, the most noted section of which is the Peruvian plateau. Goiter also occurs in Africa and Mexico. In Japan the malady is infrequent, probably because the people regularly partake of marine plants which are rich in iodine.

The absolute number of goiter subjects in countries with high endemic index is of great social and economic importance. In France, Mayet (1900) estimates the number at 400,000. The drain on the country is better expressed by the number of cretins. In Cisleithan, Austria, there were in 1883 a total of 12,815, or 71 per 100,000; in one district in Styria a proportion of 1,045 in 100,000. In Piedmont, Lombardy, and Venetia there were in 1883, 12,882 cretins in a population of 9,565,038, a rate of 135.5 per 100,000.

In Europe a high incidence is reported among school children. In Bavaria, Schittenhelm and Weichardt found an incidence as high as 77 to 89 per cent of the total school population. Kraeuter⁶ examined, in Munich, 1,840 school girls between the ages of three and nineteen, and found 56 per cent with goiters of various sizes.

Goiter in the United States.—The early explorers found goiter among the North American Indians, as Munsen has in more recent times in the Eskimos. The region of our Great Lakes shows considerable numbers; also sections of the Middle West and parts of the Rocky Mountain regions, but in the United States and Canada the goiters are usually not large and cretinism is rare.

Marine and Kimball⁷ in April, 1917, examined 3,872 school girls in the second decade of life, in the city of Akron, Ohio. Of this number, 2,184, or 57 per cent, were found to present simple goiter. The frequency increased with the years of age, being 41 per cent for the years ten to twelve, inclusive, and 60 per cent for the years eighteen to twenty. In West Virginia, Clark⁸ examined 13,836 school children of 11 counties, in 1913, and found 1,234 cases of goiter, which is 9 per cent of the number examined. In Virginia, the same worker examined 6,432 school children in 9 counties,

⁶ *München, med. Wchnschr.*, 1922, 69: 47.

⁷ *J. Lab. & Clin. M.*, 1917, 3: 1.

⁸ *U. S. Pub. Health Rep.*, 1914, 29: 939.

and found 817 cases of goiter—12 per cent. In Huntington, West Virginia, 50 per cent of the girl students were found to be affected; in Virginia, less than 0.1 per cent of the goiters were among boys. Hall⁹ found 18 per cent of 2,086 men at the University of Washington to have enlarged thyroid glands, and 31 per cent of 1,252 women students. Smith,¹⁰ an army surgeon, in 1918 examined 65,507 men between the ages of eighteen and thirty-one at Jefferson Barracks, and established an incidence of 1.63 per cent. Kerr¹¹ found 21 per cent of 21,182 troops at Camp Lewis, Washington, to have goiter.

Olesen¹² surveyed 47,493 school children in Cincinnati, and found 26.6 per cent among boys and 39.8 per cent among girls. After the age of thirteen, goiter decreased in the boys, but increased in the girls. In the University of Wisconsin, 28 per cent of 13,706 students had thyroid enlargement, and 6 per cent exophthalmic goiter. There were twice as many females as males.¹³ In Utah, Wallace¹⁴ studied 69,256 pupils in 1925, and found 31 per cent of the boys and 54.3 per cent of the girls with enlarged thyroid. The condition is much more prevalent in some places than in others. It was found in this region that communities getting their water supplies from the hills generally show a higher incidence than those using well water.

Goiter in Animals.—Sheep and swine, and also mules, horses and pigs have goiter, though not constantly, in endemic centers. Marine and Lenhart¹⁵ also observed goiter among brook trout in fish hatcheries; rats and mice are susceptible. Marine¹⁶ found that 90 per cent of the dogs in Cleveland are affected with goiter. It is perhaps an index of our comparative concern in human and animal welfare that goiter among animals was the object of prophylactic measures before that of man received much attention. The early days of the sheep industry in Michigan were found to be unprofitable in that a large percentage of the animals presented goiter. The abandonment of the industry was contemplated, when salt mines were opened up in the vicinity of Detroit. Heretofore, the sheep had been fed with salt obtained from a remote source. It was soon observed that the sheep fed with the local salt were better in every way; the young were born free of goiter and did not subsequently develop it. Careful investigation established the fact that the local supply of salt contained minute traces of iodine as an impurity. In Montana alone, Smith reports that about 1,000,000 pigs were lost annually on account of this disease. The condition was present at birth, the young pigs being born hairless and generally stunted. Many were dead at birth, and few survived more than twenty-four to thirty-six hours. The affected areas in some instances are sharply demarked. At times

⁹ *Northwest Med.*, 1914, n. s., 6: 189.

¹⁰ *J. Am. M. Ass.*, 1919, 72: 471.

¹¹ *Arch. Int. Med.*, 24: 347.

¹² *U. S. Pub. Health Rep.*, 1924, 39: 1777.

¹³ *Wis. M. J.*, 1921, 19: 561.

¹⁴ *Calif. and West. Med. J.*, Sept. 1924.

¹⁵ *Johns Hopkins Hosp. Bull.*, 1910, 21: 95.

¹⁶ *Arch. Int. Med.*, 1918, 22: 41.

the district is confined to a creek bottom one-half mile wide. The affected pigs had large thyroids with low iodine content. The addition of small quantities of iodine salts to the food eliminated the condition.

Goiter occurs among fish, seldom in the native state, but usually under conditions of artificial propagation. It was at first thought that the amount of goiter in any hatchery is in proportion to the uncleanness and general lack of sanitation in the hatchery in which the fish propagate. Through the work of Marine and Lenhart, the nature of the disease was established and simple means were evolved for its reduction. In general, the measures consist of continued cleanliness of the fisheries and the supplying of food containing proper constituents, but particularly to the addition of small traces of iodine or iodine salts to the water in which fish live.

Relation to Water.—The cause of goiter was long a mystery, and certain features of the condition are still not entirely clear. It has long been associated with drinking water. As early as 1850, Chatin¹⁷ advanced the hypothesis that simple goiter is correlated with a low iodine content of drinking water, but his opinion was not accepted. Remarkably good effects have been obtained in parts of Switzerland and Italy by changing the drinking water. There are goiter wells in France and Switzerland, the waters of which were used successfully for the intentional production of the disease with a view to escaping compulsory military service.

The relation of water to goiter is also illustrated in Vienna. This city long boasted of the best water among all European cities. It is brought in long aqueducts and subterranean pipes from the Schneeberg, a mountain group about 6,000 feet high and 85 miles to the north of the city. This water, used since 1872, put a stop to typhoid and other gastro-intestinal diseases. The water comes from limestone formations, and has a low degree of hardness, owing to the absence of vegetation upon the catchment area. Since 1873 the number of goiters in Vienna has increased 200 per cent, and popular belief always pointed to the water as the cause. The water used by the inhabitants in many of the goiter regions in Switzerland comes from similar limestone formations.

Further presumptive evidence that goiter is a water-borne disease is found in certain villages in the Gilgit District in India. Here eight villages adjacent to each other derive their water from a neighboring stream, and all are badly affected with goiter. Another village in the same district takes its water from a spring and has no goiter.¹⁸

Another instance in which the change of water supply is said to have influenced the prevalence of goiter is that of the village of Bozel in Tarentaise. In this village, during 1848, of a population of 1,472, there were 900 cases of goiter and 109 cretins. About this time a new water supply was introduced from a source only 800 meters distant, and sixteen years afterward, among practically the same population, there were only 39 cases of

¹⁷ *Compt. rend. Acad. de sc.*, 1850.

¹⁸ McCarrison, *Ind. J. Med. Res.*, 1914, 2: 778.

goiter and 58 cretins. On the other hand, goiter has appreciably increased in the Pacific Northwest since the introduction of a chemically and bacteriologically pure water from the Cascade Mountains.

Recent studies indicate that goiter prevails in any area directly in proportion to the deficiency of iodine in the drinking water. In the United States there is a clear correlation between the prevalence of goiter and deficiency of iodine, but owing to migration and travel habits of the population, and the transportation of food and drink, such sharp distinctions as are found in Switzerland and India are obliterated. Furthermore, this generalization does not apply to all districts. For example, in Provincetown, Massachusetts, and other localities in which simple goiter is relatively rare, the presence of iodine cannot be demonstrated. Obviously the iodine required for the maintenance of thyroid equilibrium in these regions is obtained from food, especially crustaceans and deep sea fish.

The Rochester Experiment.—An interesting experiment was begun in Rochester, New York, in April, 1923, to supply the iodine deficiency of the drinking water. Rochester is located in the goiter district of the Great Lakes. Dr. Goler, Health Officer of the city, conceived the idea of adding sodium iodide to the water for the purpose of preventing goiter.

The program consists in the daily dosing of the entire supply for a period of three weeks in the spring and three weeks in the fall. The quantity of sodium iodide used is 16.6 pounds per day, or 0.664 pounds per million gallons, which is equivalent to 75 parts per billion. The city water contains normally only 4 or 5 parts per billion, and the amount actually found in the city tap water after treatment with the iodide is somewhat more than 50 parts. The cost of the experiment is estimated to be about \$3,000 per year, or one cent per capita per year. This is on the basis of \$4.35 per pound for 7,000 pounds of sodium iodide.

It is too early to appraise the results of this novel method of prevention. Opinions vary as to the practicability of the measure. To some it seems wasteful and also uncertain in efficiency and dosage. Time will tell.

Iodine and the Thyroid Gland.—The idea that goiter is due to the lack of iodine is not new. That iodine exerts a definite influence upon the thyroid gland has been known for centuries. The early Greeks treated goiter by administering the ash of burned sea sponges, a substance rich in iodine. Beginning with the intentional administration of iodine by Coindet¹⁹ in 1820, this medicament experienced an extensive vogue in goiter therapy for a period of seventy-five years. Prevost, Maffoni, Inglis, Marchand and Niepce associated goiter with a lack of iodine, but it was particularly Chatin²⁰ who claimed to have demonstrated a lack of iodine in the air, soil and water in districts of endemic goiter. His claims were attacked, and unfortunately so discredited on the grounds of faulty and inadequate methods that further study of this important subject was blocked for forty-five years. It was

¹⁹ *Ann. de chem. et phys.*, 1820, 15: 49.

²⁰ *Gaz. d. hôp.*, 1852, 25: 14, 38, 50, 86, 94.

not until 1895, when Baumann²¹ of Freiburg discovered that iodine in a firm organic combination was a normal constituent of the thyroid, that the possible importance of iodine in thyroid function was revived. He isolated a substance from the colloid material which he called iodothyron, and which contained iodine amounting to 9.3 per cent of the dry weight. Kendall's brilliant contribution came in 1914.²² He succeeded in isolating a pure crystalline body of definite composition, which he named thyroxine. It gives the same physiologic and therapeutic effects as thyroid extract. Thyroxine is the specific hormone of the thyroid gland; it contains 61.5 per cent of iodine. As little as one milligram of thyroxine given to an adult will cause an increase of 2 per cent in the basal metabolism.

The finishing touches were put on by Marine and Williams and Marine and Lenhart, who demonstrated the practical application of iodine in the prevention of goiter. The convincing proof was the demonstration made by Marine and Kimball²³ in the schools of Akron, Ohio, in 1917. McCarrison,²⁴ working in India, showed conclusively that thyroid enlargement is readily produced by the use of diets deficient in iodine, and also by diets containing an excess of fat or protein; conversely, that iodine in minute doses will prevent and cure goiter.

It takes very little iodine to keep the gland saturated, as a normal healthy human thyroid contains only 30 milligrams of iodine, hardly one-half grain. The amount shows variation, but usually there are one to two milligrams per gram of gland. This may be increased by several hundred per cent in animals by feeding iodine. Seidell and Fenger²⁵ found that the iodine content of the thyroid shows seasonal variation, the maximum occurring in summer and early autumn. When the amount falls below 0.1 per cent, according to Marine, enlargement of the gland takes place.

The significance of the work on this subject clearly demonstrates that thyroxine is the internal secretion of the thyroid gland; that it is the specific hormone distributed by the blood to the other glands and structures of the body; and that its chief function seems to be to regulate the rate of metabolism.

Prevention.—*Administration of Iodine.*—Simple goiter is the easiest and cheapest of diseases to prevent, and its control may be accomplished by available methods as soon as organized society determines to make the effort. Various methods of administering iodine have been recommended and used. It is efficacious when given by inhalation, by external application or by the mouth. The essentials of goiter prophylaxis are low cost, palatability, ease of administration, minute dosage, harmlessness and efficiency of the iodine preparation used. The method most favored at the present time is the use of a chocolate tablet containing 10 milligrams of iodine in the form of an organic

²¹ *Ztschr. f. physiol. Chem.*, 1896, 21: 319.

²² *Collected Papers of the Mayo Clinic*, 1916, 513.

²³ *J. Lab. & Clin. M.*, 1917, 3: 40.

²⁴ *The Thyroid Gland in Health and Disease*, William Wood & Co., N. Y., 1917.

²⁵ *J. Biol. Chem.*, 1913, 13: 517.

acid.²⁶ This form of iodine has the advantage of being tasteless, non-hydroscopic and very stable. It is so much more pleasant and practical than sodium iodide that Kimball recommends it.

One or two of these tablets, according to the age and requirements, should be given each week during the school year to boys and girls, especially between the ages of eleven and sixteen. Owing to the prevalence of goiter in younger children, it is profitable to begin the prophylaxis with iodine at a much earlier period. The iodine should be given to children in whom there is no evidence of thyroid enlargement, as well as to those showing enlargement. In the latter instance, however, the exercise of nominal medical supervision is desirable. Goiter prevention for women during pregnancy is enormously important, for the benefit of both the mother and the child. This should always be given under the direction of a physician.

Iodized salt is meeting with favor, and holds forth considerable promise as an efficient means of preventing endemic goiter in a wholesale manner. However, the difficulties of gauging the dose and of excluding from treatment the hypersusceptible are manifold handicaps to its uncontrolled use. Bayard²⁷ has worked out that the addition of 20 milligrams of potassium iodide (15 milligrams iodine) to each 5 kilograms of table salt, which is about a year's consumption, averaging 13.7 grams per day, is ample to prevent the development of goiter. Klinger,²⁸ in Switzerland, prevented and cured simple goiter by giving each week for part of the year 3, 4 or 5 milligrams of iodine as sodium iodide. Iodide added to the municipal water supply, as at Rochester, is an ingenious method that needs further study before its limitations and advantages can be appraised. Kelp, of which the *Macrocystis pyrifera* is the variety containing the greatest amount of assimilable iodine, is also being used in preventing and treating goiter. The natural tendency is to supply the deficiency with a liberal hand, which may do harm. It is alluring for manufacturers to place iodine in foodstuffs of great variety and description, which is an unfortunate tendency. However, there is almost universal testimony rapidly accumulating that there is little if any danger in iodine prophylaxis when carried out intelligently. The procedure is rational and sound, and the results so striking as to make its extensive application both justifiable and advisable.

The goiter problem is not so simple as it at first seems to be. Goitrous persons, especially adults, should take iodine only under the advice and supervision of a skilled physician. The wholesale administration and broadcast distribution of iodine in endemic centers must also take into account the fact that the prevention and especially the treatment of simple goiter is sometimes an individual problem. Some are hypersusceptible, others need more than the average amount, especially at puberty and during pregnancy. Finally, the marvelous effects of iodine must not blind us to the fact that

²⁶ U. S. Pub. Health Rep., 1924, 39: 44.

²⁷ Schweiz. med. Wchnschr., 1923, 53: 703.

²⁸ Schweiz. med. Wchnschr., 1921, 51: 12.

there are accessory factors which predispose to and aggravate goiter, and therefore attention must be paid to hygiene and sanitation. It is important to avoid undue demand upon the physiologic function of the thyroid in those who are disposed to enlargement of the gland.

LEAD POISONING

Lead Poisoning.—Lead is practically never found in natural waters. The source of the lead in the water is almost always lead service pipes, or some other lead object used in collecting, storing, or delivering the water. Lead is the most dangerous inorganic substance with which our drinking water is ordinarily contaminated. Lead poisoning from this source is much more common than it is given credit for. A celebrated instance of lead poisoning occurred in Lancashire and Yorkshire, England. The water came from peaty moorlands and was delivered through lead pipes. The citizens of these towns experienced a mysterious bodily derangement for some years, until it was finally discovered that lead poisoning was evident. In many other places, as Somerfeld, Germany, and Lowell, Massachusetts, numerous cases of lead poisoning due to the action of water in lead pipes have been reported.

Enormous quantities of lead service pipes are still in use, not only in the old plumbing, but in the newer installations. It is so pliable that plumbers find it much easier to bend it around corners and angles than to make the usual connections with iron or brass pipe, and it is therefore a great temptation to put in short lengths of it in difficult places. Lead poisoning may, under certain circumstances, come from a few feet of lead pipe. The various factors that determine the corrosive action of water upon lead are very complex. It is not possible to determine in advance whether or not a water will have serious plumbisolvant action. All natural waters have some solvent power. The only sure method of determining to what degree a given water will take up lead is by testing the question under practical conditions.

The way by which water takes up lead is first through the formation of lead oxid. This oxidation is favored by the amount of oxygen carried in the water, possibly aided by the nitrates and nitrites serving as oxygen carriers. The lead oxid may then be dissolved, more rapidly if the water is acid, or may be washed away by the currents in the state of a fine powder in suspension.

As a general rule clean (pure) waters have a greater corrosive action upon lead than turbid waters. This is partly for the reason that the mud coats the pipes and protects them mechanically. Acid waters are almost sure to take up lead if allowed to come in contact with that metal. Even so feeble an acid as carbonic acid may under certain circumstances greatly increase the plumbisolvant action of water. Soda water (highly charged with carbon dioxid under pressure) takes up relatively large quantities, if lead pipes are used in soda water fountains or "syphon" bottles. Waters

containing carbonates or sulphates are not apt to take up lead because the corresponding salts of lead are insoluble, and thus form a protecting coating. Even though a water has no plumbisolvant action, the use of lead piping, lead cooking utensils, lead-lined cisterns, etc., is entirely unjustified for domestic service, for the reason that under certain circumstances electrolytic action, changes in the character of the water, or other causes may lead to lead poisoning.

Various conditions affect the plumbisolvant action of water, such as the duration of contact, the temperature, the pressure, the season of the year, the purity of the lead, etc. Water remaining in the pipes all night naturally takes up more lead than the water that flows more or less rapidly during the day. Lead pipes were formerly used in soda water fountains and the employee who took the first drink in the morning before the proprietor arrived received a concentrated dose. Hot water has a greater solvent action than cold water; so, also, increase in pressure up to 140 pounds to the square inch. For some unexplained reason more lead is often found in the water during the winter than during the summer. New pipes give up more lead than old pipes. However, in some cases the poisoning manifests itself only after the pipe has been in use for years. Lead pipes are purer now than formerly, owing to profitable methods of extracting the silver and other metals with which it is frequently associated. If the lead is combined with copper the lead passes into the water more quickly in consequence of galvanic action than when pure lead is used. Electrolytic action favors the solution of lead, and the modern method of grounding electric currents adds to the danger.

The various conditions of water that favor plumbisolvant action are: those containing mineral acids, or free carbonic acid, such as soft, peaty waters; those containing much oxygen and little dissolved salts, that is, soft waters, such as rain water; those containing organic matter, nitrites, and nitrates, that is, sewage-contaminated water in the stage of oxidation. Waters that act least upon lead are turbid waters and hard waters, especially those containing free carbon dioxid, for here again carbonates are formed which protect the lead with an insoluble film. However, if carbon dioxid is present in excess or under pressure the carbonates are redissolved.

It will therefore be seen that the purest, softest, and best aerated waters are especially prone to act upon lead. Distilled water will take up lead even from impure zinc pipes (containing some lead) used on board ships. The plumbisolvant action is in part a mechanical erosion, in part a chemical solution, and in part results from electrolytic action.

Illustrative Instances.—Lead poisoning may occur when a comparatively small surface of lead is exposed to the solvent action of the water. This is well illustrated in the following cases: ^{29, 30}

Case 1.—A man about fifty years old contracted lead poisoning from using cistern water. Twelve feet of the service pipe was lead, and almost wholly

²⁹ *Bull. State Board of Health, Maine*, Jan., 1909, Vol. 1, No. 21.

³⁰ *Mass. State Board of Health Ann. Report*, 1898, p. 32.

in the water, as it was bent at right angles and ran across the cistern under the water.

Case 2.—Mrs. W., sixty-six years of age, contracted lead poisoning from a well water which was contaminated from an old lead clock weight which had been accidentally dropped into the well. The clock weight had been in the water about fourteen months before the appearance of symptoms. The well was pumped free of water and the clock weight found and removed. In two weeks from this time Mrs. W. noticed an improvement in her lameness, and in four months she was entirely well.

Case 3.—In this case the patient was poisoned by cistern water pumped through ten feet of lead pipe. The symptoms were acute multiple peripheral neuritis, with extensive paralysis. After the lead in the water was removed recovery was only partial after a period of two years.

Amount of Lead.—The exact amount of lead which may be taken into the system without producing harm is not definitely known. The amount that produces symptoms of poisoning varies with different persons and even in the same person at different times. Individual susceptibility is marked. The continuous use of water containing quantities of lead as small as 0.5 of a part per million, or 0.5 mg. per liter, or about $\frac{1}{33}$ of a grain per gallon, has caused serious injury to health. Private water companies in Massachusetts take notice when the amount of lead is more than 0.5 mg. per liter, although some English supplies contain 1 mg. per liter. The maximum allowable limit should be 0.1 mg. per liter.

No instances have been recorded of ill effects upon health of persons drinking water due to copper or zinc-lined pipes.

The report of the Advisory Committee on Standards for Drinking Water of the U. S. Public Health Service³¹ recommends the following limits for poisonous metals in drinking water: lead 0.1 p. p. m.; copper 0.2 p. p. m.; zinc 5.0 p. p. m.

SPECIFIC DISEASES DUE TO WATER

The principal diseases of man contracted by drinking infected water are typhoid fever, cholera, and dysentery. Water-borne epidemics of these diseases have frequently occurred in the history of the world. It should be remembered that endemic and sporadic cases may also contract their infections through water. The great water-borne tragedies have for a time occupied an exaggerated position. They overshadowed the less dramatic, but more insidious, and nevertheless frequent modes of transmission of infection through other channels. A quantitative estimate of the amount of these diseases spread by means other than water has been realized only in recent years, and since these infections have been eliminated from the water supplies of most large communities, epidemics of typhoid, cholera, and dysentery usually occur independent of water-borne infections.

³¹ U. S. Pub. Health Rep., 1925, 40, No. 15.

It is worthy of note that almost all the large water-borne outbreaks that have been investigated have been traced to a quick transfer of the infected material from the patient to the victim. Even in Pittsburgh the Typhoid Fever Commission showed that most of the fever there had been due to nearby rather than to remote pollution of the river. The greater the distance and the longer the time between the source of the infection and the use of the water, the less are the chances of harm because of nature's purifying agencies.

Typhoid, cholera, and dysentery bacilli are not known to multiply in water under natural conditions. Almost all the great water-borne epidemics of typhoid fever occur in the spring, winter, or fall of the year, when the water is very cold. Water-borne epidemics of typhoid in the summertime, when the conditions seem favorable for multiplication of the bacilli, are relatively infrequent. The dilution must have been enormous in many of the cases recorded; that is, there must have been very few typhoid bacilli in a tumblerful of water. This illustrates how very few bacteria, when fresh and virulent, may induce disease.

Many large epidemics have been traced to individual instances of pollution. The typhoid epidemics at Butler, Plymouth, New Haven, Nanticoke and Reading, involving 3,929 cases with 361 deaths, were caused in each epidemic by the careless treatment of the discharges of one individual patient.

Outbreaks due to water are usually caused by the contamination of surface supplies; less often by wells and springs. The great epidemics have always been caused by polluted river or lake waters, and not by ground waters. Ground water, however, is sometimes responsible for outbreaks of typhoid fever, especially in limestone districts, as at Lausen, Switzerland; Paris, France, etc. Usually when a well becomes badly infected it is from a nearby privy or broken sewer, as in the instance of the Broad Street cholera epidemic in London.

Public water supplies become contaminated in various ways. The use of a raw water into which is continually discharged the sewage of other towns has occurred at Pittsburgh, Lawrence, Niagara Falls, Albany, and Philadelphia. A city may drink the water of a lake which has become its own cesspool, as did Chicago, Cleveland, and Burlington. The pollution may come from the wastes of individual houses, as at Plymouth, or from institutions or factories; or the pollution may come from privies situated directly over the stream or on its banks, as at Ithaca; or the pollution may come indirectly after the offending matter has been deposited on the surface of the ground, later gaining access to the water course by the washing of rain or seepage through ground seams. In some instances epidemics originate through carelessness in a town that has been supplied with a pure or purified water. Thus a water pipe laid through a polluted pond may become sufficiently disjointed to permit admission of the infected water, as occurred at Baraboo, Wisconsin, and Palmerton, Pennsylvania. The admission of pol-

luted water to a pure city supply at any time is inexcusable. There is a record of twenty epidemics due to mixing in cities having dual water supplies. Epidemics have originated as a result of the unusual drain upon the water supply at times of fire, as in the case of Lawrence; or through failure of valves to operate, as in the case of Wilksburg, Pennsylvania; when the ordinary water supply was judged to be insufficient and no public warning was given of the substitution as at Newburyport; or when polluted water was furnished temporarily while the filter plant was undergoing repair, as at Lawrence, Massachusetts, in 1902, in Brewer, in Poughkeepsie, New York, and Millinocket, Maine. Various public wells have become infected through ground seams, and have thus caused epidemics of typhoid fever at Trenton, Newport, and Mt. Savage, Maryland. In recent years outbreaks have resulted from the failure of chlorination plants, or other processes used to purify a polluted supply.

In addition to the usual sources of pollution of a surface water, the following, while relatively infrequent, may be particularly dangerous, for the reason that they are apt to take place near the source of supply: discharges from water-closets of railroad trains while crossing bridges or passing the banks of reservoirs and streams; picnic parties; camping parties; construction gangs; fishermen; ferryboats and other craft upon navigable streams. The large boats plying our Great Lakes may discharge dangerous and obnoxious material very near an intake.

Water is suspected of conveying infections other than typhoid fever, cholera and dysentery. Water-borne outbreaks of infectious jaundice due to spirochetes have been described. Water fouled with manure contains protozoa which may endanger health.

The following illustrative water-borne outbreaks are given because they are instructive and because they are also classic in the historical annals of sanitation.

CHOLERA

Cholera in London in 1854; the Case of the Broad Street Pump.—

Cholera was prevalent in London in 1854, but prevailed with epidemic intensity in the district about Broad Street. This focus was conspicuously circumscribed in area, and the disease was virulent, with great fatality. This case has become classic because it was one of the earliest instances, if not the first, in which water was proved to convey a specific disease. The circumstances were studied by John Snow and by John York, Secretary and Surveyor of the Cholera Inquiry Committee.³² No less than 700 deaths occurred in St. James Parish during the seventeen weeks that the cholera raged. The death rate was 220 per 10,000 in the parish, which contained a population in 1851 of 36,406. In the adjoining districts the death rate varied from 9 to 33 per 10,000.

³² The complete original report is entitled *Report on the Cholera Outbreak in the Parish of St. James, Westminster, during the Autumn of 1854. Presented to the Vestry by the Cholera Inquiry Committee, July, 1855.* London, J. Churchill, 1855.

Snow made a careful epidemiological study of the outbreak and compiled a statistical statement of special value, which is given in its original form:

THE BROAD STREET (LONDON) WELL AND DEATHS FROM ASIATIC CHOLERA NEAR IT
IN 1854.

Date	Number of Fatal Attacks	Deaths	Date	Number of Fatal Attacks	Deaths
Aug. 19.....	1	1	Sept. 11.....	5	15
Aug. 20.....	1	0	Sept. 12.....	1	6
Aug. 21.....	1	2	Sept. 13.....	3	13
Aug. 22.....	0	0	Sept. 14.....	0	6
Aug. 23.....	1	0	Sept. 15.....	1	8
Aug. 24.....	1	2	Sept. 16.....	4	6
Aug. 25.....	0	0	Sept. 17.....	2	5
Aug. 26.....	1	0	Sept. 18.....	3	2
Aug. 27.....	1	1	Sept. 19.....	0	3
Aug. 28.....	1	0	Sept. 20.....	0	0
Aug. 29.....	1	1	Sept. 21.....	2	0
Aug. 30.....	8	2	Sept. 22.....	1	2
Aug. 31.....	56	3	Sept. 23.....	1	3
Sept. 1.....	143	70	Sept. 24.....	1	0
Sept. 2.....	116	127	Sept. 25.....	1	0
Sept. 3.....	54	76	Sept. 26.....	1	2
Sept. 4.....	46	71	Sept. 27.....	1	0
Sept. 5.....	36	45	Sept. 28.....	0	2
Sept. 6.....	20	37	Sept. 29.....	0	0
Sept. 7.....	28	32	Sept. 30.....	0	0
Sept. 8.....	12	30	Date unknown.....	45	0
Sept. 9.....	11	24			
Sept. 10.....	5	18	Total	616	616

Many of the facts of this epidemic are taken from Sedgwick's excellent account in his *Principles of Sanitary Science and the Public Health*, 1902, which the student is advised to read.

It will be seen that the disease broke out with special intensity upon August 30 and declined noticeably after September 10. The pump had been removed on September 8. Snow's inquiry showed that most of the victims had preferred or had access to the water of the Broad Street well, and in a few cases only was it impossible to trace any connection with that source. Thus, with regard to 73 deaths occurring in the locality of the pump and studied especially with reference to this point, it was found that there were 61 instances in which the deceased persons used to drink the water from the pump in Broad Street, either constantly or occasionally. In 6 instances no information could be obtained, and in 6 cases it was stated that the deceased persons did not drink the pump water before their illness.

On the other hand, Snow discovered that, while a workhouse (almshouse) in Poland Street was three-fourths surrounded by houses in which cholera deaths occurred, out of 535 inmates of the workhouse only 5 cholera deaths occurred. The workhouse, however, had a well of its own in addition to the city supply, and never sent for water to the Broad Street pump. If the

cholera mortality in the workhouse had been equal to that in its immediate vicinity it should have had 50 deaths.

A brewery in Broad Street employing 70 workmen was entirely exempt, but, having a well of its own, and allowances of malt liquor having been customarily made to the employees, it appeared likely that the proprietor was right in his belief that resort was never had to the Broad Street well.

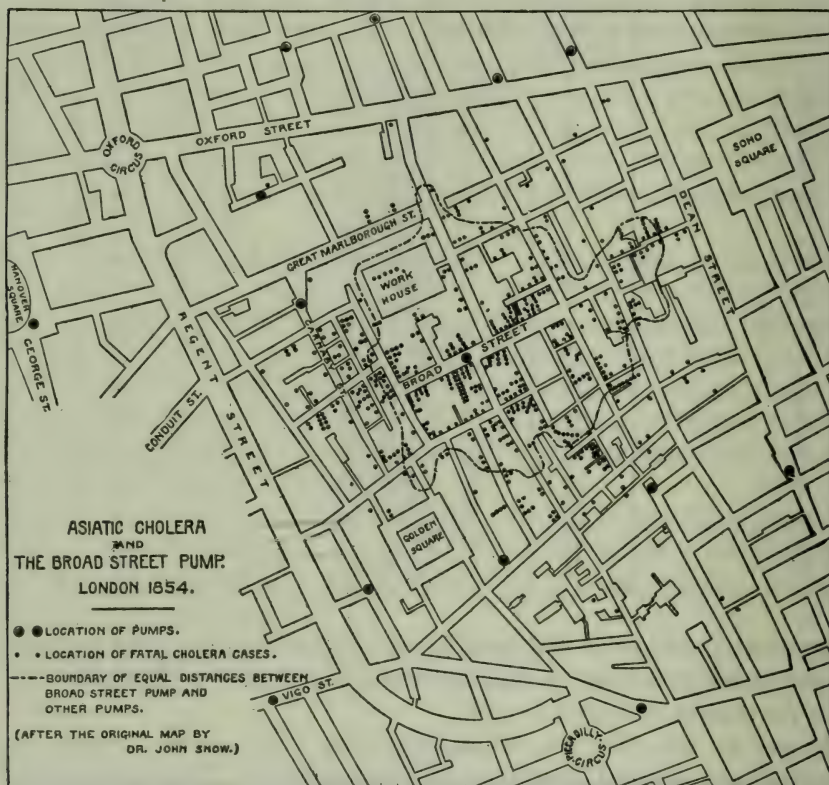


FIG. 95.—ASIATIC CHOLERA AND THE BROAD STREET PUMP.

It was quite otherwise in a cartridge factory at No. 38 Broad Street, where about 200 workpeople were employed, two tubs of drinking water having been kept on the premises and always filled from the Broad Street well. Among these employees 18 died of cholera. Similar facts were elicited for other factories on the same street, all tending to show that in general those who drank the water from the Broad Street well suffered either from cholera or diarrhea, while those who did not drink that water escaped. The whole chain of evidence was made absolutely conclusive by several remarkable and striking cases in Snow's report like the following:

"A gentleman in delicate health was sent for from Brighton to see his brother at No. 6 Poland Street who was attacked with cholera and died in twelve hours, on the first of September. The gentleman arrived after his

brother's death, and did not see the body. He only stayed about twenty minutes in the house, where he took a hasty and scanty luncheon of rump steak, taking with it a small tumbler of cold brandy and water, the water being from the Broad Street pump. He went to Pentonville, and was attacked with cholera on the evening of the following day, and died the next evening.

"The deaths of Mrs. E. and her niece, who drank the water from Broad Street at the West End, Hampstead, deserve especially to be noticed. I was informed by Mrs. E.'s son that his mother had not been in the neighborhood of Broad Street for many months. A cart went from Broad Street to West End every day, and it was the custom to take out a large bottle of the water from the pump in Broad Street, as she preferred it. The water was taken out on Thursday, the 31st of August, and she drank of it in the evening and also on Friday. She was seized with cholera on the evening of the latter day, and died on Saturday. A niece who was on a visit to this lady also drank of the water. She returned to her residence, a high and healthy part of Islington, was attacked with cholera, and died also. There was no cholera at this time, either at West End or in the neighborhood where the niece died. Besides these two persons only one servant partook of the water at West End, Hampstead, and she did not suffer, or, at least, not severely. She had diarrhea."

John York, Secretary and Surveyor of the Cholera Inquiry Committee, was instructed to survey the locality and examine the well, cesspool, and drains at No. 40 Broad Street. His report revealed the following condition of affairs: The well was circular in section, 28 feet 10 inches deep, 6 feet in diameter, lined with brick, and when examined (April, 1855) contained 7 feet 6 inches of water. It was arched in at the top, dome fashion, and tightly closed at a level 3 feet 6 inches below the street by a cover occupying the crest of the dome. The bottom of the main drain of the house from No. 40 Broad Street lay 9 feet 2 inches above the water level, and one of its sides was distant from the brick lining of the well only 2 feet 8 inches. This was an old-fashioned drain 12 inches wide, with brick sides; the top and bottom were

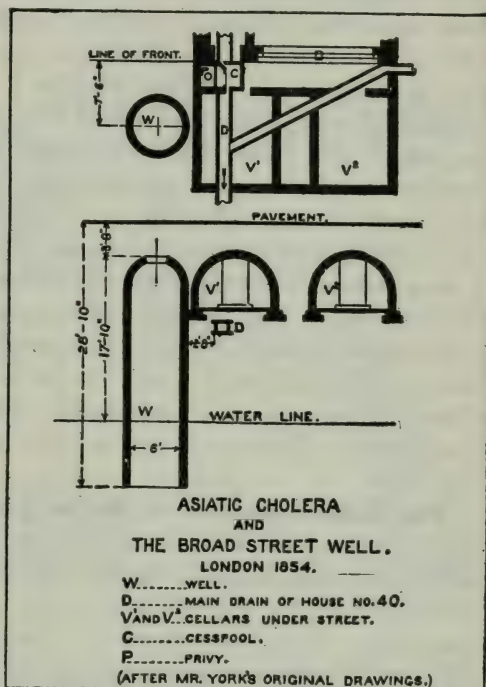


FIG. 96.—ASIATIC CHOLERA AND THE BROAD STREET WELL.

made with old stone. It had a small fall to the main sewer. The mortar joints of the old stone bottom were found to be perished, as was also the jointing of the brick sides, which had brought the brickwork into the condition of a sieve, through which the house drainage must have percolated for a considerable period. Snow found the cesspool intended for a trap, but misconstructed, and upon and over a part of the cesspool a common privy, without water supply, for the use of the house had been erected. The brickwork of the cesspool was found to be in the same decayed condition as the drain. Snow states that, "from the charged condition of the cesspool, the defective state of its brickwork, and also that of the drain, no doubt remains upon my mind that constant percolation, and for a considerable period, had been conveying fluid matter from the drains into the well. A washed appearance of the ground and gravel flow corroborated this assumption. The ground between the cesspool and the well was black, saturated, and in a swampy condition, clearly demonstrating the fact." This evidence, while only circumstantial, is sufficient to connect the cesspool with the well, and can leave no doubt in the minds of those who study this interesting and instructive instance that the water became infected with cholera germs through this channel. It should be remembered that this outbreak occurred before the days of bacteriology, so that direct proof is not at hand. As far as could be determined, the infection of the well came from an unrecognized case of cholera in the house at No. 40 Broad Street. There were four severer cases of cholera subsequently in the same house.

The Cholera Epidemic in Hamburg in 1892.—This epidemic stands out clearly, not only as the most devastating of its kind, but as one of the most instructive. The relation between the infected water and the disease was conclusively proved, and the value of slow sand filtration placed upon a strong foundation. The conditions of the epidemic were equal to those of a well-controlled laboratory experiment, and the bacteriological and epidemiological evidence corroborated each other in every essential particular.

From August 17 to October 23, 1892, a little over two months, there were nearly 17,000 cases of cholera in Hamburg (population 640,000), with 8,605 deaths. On one day during the height of the epidemic over 1,000 new cases occurred. This was a pandemic year for cholera in the sense that it showed a remarkable tendency to spread to all parts of the world. It traveled from the valley of the Ganges through Persia, to Russia, Germany, Austria, France, Belgium, Holland, and the disease was brought to our own doors and several cases occurred in New York City.

The epidemic involved Hamburg, Altona and Wandsbeck. Hamburg and Altona are adjacent but separate cities. Hamburg, being an old Hanseatic city, has its own government. Altona, however, is in Prussia. Wandsbeck (population 20,000) is a nearby suburban town. Each of these three places at the time of the epidemic had a separate water system. Wandsbeck drank from an independent supply taken from a small lake. Hamburg and Altona were both furnished with water from the Elbe River, which is a grossly pol-

luted stream. Both the cities of Hamburg and Altona rest upon the bank of the Elbe River, but Altona is below or downstream. At the time of the epidemic the intake for the water supply of each city was directly at the river front, and the sewers of the city emptied into the river at various points along the same river fronts. It will therefore be seen that Altona had Elbe River water plus Hamburg's sewage. Altona, however, filtered this water by the slow sand process; Hamburg furnished its citizens with the raw, unfiltered Elbe River water. This water was first pumped to a single reservoir, which

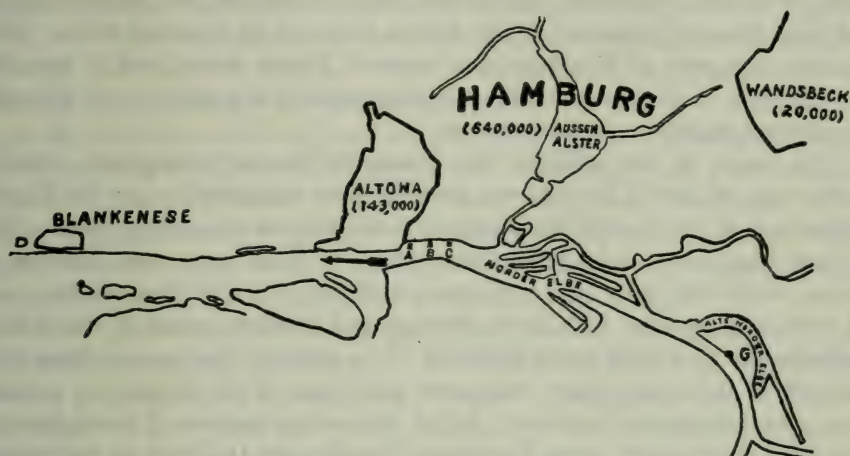


FIG. 97.—WATER SUPPLY OF HAMBURG.

Hamburg received its water supply from the River Elbe (unfiltered) at G. The sewerage of Hamburg entered the Elbe at ABC. Altona received its water supply from the Elbe at D, about 8 miles below ABC. The sand filters which purified this water were located at Blankenese. Wandsbeck had an independent water supply from a small lake.

at one time held approximately a day's supply, but had long become inadequate. It will therefore be seen that these three cities, with a homogeneous population, with the same climate, the same low-lying site, and all other conditions similar, differed only in their water supplies.

Relatively few cases occurred in Altona, and most of these were on the boundary, where the people probably had access to Hamburg's raw, unfiltered Elbe River water. In Koch's own words, "cholera in Hamburg went right up to the boundary of Altona and there stopped. In one street, which for a long way forms the boundary, there was cholera on the Hamburg side, whereas the Altona side was free from it."

During the epidemic the deaths in the several cities were as follows:

City	Population	Deaths	Deaths per 10,000 Inhabitants
Hamburg	640,400	8,605	134.4
Altona	143,000	328	23.0
Wandsbeck	20,000	43	22.0

Further evidence consisted in the fact that at one point close to and on the Hamburg side of the boundary line between Hamburg and Altona is a large yard known as the Hamburger Platz. It contains two rows of large and lofty dwellings containing seventy-two separate tenements and some 400 people belonging almost wholly to those classes who suffered most from cholera elsewhere in Hamburg. While cholera prevailed all around no single case occurred among the many residents of this court during the whole epidemic. Koch found that, owing to local difficulties, water from the Hamburg mains could not easily be obtained for the dwellings in question, and hence a supply had been obtained from one of the Altona mains in an adjacent street. This was the only part of Hamburg that received Altona water, and it was also the only spot in Hamburg in which was aggregated a population of the class in question which escaped the cholera.

The source of the epidemic was traced to Russian immigrants crowded in barracks on one of the wharves pending their embarkation for the United States, and at the time of the outbreak there were on an average about 1,000 of these people on hand all the time. Many of them came from districts in Russia which had been, and were then, suffering severely from cholera, and all were well supplied with dirty clothing and blankets, some of which they washed while they were being detained. It is believed that among those that had arrived there must have been some mild cases of the disease, or at least some convalescents and carriers. All of the sewage matters of every description from these people were discharged directly into the river at the wharf. After the Elbe River once became seeded with the cholera vibrio the people in Hamburg who drank this infected water took the disease, and their discharges, returning to the river, added fuel to the flames. A vicious circle was thus set up, so that the infection became exceedingly concentrated and intense, and as the circle was a short one the time interval was correspondingly brief and the virulence unusually severe.

The Hamburg outbreak will ever remain classic on account of the clearness of the circumstances and the fact that there is no missing link in the chain of evidence as the specific organism was readily isolated from the Elbe River water. The conditions proved the effectiveness of filtering out cholera, and gave a greater impetus to slow sand filtration, thus illustrating the saying that an epidemic may save more lives than it costs.

TYPHOID FEVER

The Influence of Pure Water upon Typhoid Fever.—The typhoid figures present clear and often dramatic proof of the value of clean water in the conservation of health. The following outbreaks are instructive examples:

The Typhoid Epidemic at Lausen, Switzerland.—The epidemic of typhoid fever which occurred in Lausen, Switzerland, in 1872, was the first to attract general attention, "and, because of certain peculiar conditions connected with it, and especially because of its influence upon the theory and

practice of the purification of water by filtration, it deserves the most careful consideration by all students of sanitation." It is also interesting because of the remoteness and unusual method by which the infection reached the water supply. The following account of this epidemic is from the description by Sedgwick, quoting Hagler's³³ report:

The epidemic occurred in the little village of Lausen in the canton of Basel in Switzerland in August, 1872. Lausen was a well-kept village of 90 houses and 780 inhabitants, and had never, so far as known, suffered from a typhoid epidemic. For many years it had not had even a single case of typhoid fever, and it had escaped cholera even when the surrounding country suffered from it. Suddenly, in August, 1872, an outbreak of typhoid fever occurred, affecting a large part of the entire population.

A short distance south of Lausen is a little valley, the Fürlethal, separated from Lausen by a hill, the Stockhalden, and in this valley, on June 19, upon an isolated farm, a peasant, who had recently been away from home, fell ill with a very severe case of typhoid fever, which he had apparently contracted during his absence. In the next two months there occurred three other cases in the neighborhood—a girl, and the wife and son of the peasant.

No one in Lausen knew anything of these cases in the remote and lonely valley, when suddenly, on August 7, 10 cases of typhoid fever appeared in Lausen, and by the end of nine days 57 cases. The number rose in the first four weeks to more than 100, and by the end of the epidemic in October to about 130, or 17 per cent of the population. Besides these, fourteen children who had spent their summer vacation in Lausen fell ill with the same disease in Basel. The fever was distributed quite evenly throughout the town, with the exception of certain houses which derived their water from their own wells and not from the public water supply. Attention was thus fixed upon the latter, which was obtained from a well at the foot of the Stockhalden hill on the Lausen side. The well was walled up, covered, and apparently protected, and from it the water was conducted to the village, where it was distributed by several public fountains. Only six houses used their own wells, and in these six there was not a single case of typhoid fever, while in almost all the other houses of the village, which depended upon the public water supply, cases of the disease existed. Suspicion was thus directed to the water supply as the source of the typhoid, very largely because no other source could well be imagined.

There had long been a belief that the Lausen well or spring was fed by and had a subterranean connection with a brook (the Fürler brook) in the neighboring Fürler valley; and since this brook ran near the peasant's house and was known to have been freely polluted by the excreta of the typhoid fever patients, absolute proofs of the connection between the well of Lausen and the Fürler brook could not fail to be highly suggestive and important. Fortunately, such proofs were not far to seek. Some ten years before observa-

³³ *Sixth Report, Rivers Pollution Commission of 1868, London, 1874.*

tions had been made which had shown an intimate connection between the brook and the well. At that time, without any known reason, there had suddenly appeared near the brook in the Fürler valley below the hamlet a hole about eight feet deep and three feet in diameter, at the bottom of which a considerable quantity of clear water was flowing. As an experiment the water of the little Fürler brook was at that time turned into this hole, with the result that it had all flowed away underground and disappeared, and an hour or two later the public fountains at Lausen, which, on account of the dry weather prevailing at the time, were not running, had begun flowing abundantly. The water from them, which was at first turbid, later became clear; and it had continued to flow freely until the Fürler brook was returned to its original bed and the hole had been filled up. But every year afterward, whenever the meadows below the site of the hole were irrigated, or overflowed, by the waters of the brook, the Lausen fountains soon began to flow more freely. In the epidemic year (1872) the meadows had been overflowed as usual from the middle to the end of July, which was the very time when the brook had been infected by the excrements of the typhoid patients. The water supply of Lausen had increased as usual, had been turbid at the beginning, and had had a disagreeable taste. And about three weeks before the beginning of the irrigation of the Fürler meadows typhoid fever had broken out, suddenly and violently, in Lausen.

In order to make matters, if possible, more certain the following experiments were made, but unfortunately not until the end of August when the water of the Lausen supply had again become clear. The hole which had appeared ten years earlier, and had afterward been filled up, was reopened, and the little brook was once more led into it; three hours later the Lausen fountains were yielding double their usual volume. A quantity of brine containing about eighteen hundred pounds of common salt was now poured into the brook as it entered the hole, whereupon there appeared very soon in the Lausen water first a small, later a considerable, and finally a very strong reaction for chlorin, while the total solids increased to an amount three times as great as before the brine was added. In another experiment five thousand pounds of flour (Mehl), finely ground, were likewise added to the brook as it disappeared in the hole; but this time there was no increase of the total solids, nor were any starch grains detected in the Lausen water.

It was naturally concluded from these experiments that while the water of the brook undoubtedly passed through to Lausen and carried with it salts in solution, it nevertheless underwent a filtration which forbade the passage of suspended matters as large as starch grains. Hagler, from whose report the foregoing facts are taken, was careful, however, to state that "it is not denied that small organized particles, such as typhoid fever germs, may nevertheless have been able to find a passage." As a matter of fact Hagler's minute account does to-day give us some indication that such germs might easily have passed from the brook to Lausen, for the turbidity of which

he repeatedly speaks is evidence of the passage of particles as small as, and possibly smaller than, the germs of typhoid fever.³⁴

Unfortunately this was before pure cultures of bacteria were known, and no experiments were made with suspended matters as small as bacteria. The conclusion was inevitable that although filtration had in this case sufficed to remove starch grains, it had been powerless to remove the germs of typhoid fever; and, accordingly, filtration as a safeguard against disease in drinking water fell for a time into disrepute.³⁵

The Typhoid Epidemic in Plymouth, Pennsylvania.—In 1885 the mining town of Plymouth, Pennsylvania, with a population of about 8,000, suffered from a severe outbreak of typhoid fever which involved one in every eight of the inhabitants. Plymouth received its water from a mountain brook which drained an almost uninhabited watershed. The stream was dammed at intervals and the water was stored in a series of four small impounding reservoirs. The source of the infection was traced to a citizen who spent his Christmas holidays in Philadelphia and returned home in January. He contracted typhoid; the excreta were not disinfected, but were thrown either into the frozen creek or upon its banks within 25 or 30 feet of the edge of the stream (see map). At this time the brook was frozen and remained so until spring. There came a thaw in March and the entire accumulation was washed into the brook and thence into the water-main. Three weeks thereafter cases of typhoid by the score made their appearance throughout the town. On some days more than 100 new cases occurred. In all, 1,004 cases were reported. Some estimates placed the number at 1,500, that is, 1 in every 5 of the inhabitants. There were 114 deaths. The epidemic was limited to the houses supplied with the town water or to persons who

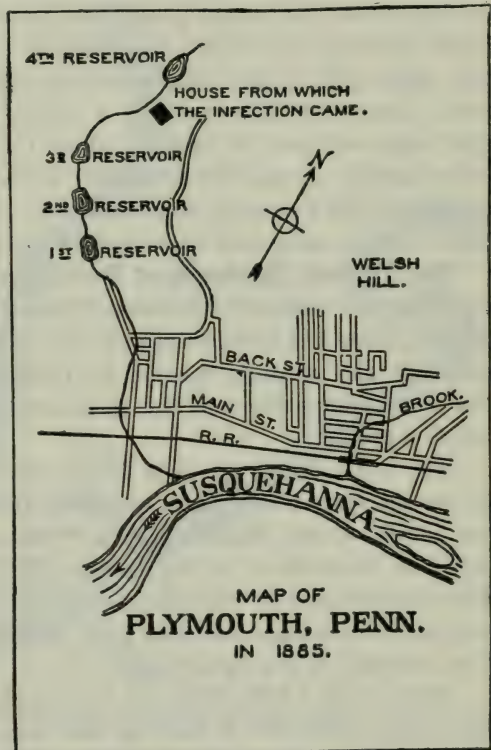


FIG. 98.—TYPHOID EPIDEMIC IN PLYMOUTH, PENNSYLVANIA.

³⁴ "Typhus und Trinkwasser," *Vierteljahresschrift für öffentliche Gesundheitspflege*, VI, 154; also *Sixth Report, Rivers Pollution Commission of 1868*, London, 1874.

³⁵ Sedgwick, *Journal New England Water Works Association*, 1901, 15, No. 4, 330.

drank of the public water supply. The distinction was particularly emphasized on one street where the houses on one side had one or more cases while the houses on the other side had none at all. The former were supplied by the town water, the latter depended upon wells.

This epidemic will ever stand out in the literature as a clear-cut instance of water-borne typhoid caused by the quick transfer of virulent material from a single case. It proves further that freezing alone was not sufficient to destroy the typhoid infection, and on account of the coldness of the water it is not possible that any multiplication of the typhoid bacilli occurred. The infection, although greatly diluted, was nevertheless sufficiently virulent to induce the disease in many of those who drank the water. It further teaches the lesson how one person is sufficient to defile the "pure waters of a mountain brook draining an almost uninhabited territory." Laboratory analysis of this water prior to the thaw would not have disclosed danger, but the hazard would have been discovered by a sanitary survey. This epidemic was the first large outbreak in America where the cause was definitely traced to the water supply. It stands out sharply in the sanitary annals of our country on account of the lessons it taught and the good influence it had in stimulating other cities to safeguard and improve their water supplies.

The Typhoid Epidemic at New Haven.—Very similar to the Plymouth outbreak was that at New Haven, Connecticut, during April, May, and June of 1901, when 514 cases of typhoid fever occurred, resulting in 73 deaths. The outbreak was carefully studied by Professor Herbert E. Smith, who found that it was unquestionably due to an infection of one of the sources of public water supply.

The water supply in New Haven was drawn from five distinct systems. It was all surface water and was used without filtration. One of the sources was known as the Dawson supply. Dawson Lake was a storage reservoir located on West River in Woodbridge, five miles from New Haven. Dawson Lake had an area of 60 acres and a capacity of 300,000,000 gallons. There was no direct sewage pollution upon the catchment area and the rural population was only 25 per square mile.

A mile and a half above the Dawson Lake a small stream flowed into the river, and about half a mile up this stream there was a farmhouse situated at an elevation of about 180 feet above the water in the lake. Several cases occurred in this house during January and February, 1901. The excreta were thrown into a shallow privy vault without disinfection (for the reason that typhoid fever was not at first recognized). Here they accumulated and remained more or less frozen for six weeks or more. This privy was 325 feet from the brook and 40 feet above it. On March 10 and 11 there was a heavy rainfall (2.46 inches) and a sudden thaw. The flow was so large that in spite of the intervention of the storage reservoir the water in the city was in a turbid condition on the afternoon of March 11. The typhoid fever outbreak began about 10 days later, and there seems to be little doubt that infection took place at this time. Professor Smith found that 96 per cent of the cases

that occurred were in the districts supplied with water from the Dawson Lake (Whipple).

This outbreak again illustrates the resistance of the typhoid infection to freezing, and the danger from a surface supply that for years may run satisfactorily. Even the storage reservoir failed in this case, as in the Plymouth case, to check the quick transfer of the infection. Had the Dawson supply been protected, filtered or otherwise purified the epidemic could have been averted.

The Typhoid Epidemic at Ashland, Wisconsin.—This outbreak is cited from Harrington and is one of peculiar interest, in that, in addition to serving as an excellent illustration of the danger of using the same body of water as a place for the disposal of sewage and as a source of drinking water, it was made the basis of an action at law, which established the liability of water companies and municipalities in case of sickness and death caused by the distribution and use of infected water.

The city's supply is derived from an arm of Lake Superior, Chequamegon Bay, upon which the city is situated. This bay, which is about twelve miles long, and has an average width of five miles, varies from eight to thirty-six feet in depth. North of the city, and



FIG. 99.—TYPHOID EPIDEMIC AT ASHLAND, WISCONSIN.

extending outward in a northwesterly direction, is a breakwater constructed for the protection of the harbor against northerly gales. The mouth of the water intake is located about a mile from the shore between the breakwater and the city (see Fig. 99). The sewage of the city is discharged further to the west and south. The currents in the bay follow the course indicated by the arrows in the figure, and carry the sewage toward the breakwater and over the mouth of the intake. This condition of affairs was brought to the attention of the company by the health boards of the city and state repeatedly, but without results. That the water was polluted was evident on mere ocular inspection, for it was often cloudy or markedly turbid.

During the winter of 1893-94 typhoid fever made its appearance in the city, and from the initial cases a disastrous epidemic developed, which led to the establishment of a model filtering plant.

The action at law referred to above was brought by the widow of one of the victims. In evidence it was shown that he lived continuously in Ashland, and drank no water other than that supplied by the water company; that previous to his sickness the disease had prevailed in the city, and that the discharges from the antecedent cases had passed into the waters of the bay by way of the city sewers. The court found for the plaintiff in the sum of \$5,000.

The Typhoid Epidemic in Mankato, Minnesota.—Mankato (population 11,553) receives its water supply from four deep artesian wells on Washington Street. Two of these wells are within from 16 to 18 feet of the pumping station. The main outlet of the sewer runs down Washington Street, emptying into the river. A great flood occurred May 20 to 24, 1908. The gate in the main trunk of the sewer was let down on the night of June 24, 1908, in order to keep the river from backing up into the sewers. This caused a backing up or stasis of the sewage, which in turn backed up into a well pit of the new artesian well near the pumping station, hence sewage was pumped into the water system. Two of the other wells and suction mains were rusty and leaked and had not been properly looked after for a number of years. Then came a sudden sharp epidemic of diarrhea, June 26. Probably 2,000 persons were affected. It soon developed that the prevailing disease was typhoid fever. The epidemic began June 26 and gradually died out by Nov. 20, 1908. From July 7 to Nov. 20, 464 cases of typhoid fever were reported to the Health Officer. Four hundred and one of these cases were considered primary and 57 secondary or contact cases and 6 outside or imported infection.

This water-borne outbreak of typhoid fever is particularly instructive from the fact that Delia McKeever and Kate Flanagan, administratrices of the estates of their husbands, who had died of the fever, sued the city of Mankato for damages. The city demurred to this complaint on the grounds that as a government it was exempt because it was carrying out a government function. The Supreme Court of Minnesota held that "the state is liable if negligence can be proved." The decision of the Supreme Court in holding the city liable sets an excellent precedent which places the responsibility where it should be. Citizens are evidently as much entitled to reasonable sanitary protection as they are to police protection, or to protection from accidents at grade crossings. It is a fortunate day for preventive medicine when the principle is recognized that sanitary negligence is just as culpable as the negligence which fails to place a red flag or a red lantern to warn against a pitfall in the public highway.

The Typhoid Epidemic in Ithaca, New York.—In the winter of 1903 Ithaca, New York, the seat of Cornell University, was visited by a severe epidemic in the course of which 1,350 cases of typhoid fever occurred in a population of about 13,156. The population included about 3,000 students

at the university. More than 500 homes were visited and there were 82 deaths. The epidemic covered a period of about 3 months and extended from about the 11th of January, 1903, to the 1st of April, although for several months before the epidemic began typhoid fever had been unduly prevalent. The epidemic was carefully studied by George A. Soper, who clearly showed that the disease was due to the public water supply, although the original case or cases which gave rise to the epidemic were not ascertained. Ithaca had at that time three separate sources of water supply. The largest one was derived from Six-Mile Creek and the second supply from Buttermilk Creek, and the third was an independent supply for the university. The conditions on the two streams were similar. Both streams were considerably polluted by the population which lived largely in villages bordering on the streams. The nearest of these villages was five miles above the intake. Soper found numerous other sources of contamination on the watershed, and some even in the city of Ithaca a few rods above the intake of the waterworks where there were no less than 17 privies located on the precipitous banks of the creek. It was known that during the year previous to the epidemic there had been at least 6 cases of typhoid fever on the watershed. The typhoid epidemic in Ithaca followed a flood in the river.

One episode of the epidemic is worthy of special mention, namely, a secondary outbreak which resulted from the infection of a well. This well had become popular among the residents of a certain district at the time when the public supply came to be distrusted, and its good quality was taken for granted. But the wife of the owner was taken sick with typhoid fever during the epidemic, and her dejecta passed without disinfection through the water-closet, and into a drainpipe which ran within three or four feet of the well. The joints of the drainpipe were insecure; and the well water, which had probably been for some time grossly contaminated, finally became infected. As a result about 50 cases of typhoid fever and five deaths were traced to people who used this well water (Whipple).

The Typhoid Epidemic in Butler, Pennsylvania.—Butler, Pennsylvania (population 16,000), had an epidemic of typhoid fever in 1903. There were 1,270 cases; that is, about 8 per cent of the population were attacked. Infection in this case was clearly water-borne and was traced to one of various points of the stream, small tributaries, or creeks. One house in particular, provided with an overhanging privy, emptied into the creek within a short distance of the pumping station.

The Typhoid Epidemics of Lawrence and Lowell.—During the years 1890-91 a typhoid fever epidemic occurred in Lowell and Lawrence, Massachusetts. This epidemic illustrates with great clearness what occurs on streams which are used both as sources of water supply and as receptacles for sewage. Both cities are on the Merrimac River, which was grossly polluted by the sewage of Manchester (population 44,126), Haverhill (population 27,412), Nashua (population 19,311), Concord (population 17,004), Fitchburg (population 22,037), Newburyport (population 13,947), Marlborough

(population 13,805), Clinton (population 10,424), and from other sources of pollution. In Lowell 550 cases of typhoid fever occurred from September, 1890, to January, 1891. The epidemic was carefully studied by William T. Sedgwick, who made a most thorough investigation.

A short time after the epidemic in Lowell typhoid fever broke out in Lawrence, nine miles downstream, and rapidly increased. The relation between these two epidemics was most striking. Lowell discharged its sewage into the river, Lawrence drank the water without filtration. The climax of the Lawrence epidemic occurred about one month after that in Lowell. In 1892 there was a repetition of this episode. Typhoid fever in Lowell was again responsible for an increase of typhoid fever in Lawrence. As a consequence of these occurrences Lowell abandoned the river and introduced a ground water supply, while at Lawrence a filtration plant was constructed which has materially reduced the amount of typhoid fever in that city (Whipple).

The Typhoid Epidemics of Pittsburgh and Allegheny.—These two Pennsylvania cities are situated at the junction of the Allegheny and Monongahela Rivers, where they unite to form the Ohio. In 1900 Pittsburgh had a population of 321,616 and Allegheny 129,896. Pittsburgh took its water from the Allegheny River at Brilliant Station, six miles above the junction of the rivers, and from the Monongahela River at a point three miles above the junction. Allegheny received its water supply from the Allegheny River at Montrose, ten miles from the point; it was drawn from a rock-filled crib, and was practically unfiltered water. Both the Monongahela and the Allegheny Rivers are grossly polluted streams, receiving the sewage from a populous watershed; in addition the sewers of the cities of Allegheny and Pittsburgh empty directly into these streams, and on account of the rapid growth of these cities much of this sewage entered the river dangerously near to the water intakes. The records of the Board of Health show that at this time there occurred annually upward of 5,000 cases of typhoid fever.

For about ten years centering around 1900 Pittsburgh and Allegheny had the unenviable distinction of having the highest typhoid death rate of any city in this country and probably of any large city in the world. At times the rates ran above 150 per 100,000. The conditions were at once improved by the introduction of slow sand filtration for the city of Pittsburgh. Allegheny, which is now officially known as North Pittsburgh, was furnished (1912) with filtered water.

The Typhoid Epidemic at Chicago.—The Chicago epidemic is an illustration of a city using a lake water which is infected with its own sewage. The water in 1892 was taken from Lake Michigan opposite the city at several "cribs" which were 1.5 to 4 miles off-shore. The Chicago sewage was discharged all along the water-front, while the Chicago River penetrated the city with its north and south branches and, polluted almost beyond endurance, flowed out into the lake about midway between the upper and lower cribs. The pollution of the lake water was at times so intense that the foul river

water could be traced to the intakes with the eye. This intolerable situation resulted in the building of the Chicago drainage canal, the object of which was to keep the sewage out of the lake and carry it down the Des Plaines and Illinois Rivers into the Mississippi. By the construction of this canal the flow of the Chicago River was reversed so that, instead of the sewage entering the lake and polluting the water supply, the water of Lake Michigan now flows into the Chicago River and thence, through the drainage canal, into the tributaries of the Mississippi. In other words, the sewage of Chicago, instead of entering Lake Michigan, drains to the Gulf of Mexico. During the years 1890, 1891, and 1892 typhoid fever was unusually prevalent in Chicago. In 1890, 1,008 of the inhabitants died from typhoid fever, in 1891 the death toll from this preventable disease was 997 and in 1892, 1,489. The present conditions in Chicago, owing to the improvements in the water supply, including chlorination, general pasteurization of the milk supply, and an attack upon the residual typhoid as contact infection, have reduced the death rate to 1.6 per 100,000 in 1924.

The above water-borne typhoid fever epidemics have been selected as examples. Many more may be found in the literature. Whipple, in his book, *Typhoid Fever*, cites numerous instances and gives in tabular form an impressive list of such outbreaks, with reference to the literature.

DYSENTERY

Both bacillary dysentery and amebic dysentery may be transferred through drinking water. The microorganisms in both types of dysentery are discharged in the feces and taken in by the mouth; there is, therefore, every opportunity for water to play the same rôle in dysentery that it plays in typhoid. However, comparatively few water-borne epidemics of bacillary dysentery have been reported; these few, nevertheless, are sufficiently conclusive to be convincing. Amebic dysentery does not occur in epidemic form, but the known facts are sufficient to incriminate water as one of the vehicles of convection.

Shiga reports outbreaks in Japan from the use of well and river water. Eldridge states that dysentery is a rural disease in Japan; the use of human feces as a fertilizer and the frequency of the infection of the numerous small streams and wells render it preëminently a water-borne disease. Whittaker³⁰ reports a water-borne epidemic of dysentery at South St. Paul, Minnesota, caused by using infected water from a fire connection. The epidemic described by Duprey which occurred at Grenada Island in 1901 is one of the best examples of a water-borne epidemic of dysentery.

Epidemics of bacillary dysentery in this country in institutions and camps have not, as a rule, been associated with water.

The *Entameba histolytica*, causing amebic dysentery, was recovered by

³⁰ U. S. Pub. Health Rep., 1915, 30: 3473.

Musgrave and Clegg³⁷ from 17 to 61 samples of the public water supply of Manila and was found in tanks used for holding distilled water and also in many wells. Allan³⁸ has reported a small outbreak of amebic dysentery in North Carolina due to an infected well.

Diarrhea.—Polluted waters not infrequently cause diarrhea, sometimes as widespread epidemics, sometimes as small outbreaks or sporadic cases. Whenever there is a water-borne outbreak of typhoid fever or cholera there are also a large number of cases of diarrhea and gastro-intestinal disturbances in which the precise etiological factor has not been discovered. Some of these cases may be mild instances of the major disease. Infantile diarrheas are especially prevalent at such times and very likely due to the contaminated water. Thus Reincke states that infantile diarrhea was greatly lessened after the improvement in the water supply of Hamburg. The same phenomenon was noted by Hiram O. Mills after the filtration of the water supply of Lawrence, Massachusetts. Sedgwick noted an excessive prevalence of both typhoid fever and diarrhea in Burlington and attributed the diarrhea to the sewage contamination of the water supply. Whipple states that in Albany there was a reduction of 57 per cent in the mortality from diarrheal diseases after the introduction of filtration in 1898. Chapin questions whether such statistical evidence is sufficient to incriminate water as an influence to the causation of diarrheal diseases.

Numerous outbreaks of diarrhea have been attributed to the following microorganisms in water, viz.: *Bact. coli*, *B. enteritidis* of Gärtner, *B. pyocyaneus*, *B. proteus*, *B. aerogenes capsulatus* of Welch, *B. mesentericus*, and streptococci. Water containing these and other organisms is not infrequently regarded as the cause of outbreaks of gastro-intestinal irritation. The symptoms vary greatly in intensity, but usually the disease is not fatal excepting in the young and feeble. The relation between the diarrhea and the water is usually based upon the fact that the same species of microorganisms are found both in the water and in the stools. Corroborative evidence, such as the finding of specific agglutinins and other antibodies in the blood, lends countenance to the claim that the particular microorganism is, in fact, the cause of the complaint. While the evidence is not conclusive, it is suggestive, and in many cases doubtless correct.

So-called "winter cholera" is a mild diarrheal disease. It is probably water-borne, but the cause is not known. It occurs in epidemic outbreaks.

ANIMAL PARASITES

The eggs, larvæ, or other stages in the life cycle of various intestinal parasites may enter the body in drinking water. Thus the eggs of *Ascaris lumbricoides* discharged in the feces, which require a month for development, may

³⁷ Musgrave and Clegg, *Bull.* 18, Bur. Gov. Lab., P. I., 93; *Rep. Bd. Health*, P. I., 1904-05, 10.

³⁸ *J. Am. M. Ass.*, 1909, 53: 1561.

contaminate streams and then be returned to the mouth. Some cases of infection with this parasite probably occur in this way. *Oxyuris vermicularis*, the pinworm, and *Trichuris trichiura*, the whipworm, may similarly be contracted through drinking water. The guinea worm, *Dracunculus medinensis*, may be contracted by the mouth, in drinking water. The living embryos of this worm are liberated and find their way into fresh water. There they enter the bodies of small fresh water crustacea, *Cyclops*, which act as intermediate hosts.

It is fairly well established that the larvæ of the hookworm may be taken into the stomach through drinking water, although this is not usual, and the same is assumed of the similar parasite of Cochin China diarrhea.

THE SANITATION OF SWIMMING POOLS

The swimming pool is a good example of an institution devised to improve hygienic conditions, yet one wherein the device itself may be a hygienic menace. Swimming is one of the best and most exhilarating forms of exercise. It is especially suitable for the tropics. Swimming is about the only form of violent exercise that can be practiced in hot weather without danger of overheating the body. Swimming has the added hygienic advantage of promoting cleanliness.

Swimming pools are nothing more nor less than common bath tubs. Their growing popularity has led to an increased interest in the sanitary conditions that prevail in them. There is no longer any doubt that they can and sometimes do transmit disease; therefore, a serious duty of sanitary supervision and responsibility arises.

The diseases contracted in swimming pools are inflammatory infections of the upper respiratory tract and conjunctiva; injury and inflammation of the ears; venereal and skin diseases; intestinal infections, etc. Typhoid fever and diarrheal conditions have been traced on reasonably reliable evidence to swimming pools such as are installed by private individuals, or found in colleges and universities, public and private schools, gymnasiums, steamships, and special bathing establishments.

The chief danger of infection comes from the water, if not kept clean, or from the towels and swimming suits, if not disinfected. The source of the infection comes in almost all instances from the persons using the pool. One of the first essentials, therefore, in the sanitation of swimming pools is to require a shower bath with the liberal use of soap before entering the tank. At the same time stringent regulations should be in force to prevent use of the pool by persons suffering with skin diseases, running ears, ulcers, conjunctivitis, venereal disease, or signs of inflammation of the upper respiratory tract.

Full showers, with soap, under inspection, should be demanded of all bathers *before* entering the pool. Especial attention should be given to the perineal region. When water strikes a person's body, the natural tendency

is to urinate. For this reason ample toilet facilities should be provided, and they should be so located that exit from them is through the showers and not directly to the pool.

Bathers should be instructed in pool sanitation. Nude bathing should be encouraged, because it favors inspection and does away with the danger of contracting infection from the swimming suit.

The water should have an initial purity equal to that of a safe drinking water and should be kept fairly clean by filtration and reasonably safe by disinfection with ozone, ultraviolet rays, chlorin or bleaching powder. The combination of refiltration to clean, and chlorin, ultraviolet rays or ozone to disinfect the water is the best present available method to keep the water of swimming pools in a satisfactory sanitary condition. Occasional use of sulphate of copper may be necessary to keep down excessive growth of algæ.

The bathers are constantly introducing pollution and occasionally infection. To offset this, disinfection should be continuous, at least it should be most effective at the time when the pool is in use. Chlorin or bleaching powder are effective, but they are soon oxidized and their disinfecting power rapidly disappears, hence the need for continuous application. Ultraviolet rays and ozone are effective over a still shorter period of time. If too much chlorin or its compounds are added, the water will smart the eyes and have an unpleasant odor and taste.

The disinfection of the water in the pool is economical, in that it is not necessary to change the water as often as without treatment. Decency and safety require frequent additions of fresh water. If clarification and disinfection are properly conducted, the demands of decency and safety will be met by the daily addition of fresh water in a volume equal to at least 10 per cent of that of the pool. Wherever possible, fresh water should be added continuously.

The examination of swimming-pool water for colon bacilli as an index of pollution is as logical a method of control as it is in the case of drinking water, but standards of quality are not as rigorous for pool waters as for drinking waters.

In localities where blood-flukes, *Schistosoma*, occur, special precautions are necessary with respect to swimming pools. According to Leiper, the cercariæ, which is the infective stage of the parasites and whose penetration through the skin results in infection, are unable to live in water more than thirty-six hours after their escape from the snails which act as intermediate hosts. Consequently, he recommends that water before it is used be stored for not less than thirty-six hours, better forty-eight hours, in reservoirs that are protected from invasion by snails.

The sanitation of swimming pools requires good design and construction and efficient management. The tank should be large. Roughly, at least 400 gallons of water per bather are needed. The pool should have a smooth lining. When empty, the tank should be scrubbed, flushed and dried. The cleanliness of the tank is aided by the construction of troughs at the edges to afford

place for expectoration and to prevent dirt from the floor draining into the pool. Sediment on the bottom should be removed with suction pumps. The filtering should be continuous and the filter of sufficient capacity to refilter all the water in the tank in eight to twelve hours. Clear water is essential not only for æsthetic reasons, but to reduce the hazard of drowning.

Satisfactory hygienic conditions in swimming pools require suitable administration of the plant, including the supervision of the working force, the inspection and ablution of the bathers before they enter the water, and their instruction in pool sanitation. Finally, sterilization of the towels and bathing suits by boiling or steaming after each use will avoid one of the sources of conveying infection.

DRINKING FOUNTAINS

The movement toward the abolition of the common drinking cup has led to the development of so-called drinking fountains, of which there are many types on the market. They may be divided into intermittent and continuous, and each in turn into those with and without suitable mouthguards.

The water should not pass through a cup sure to become contaminated. Experiments show that it takes a long time for bacteria to be eliminated from these cups. The design should be such that the user cannot touch the ball with the lips, and that waste water cannot remain to endanger the next user of the fountain. The Committee on Sanitary Drinking Fountains³⁹ recommended that: (1) Mouthguards are a necessity. (2) The intermittent vertical jet fountain is unqualifiedly condemned. (3) Continuous vertical jet fountains are open to suspicion. (4) A slanting jet projected with a mouthguard is perfectly safe.

ICE

Ice was not suspected of being a vehicle by which infection could be spread until it was shown in bacteriological laboratories that typhoid and other cultures are not killed by freezing. Leidy in 1848 showed that water derived from melted ice contained not only living infusoria, but also rotifers and worms. Macfadyen proved that the temperature of liquid air (-315° F.) does not kill bacteria. In fact, some bacteria and molds grow and multiply at temperatures as low as 0° C. See also effects of cold, page 675.

Sedgwick and Winslow⁴⁰ (1902) were the first to make quantitative studies on the effect of freezing upon pathogenic bacteria. They used cultures of the typhoid bacillus and showed that 50 per cent of the organisms die at the end of the first week, 90 per cent at the end of the second week, and prac-

³⁹ *J. Am. Water Wks. Assn.*, 1918, 5: 110.

⁴⁰ W. T. Sedgwick, and C.-E. A. Winslow: (1) "Experiments on the Effect of Freezing and Other Low Temperatures upon the Viability of the Bacillus of Typhoid Fever, with Considerations Regarding Ice as a Vehicle of Infectious Disease." (2) "Statistical Studies on the Seasonal Prevalence of Typhoid Fever in Various Countries and Its Relation to Seasonal Temperature." *Mem. Am. Acad. Arts and So.*, Aug. 1902, 12, No. 5. Summary, *Boston Soc. Med. Sc.*, 1899-1900, 4: 181.

tically all at the end of twelve weeks. They consider that we may be sure that in nature the destruction would exceed rather than fall short of these figures, for the experiments were made in a test-tube where all the bacteria are imprisoned, while in nature perhaps 90 per cent are extruded during the purifying process of freezing.

Ruata ⁴¹ finds even more rapid destruction. Thus, cultures of *Bact. coli*, four strains of another bacillus and a streptococcus were killed after four days at -3° C. to -12° C.

As water crystallizes it extrudes suspended matter and even dissolved substances. The extent to which water thus purifies itself depends, however, upon conditions, for under certain circumstances the impurities may be entangled or even concentrated during the process of freezing.

S. C. Keith ⁴² considers that low temperatures alone do not destroy bacteria. On the contrary, they appear to favor bacterial longevity, doubtless by diminishing destructive metabolism. Frozen food materials, such as ice cream, milk and egg substance, favor the existence of bacteria at low temperatures, not because they are foods, but apparently because they furnish physical conditions somewhat protective to the bacteria. It seems likely that water-bearing food materials freeze in such a way that most of the bacteria are extruded from the water crystals with other non-aqueous materials (including air) and the bacteria then lie in or among these substances without being crushed or otherwise injured. In pure water, and above all, in water in which the whole mass becomes solidly crystalline, the bacteria have no similar refuge, but are caught and ultimately mechanically destroyed between the growing crystals. This explanation would account in part for the absence of live bacteria in clear ice, their comparative abundance in "snowy" ice and "bubbly" ice, and also the fact that the more watery food materials when frozen contain the fewest, and the least watery the most living bacteria. At low temperatures metabolism ceases and the bacteria continue to exist in a state of suspended vitality similar to that exhibited by many other and higher plants which, in the far North, are subject, without apparent injury, for long periods to temperatures much below the freezing point of water.

Hilliard and Davis ⁴³ state that intermittent freezing exerts a more effective germicidal action than continuous freezing. The reduction is much less in milk and cream than in pure tap water. The degree of cold below freezing is not a very important factor in the destruction of bacteria. The bacteria are probably killed by crushing in the crystallization rather than by the cold, or by the "shock" of freezing and refreezing.

It is necessary to distinguish between natural ice and manufactured ice.

Natural Ice.—Natural ice should be harvested from water of good sanitary quality and handled in a cleanly manner. Even when natural ice is obtained

⁴¹ *Ann. d'ig.*, Roma, Jan. 1918, 28, No. 1, 1125.

⁴² *Science*, n. s., June 6, 1913, 37, No. 962, 877-879.

⁴³ *J. Bact.*, July, 1918, 8, No. 4, p. 423.

from a polluted water the danger is greatly reduced, not only because ice purifies itself in freezing, but because natural ice is usually stored weeks and months before it is used. There are plenty of clean, fresh streams, lakes and ponds from which an abundant supply may be obtained. It is comparatively easy to protect most ponds, from which ice is harvested, from undesirable pollution. Under natural conditions the surface layer of ice contains most of the impurities and the lower layers are relatively purer, for the reason that ice grows from above downward and extrudes both suspended and dissolved matters; the surface, however, receives additional contamination from the dust, snow, flooding and other sources. It is, therefore, good practice to plane the surface of snow ice.

The fact that natural ice is usually purer than the water from which it is taken is shown by the following analyses which give the chemical and bacterial composition of natural ice and the water from which it was frozen. Only the minimum and maximum values for each set of samples are given. In this case the water was a sewage-polluted stream:

	Ice 3 to 6 Inches Thick		Water	
Number of samples	6		6	
Free ammonia008—	.034	.046—	.084
Albuminoid ammonia156—	.214	.146—	.276
Nitrates05 —	.20	.35 —	.48
Chlorin	2.0 —	3.0	4.5 —	6.0
Hardness	11.0 —	28.5	57.0 —	61.5
Bacteria per c. c.	30.0 —	210.0	5200.0 —	13000.0
<i>Bacterium coli</i> in	10 c.c.—	10 c.c.	1.0 —	.1
Number of samples	7		4	
Free ammonia016—	.136	.006—	.038
Albuminoid ammonia230—	.726	.116—	.166
Nitrates0 —	.050	.260—	.400
Chlorin	0.8 —	3.5	5.5 —	
Hardness	18.0 —	34.0	58.5 —	62.0
Bacteria per c. c.	2.0 —	60.0	2500.0 —	3900.0
<i>Bacterium coli</i> in	absent		1.0 —	0.1 c.c.

The chemical figures in this table are in parts per million.

The reduction in the number of bacteria is noteworthy. It will be noticed that there was no diminution, rather an increase in the free and albuminoid ammonia. All other constituents were reduced by the freezing process.

Manufactured Ice.—Manufactured ice is now universally made by the ammonia process. The condensed ammonia in expanding requires heat which it takes from surrounding objects and in this way the water is frozen. There are two distinct processes: one known as “can ice” and the other as “plate ice.” In the first case the freezing takes place in rectangular cans, the water freezes from the sides of the can toward the center, and the impurities are extruded and concentrated in the core, which is often visible in a cake of can ice. In well-equipped plants this visible core of concentrated impurities is removed by suction apparatus before it freezes, and clean water sub-

stituted. In making can ice the water must first be distilled or boiled in order to drive out the air, else the resulting product will be bubbly. Plate ice is made by freezing water in large shallow tanks. The water freezes upon the surface and when of sufficient thickness is cut out and removed in blocks. In this method it is not necessary to distill or boil the water for the reason that the air is extruded naturally during the process of freezing. Manufactured ice should be made from water of good sanitary quality, especially as it is not usually stored a long time before it is used.

When ice is made from distilled or boiled water it should be above reproach. I have found, however, that manufactured ice may contain more bacteria than the water from which it was made. This is due to unclean methods. Thus six specimens of plate ice made from water containing 64 bacteria per cubic centimeter and no colon bacilli gave the following results:

Number of Sample	Manufacturer	Organisms per Cubic Centimeter	Colon Bacillus
24	C. P. Co.	455	Absent
29	C. P. Co.	5,000	In 1 c.c.
26	G. Ice Co.	230	In 10 c.c.
27	G. Ice Co.	650	Absent
32	C.-S. Co.	470	Absent
34	P. Ice Co.	8	In 1 c.c.

The laborers who work "on ice," as it is termed, are liable to scrape considerable amounts of dirt from their shoes in walking over the cans and tanks, and pollution may come from other sources.

The chemical examination of manufactured ice may show conspicuously less total solids, less chlorin and less nitrates than found in the water from which it was made. On the other hand, it may be very high in free ammonia. This is accounted for by the fact that there is always some leakage of this gas about ice factories using the ammonia process. Sometimes ammonia occurs in such quantities as to impart a distinctly alkaline taste to the manufactured ice.

There is no excuse for unclean methods in handling ice that is used on or in our foods. The fact that surface impurities may be washed from a cake of ice is no reason for dragging it over sputum-laden pavements, over dirty railroad platforms, or for using similar unclean methods. The general use of ice is a modern innovation. It has come into vogue within the past one hundred years. For the uses of ice as a preservative see page 675.

Ice and Disease.—A search of the literature discloses but few instances of disease attributable to impurities in ice. While the experimental evidence indicates that there is a quantitative reduction of the number of bacteria in freezing, and that the imprisoned bacteria gradually die, nevertheless experience has shown that low temperatures alone cannot be depended upon to remove the danger of typhoid infection. For example, we have the water-borne epidemic in Plymouth, Pennsylvania, in 1885, presumably produced

from the frozen accumulation of typhoid excrement from a single case. Very similar to the Plymouth outbreak was that at New Haven, Connecticut, in 1901. In only a few isolated instances, however, has ice itself been accused of being the vehicle by which the infection of typhoid fever has been spread. It appears probable that milder intestinal diseases may be caused by highly polluted ice, of which the Rye Beach epidemic, studied by Nichols ⁴⁴ of Boston in 1875, is a point in evidence.

Park ⁴⁵ (1901) described an epidemic which was believed to have had its origin in ice obtained from a pond on which it was shown that the excrement from a patient sick with typhoid fever had been thrown while the pond was covered with ice.

In the second annual report of the Board of Health of Connecticut for 1882 an interesting single case of typhoid fever is cited as probably derived from ice.

Dorange ⁴⁶ (1898) described an epidemic of typhoid fever attributed to ice among eight lieutenants in a regiment stationed at Rennes in the autumn of 1895. The implication of the ice in this instance rests upon a doubtful chain of evidence, however, and no mention is made of other possible factors.

Hutchins and Wheeler ⁴⁷ (1903) report an epidemic of typhoid fever in the St. Lawrence State Hospital, three miles below Ogdensburg, New York, which seems to have been due to impure ice. The disease was endemic in the hospital for ten years, increasing from 2 cases with the opening of the hospital in 1890 to 40 cases in 1900. Although the water supply, tested bacteriologically and chemically, gave negative results, all observers agreed that the disease was water-borne. In December, 1900, the source of the water supply was changed to the Oswegatchie River, a small Adirondack stream supplying Ogdensburg. This practically put a stop to the disease, for there were no cases of typhoid that were not clearly contracted elsewhere until October, 1902.

Following this 8 persons were attacked, 7 of whom were employees in the dining-room. It seems the milk "could not have been infected." The water was excluded and other sources studied, with negative results. The ice fell under suspicion. It had recently been taken from a newly opened ice-house. The ice had been harvested from the St. Lawrence River at about the same spot as the ice previously used. It was gathered in February and consequently had been stored for seven months. This ice disclosed a contamination of 30,400 bacteria per cubic centimeter on agar plates and 50,400 on gelatin. Of eight fermentation tubes three showed the presence of colon bacilli.

⁴⁴ A. H. Nichols, "Report on an Outbreak of Intestinal Disorder Attributable to the Contamination of Drinking Water by Means of Impure Ice," *Seventh Ann. Rep.*, State Bd. Health, Massachusetts, 1876, p. 467.

⁴⁵ W. H. Park, *Virchow-Hirsch's Jahrbuch f. 1901*, p. 16.

⁴⁶ Dorange, "Epidémie de Fièvre Typhoïde dû à l'Ingestion de Glacé Impure." *Rev. d'hyg.*, 1898, 20: 295.

⁴⁷ R. H. Hutchins and A. W. Wheeler, "An Epidemic of Typhoid Fever Due to Impure Ice," *Am. J. M. Sc.*, Vol. 126, 1903, p. 680.

The stock of ice was then examined. In the center of certain cakes were found foreign substances in the form of black or dark brown granular matter. Examined under the microscope, this matter was found to be teeming with bacteria, from which both the colon and typhoid bacillus were isolated in pure culture.

With the discontinuance of the use of this infected ice the epidemic gradually subsided. There were in all 39 cases. The evidence of this outbreak was studied by Hill, who doubted the relation of the ice. The disease resembled a carrier outbreak.

Special interest attaches itself to the report of an outbreak of typhoid fever due to infected ice reported by Conway.⁴⁸ The outbreak, which occurred in Elmira, New York, during the summer of 1923, included 37 cases. Conway believes that in view of the facts presented there seems little doubt that the outbreak of typhoid was directly due to the consumption of the superficial layer of natural ice, harvested from the heavily polluted Chemung River, even though stored, as it was, for a period of from five to six or possibly seven months. He also warns against the use of iced drinks chilled with ice harvested from polluted sources, even though the ice is subject to several months' storage.

Owing to the fact that ice purifies itself in the process of freezing, typhoid fever has rarely been traced to the ice supply. In fact, very few instances are on record in which ice has been accused, and in these instances the evidence is far from satisfactory.

REFERENCES: COLLATERAL READING

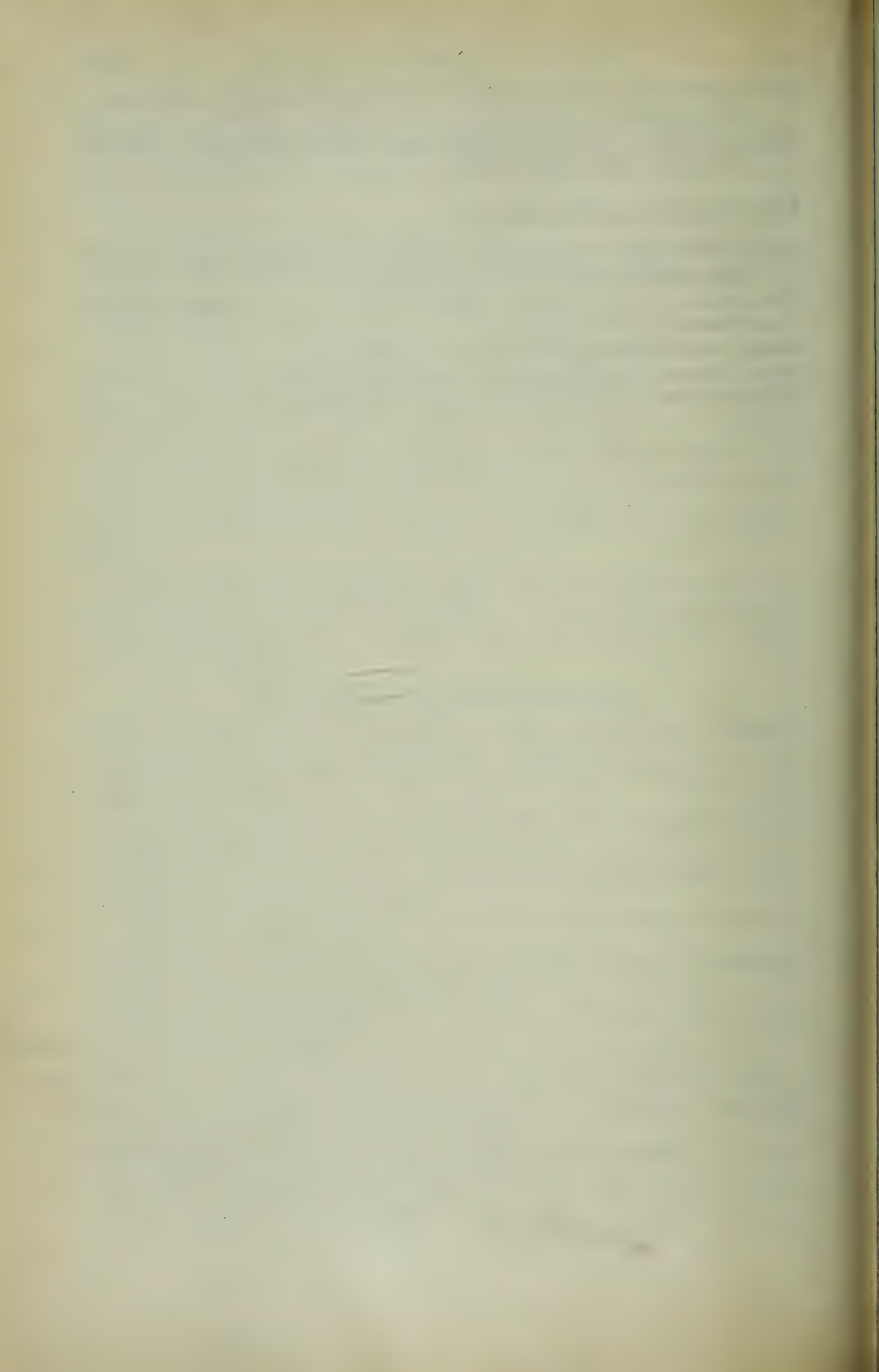
- Report of the Committee of the American Public Health Assn., "Standard Methods of Water and Sewage Analysis," 1923, 5th Ed.
- Manual of American Water Works Practice.* Williams & Wilkins Co., Baltimore, 1925.
- WHIPPLE, G. C. *The Microscopy of Drinking Water*, Wiley & Sons, New York, 1914, 3rd Ed.
- PRESCOTT, S. C., and WINSLOW, C.-E. A. *Elements of Water Bacteriology.* Wiley & Sons, New York, 1924, 4th Ed.
- SAVAGE, W. G. *The Bacteriological Examination of Water Supplies.* Blakiston's Son & Co., Philadelphia, 1906.
- THRESH, J. C. *The Examination of Waters and Water Supplies.* Blakiston's Son & Co., Philadelphia, 1914, 2nd Ed.
- TURNEAURE, F. E., and RUSSELL, H. L. *Public Water Supplies.* Wiley & Sons, New York, 1924, 3rd Ed.
- MASON, W. P. *Water Supply.* Wiley & Sons, New York, 1916, 4th Ed.
- HAZEN, A. *Clean Water and How to Get It.* Wiley & Sons, New York, 1916.
- DON, J., and CHISHOLM, J. *Modern Methods of Water Purification.* Longmans, Green & Co., New York, 1913, 2nd Ed.
- JOHNSON, GEORGE A. "The Purification of Public Water Supplies," U. S. Geological Survey, Water-Supply Paper 315, 1913.

⁴⁸ *Am. J. Pub. Health*, 1924, 16: 574.

- ELLMs, JOSEPH W. *Water Purification*. McGraw-Hill Book Co., New York, 1917, 1st Ed.
- STEIN, MILTON F. *Water Purification Plants and Their Operation*. Wiley & Sons, New York, 1919, 2nd Ed.

For typical and composite analyses see:

- CLARKE. "The Data of Geochemistry, Chemistry and Physics," 54, U. S. Geological Survey, 1908, Bull. No. 330, Series E.
- "The Municipal Water Supplies of Illinois," *Bull. of the Ill. State Board of Health*, June, 1908, 4, 6.
- Journal of the American Water Works Assn.*, Baltimore.
- Annual Reports of the Massachusetts State Board of Health.
- Bulletins of the U. S. Geological Survey. Water supply papers.



SECTION X

SEWAGE

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The water-carried discharges of the human body together with the liquid wastes from household and factory are called sewage. The discharges themselves are known as excreta and consist chiefly of feces and urine, although they may include secretions from the skin, mouth, and nose. Savage tribes and nomadic races simply move away from their excreta, but civilization requires that the wastes of the human body be removed from habitations promptly and safely. In rural communities excreta are cared for in privies; in urban communities they are removed by means of the water carriage system. The structures required by the latter constitute a sewerage system. This includes pipes or *sewers* which collect and convey the sewage, pumps that may be necessary to lift the sewage from low-lying sections, treatment works, where sewage treatment is employed, and disposal works.

The rural problem of excreta disposal is quite as large and important as the urban problem of sewage disposal. In 1915 over half the population of the United States lived in unsewered homes and this is probably still true to-day. The following tabulation gives the figures of the development of sewerage and sewage treatment in the United States:

	1905 *	1910 †	1915 ‡
Population	84,000,000	92,000,000	99,000,000
Unsewered, per cent.....	66.7	62.3	57.7
Sewered, per cent.....	33.3	37.7	42.3
With treatment works..	1.3	4.2	7.0
Disposing of sewage in lakes and streams....	24.3	} 33.5	26.7
Disposing of sewage in the sea	7.7		8.6

* Based on estimates by George W. Fuller, T.A.S.C.E., 1905, Vol. XLIV.

† Based on estimates by George M. Wisner.

‡ Based on estimates by Metcalf and Eddy, *American Sewerage Practice*, Vol. III, p. 240.

Three viewpoints must be considered in studying the disposal of human excreta: the hygienic, æsthetic, and economic. The *hygienic viewpoint* is concerned chiefly with the fate of pathogenic organisms that may be con-

¹ This section is based upon material prepared by the late Professor George C. Whipple for previous editions of this book.

tained in human wastes. The microorganisms of typhoid fever, cholera, dysentery, hookworm, and certain other intestinal diseases have their ultimate source in the human discharges, and are conveyed from host to host through many channels by water, food, soil, or contact, sometimes by human agencies, sometimes by animals, more particularly by flies. The safe disposal of excreta therefore requires that the methods chosen shall eliminate or at least minimize the following dangers:

1. The pollution of water, both surface and ground
2. The pollution of the soil
3. The pollution of food materials that are to be consumed raw
4. The pollution of animals that may have access to food

The *æsthetic viewpoint* of sewage disposal deals with the destruction of the organic matter contained in excreta and the production of offensive conditions. It demands that excreta be cared for in such a manner that they will become neither unsightly nor objectionable to the sense of smell. It also considers the privacy to be secured during defecation.

The *economic viewpoint* concerns itself chiefly with the price that must be paid to secure the requirements of hygiene and æsthetics. In western countries it does not yet deal as much as in the East with the utilization of excreta for fertilizing purposes, but the near future will probably see a large development of this aspect of the problem.

THE RURAL PROBLEM OF SEWAGE DISPOSAL²

One of the most difficult problems of modern sanitation is to secure proper disposal of fecal matter for rural communities, summer hotels, temporary camps of laborers, summer colonies at beach and mountain, and individual houses in villages and on the farm. It is difficult because the necessary structures are so small and simple that they are often thoughtlessly constructed; furthermore because adequate care of the processes is more or less disagreeable and therefore neglected; but chiefly because the inherent dangers have not been understood or appreciated.

Privies.—There are still large areas of the United States in which no attempt is made at the sanitary disposal of human excreta. Only a few years ago a survey made by the United States Public Health Service³ showed that in fifteen counties scattered through eleven states of the Union from 92 to over 99 per cent of the country homes disposed of human excreta in a grossly insanitary fashion. The corresponding figures for town homes varied between 32 and 87 per cent. A clump of bushes or trees often provided sufficient privacy for defecation. Excreta were scattered on the surface of the ground and even the Mosaic law of burial was not obeyed.

² Adapted from a lecture given by George C. Whipple before the Michigan conference of Health Officers and Public Health Nurses, and printed in the *Michigan Public Health*, March 1923, p. 125.

³ *Pub. Health Bull.*, No. 94.

The first step towards sanitation is found in the *latrine* or straddle trench used in temporary army camps. See Figures 103 to 105. This consists of a relatively shallow trench which the user straddles. A layer of earth is placed

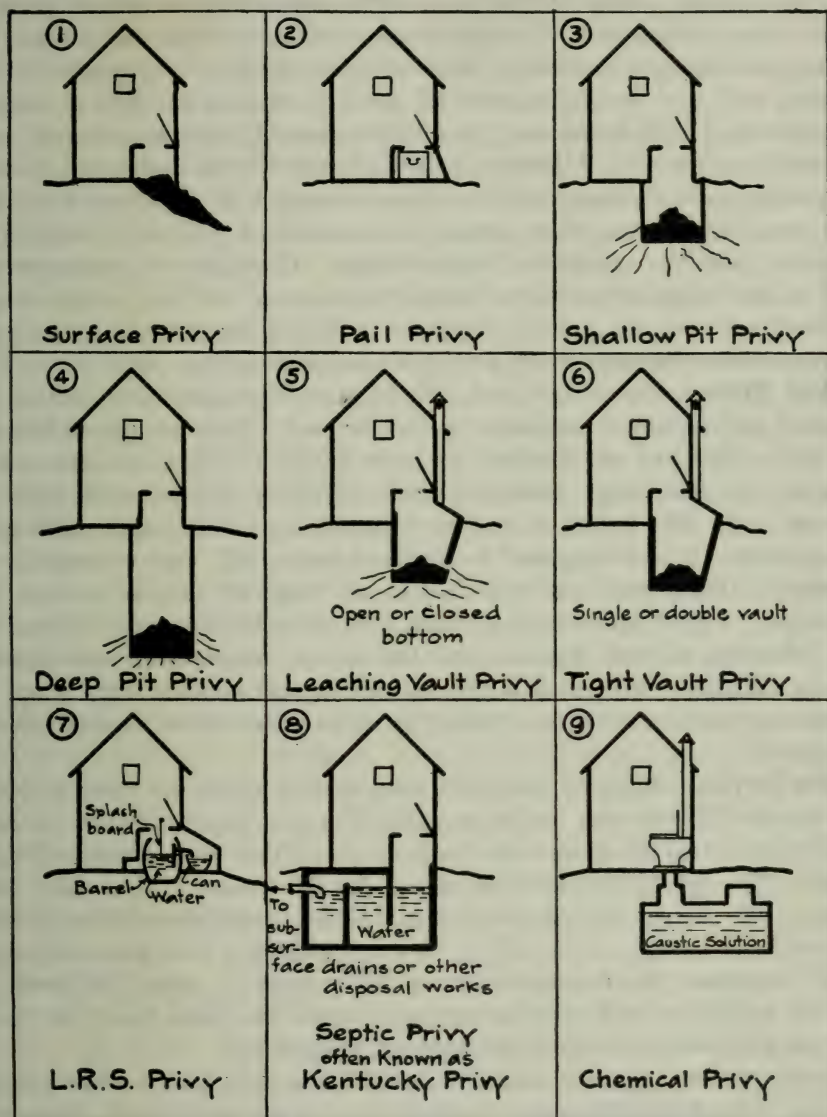


FIG. 100.—TYPES OF PRIVIES.

daily upon the fecal matter and the trench is finally back filled when camp is broken. Seating facilities are sometimes provided in the form of a supporting pole or rough box seat. This type of trench has its corollary in civil life in the so-called "umbrella type" of privy, consisting of a box placed over a hole in the ground. It is not a privy in the true sense of the word, since it affords

no privacy to its user, neither does it protect him against the elements; hence its name. A number of different types of privies are illustrated in Fig. 100.

Surface Privies.—The simplest type of privy is the surface privy. It may be open or closed at the back. The superstructure affords privacy. There is no substructure. The excreta accumulate on top of the ground, the liquids leach away or evaporate. Made of brick, or tongue and groove boards, painted, with solid foundation, with all openings screened and with a screened ventilator and self-closing seat, this is a reasonably safe arrangement and one easily maintained. Flimsy makeshifts, however, with inadequate foundation, with cracks between boards, without screens, with ill-fitting doors, are very insanitary because they permit the entrance of flies which may carry infection from the excreta to human beings. If the privy is open at the back so that animals have access to the excreta or so that the privy contents are scattered upon the ground, the sanitary danger may be very great, especially in warm climates. Such privies are an abomination.

Pail Privies.—In villages and even in some large-sized communities the so-called pail system, or scavenger system, is used. Pails are placed beneath the privy seats and are emptied at intervals by a collector; earth, ashes, sawdust, or some other absorptive material being put into the pails to prevent odor. The system is capable of furnishing a satisfactory solution of the problem. It was long used in England and is still used successfully in Australia. Great care and faithfulness are required on the part of the scavenger. Under American conditions it is often a failure, due to neglect, the difficulties of final disposal, and the cost of maintenance and upkeep. Unless well supervised, such a system, starting off satisfactorily, is apt to degenerate until, after a few years, the sanitary conditions become grossly dangerous.

Pit Privies.—Safer, on the whole, than surface privies are those in which the excreta fall into pits in the ground. The pits may be shallow or deep and built so that their contents leach away, or they may consist of tight vaults. The use of pits tends to reduce the fly menace. Flies shun dark places. The deeper the pit the fewer the flies, especially if the contents are oiled and burned occasionally as in army practice. It is said that under some conditions the decomposition of the excreta in deep pits produces enough marsh gas and carbonic acid to prevent flies from living in them. But no pit, however deep, is wholly proof against flies.

Deep pits are a greater menace to neighboring wells than surface privies, because, being below the normal bacterial zone of the soil—that is, below the zone of the living earth—the organic matter is not as quickly decomposed. Deep pits are objectionable in places where the soil water rises into them. No arbitrary rules as to the minimum distance of a privy from a well can be laid down, as everything depends upon the character of the soil, the slope of the ground, the elevation of the natural ground water, and the draught of water from the well. A distance of at least 25 feet should be secured with sandy soil and preferably 50 feet or more. In clay soils that dry and crack

and in limestone regions that contain crevices in the rock, leaching privies should not be used, as wells may be polluted 100 feet or even a mile or more away. The pollution of ground water can be prevented by using water-tight privy vaults made of brick or concrete, and this is often done. Frequent cleaning is necessary to insure safety. When properly constructed, tight vault privies and pail privies may be located near dwellings, the controlling conditions being only those relating to offensive odors, but this presupposes greater care than is ordinarily given to such matters. Preferably, therefore, privies should be situated at a reasonable distance from dwellings. Vault privies are sometimes constructed with two receiving compartments and two seats. After one vault has been in use for a time the seat above it is battened down and the other vault is called into service. This gives the organic matter in the unused vault time to decompose and reduces the frequency of vault cleaning.

Tank Privies.—Within comparatively recent times tanks of water placed beneath the privy house have come into use in this country. They are of two kinds—the septic tank and the chemical tank. The *septic tank privy* is by no means new. It is said to have originated in India where it has been used from time immemorial. It has also been called the Turkish system. The early types had a single tank; modern types often have two or more compartments. The principle is that of the septic tank described later. The excreta fall into the water, where, by the action of bacteria, the organic matter is decomposed, much of it being liquefied or gasified. The tanks are water-tight, but an exit pipe into the ground is usually provided near the top of the last compartment, through which the overflowing liquid passes into the soil. This discharge, together with decomposition and evaporation, prevents the accumulation of water. In some climates, as a matter of fact, it is necessary to add water periodically. In warm climates tank privies are decidedly superior to the older methods, but they are not well adapted to the severe weather of the North. Septic tanks are made in various ways. A simple and cheap form is the L.R.S. (Lumsden-Roberts-Stiles) privy. Another type, the so-called Kentucky privy, is made of concrete. Large tile pipe can also be used to advantage.

Chemical tanks are water-tight compartments, usually made of sheet metal, filled with water in which caustic soda or a similar chemical is dissolved. The action is practically the reverse of the bacterial method, the organic matter being decomposed chemically and the bacteria being largely destroyed. The method is a safe one, but it is relatively expensive. The cost of the chemical, which has to be renewed at intervals, is likely to induce householders to use too little of it and the process then breaks down.

Drop Privies.—Privies have sometimes been built in such a manner that they overhang brooks or rivers. The fecal matter drops into the water and is carried away by the stream. Although this presents an easy method of removing human wastes, the dangers arising from the pollution of water courses exceed by far the convenience and safety to the privy owner. Typhoid

fever epidemics have been traced to drop privies situated on tributaries of streams used for municipal water supplies. Most states, therefore, do not countenance the building of these structures.

Disposal of Privy Contents.—No matter what type of privy is adopted it becomes necessary sooner or later to remove part or all of the contents of the receiving chamber. This is especially true in the case of pail privies. Here the pails serve only as temporary storage receptacles and must be removed at frequent intervals, usually once a week. Vault privies require less frequent cleaning, particularly when the liquid wastes are permitted to leach away through the ground. A semi-annual removal of the accumulated solids usually suffices. Pit privies are sometimes cleaned but more often the superstructure is moved away from the pit when the latter becomes reasonably full. The house is placed over a new pit; the old one is filled with earth. Septic tank privies are cleansed only at rare intervals, because much of the solid material gasifies or liquefies, the liquid passing into the ground through the overflow or through subsurface tile drains. Chemical tanks too are only rarely emptied. A special cesspool is sometimes provided to receive the tank contents which are emptied into it from time to time. The tank is then refilled with caustic solution. Apart from the use of privy contents for fertilizing the fields, there are several other methods for their ultimate disposal: namely, burial, incineration, and dilution.

Fertilizing.—Human excreta, like animal manure, contain materials of fertilizing value. Urine is particularly rich in fertilizing substances. In primitive civilizations excreta are commonly used to fertilize fields upon which agricultural products are raised. From the hygienic standpoint this practice must be condemned as it infringes against all the requirements for the sanitary disposal of human wastes outlined above. Storms may wash the fecal matter into streams, the soil may become polluted, vegetables that are to be consumed raw may become contaminated, and flies may swarm from the fields to human habitations. The fresher the excreta the greater the danger. When the excreta are well rotted and plowed under and when they are not used for crops that are to be consumed raw in the immediate future, the sanitary hazards are reduced.

Burial.—A safer method of excreta disposal is to bury it in trenches or pits, a layer of earth sufficient in depth to prevent access of flies or other animals being placed over the material. Sometimes the pits or trenches are oiled, sometimes they are filled with straw or other combustible material which is burnt and forms a carbonized crust upon the surface of the wastes. Reduction of the organic matter is slow and relatively large disposal areas are required. The excreta can conveniently be mixed with other organic refuse material, such as garbage.

Incineration.—In some of the larger unsewered communities, excreta, more particularly those collected from pail privies, are destroyed by incineration, the liquids being evaporated, the solids passing into the combustion chamber of the incinerator. Small incinerators have been used in army cantonments

and construction camps. They afford an opportunity for the simultaneous destruction of garbage and rubbish. Large scale incineration has been applied successfully in Australia.

Dilution and Sewerage.—Communities situated near large rivers that do not serve as water supplies for nearby towns often dispose of the collected excreta in central disposal stations in which a jet of water washes the pail contents into a sewer discharging into the river. This method is also used in communities that contain sewered as well as unsewered areas, the contents of pail privies in the unsewered districts being emptied into one of the trunk sewers of the city.

Disposal of Water-Closet Wastes.—More and more people who live on farms or in small villages are introducing running water into their houses. The electrical distribution of power, gasoline engines, water rams, windmills, and other sources of power have made this possible. Farm plumbing is rapidly increasing and the indoor water-closet is being substituted for the outside privy. Whereas this is a sanitary advance, it brings with it new requirements that must be met if healthful conditions are to prevail. Water closets and other plumbing fixtures greatly increase water consumption and new means must be provided for getting rid of the increased volumes of waste water. This usually means the construction of a cesspool, a septic tank, or some other small sewage disposal unit.

Cesspools.—The word cesspool means a pit into which waste water flows. If the pit has permeable sides, it is called a *leaching cesspool*; if water-tight, it is called a *tight cesspool*. Cesspools are generally covered. Usually the sewage remains in them a considerable time so that the bacteria have ample opportunity to act upon the organic matter. Bacteria ordinarily found in water require oxygen, and fresh sewage from a house usually contains some of it. On reaching a cesspool the bacteria in the sewage seize this oxygen and it very speedily disappears. The resulting condition is called “anaërobic.” Some bacteria can, under these anaërobic conditions, get the oxygen they need from the organic matter—that is, from the feces, urine, and even from the cellulose of paper. In doing so, they break down the organic matter and some of the solids are changed to liquids or gases. This is called the *septic process*. It is not purification in the proper sense of the term, but it is a useful process and reduces the accumulation of solids in cesspools. It thus prevents the necessity of frequent cleaning and makes it easier for the liquids to leach into the soil.

Cesspools often receive not only fecal matter but also other domestic wastes. When the soil is sandy there is no objection to the use of leaching cesspools; in fact, this method is like that of subsoil irrigation described later in this section, except that the sewage is discharged into the soil below the depth where bacteria are at work. This may be an important difference, however, as the oxidation of the dissolved organic matter proceeds by a slow and incomplete process. Leaching cesspools should not be located near wells used for drinking water supplies. In sandy soils the danger of bac-

terial contamination is small if sufficient distance intervenes but, even so, the idea of infiltration of sewage into a well is repugnant, and the water may often be so tainted as to have a disagreeable odor, although analysis may show it to be bacterially safe.

Ordinarily, leaching cesspools should not be used in clay soils or in limestone regions. If they are to be constructed in such soils, they should be made water-tight and treated as septic tanks, the effluent being taken care of by subsurface irrigation or some form of land treatment.

Septic Tanks.—Septic tanks differ from cesspools in having water-tight sides and bottoms, in having a definite outlet as well as an inlet, in having

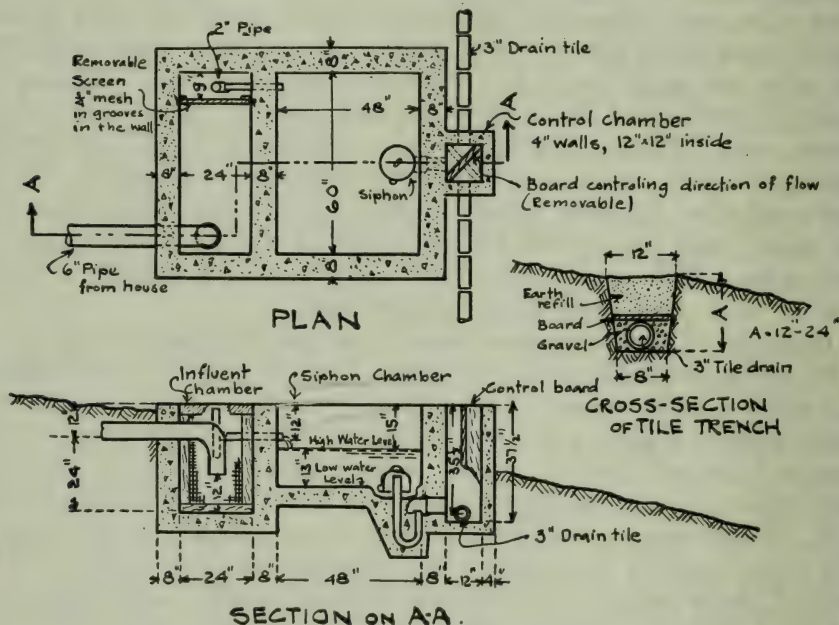


FIG. 101.—SEPTIC TANK AND SUBSURFACE IRRIGATION.

A tank with these dimensions will serve five people. *U. S. Pub. Health Serv. Bull. 101.*

a more or less constant flow of liquid through the tank, and sometimes in being subdivided into compartments. When subdivided, the second compartment commonly serves as a dosing chamber for the irrigation plant or filter. The biological action is the same as in the cesspool; there is liquefaction and gasification of a part of the organic matter, a small amount of sediment gradually accumulating at the bottom, a considerable amount of scum being lifted to the top by the entrained gases.

As in the case of cesspools, the water from septic tanks has to go somewhere. It may be distributed underground by a system of subsurface drains—a good way if the soil is porous—or it may be applied to some sort of filter. A satisfactory construction of a septic tank is shown in Fig. 99.

Other Methods of Rural Sewage Disposal.—Although the cesspool and septic tank constitute the chief methods of sewage disposal for small isolated dwellings with water supply systems, there are a number of other processes that can be employed when the above methods are not satisfactory owing to conditions of soil, location, or volume of sewage to be treated. These methods approach very closely the processes used in the treatment of sewage from urban communities described later and are therefore only enumerated here. They are: intermittent filtration following septic treatment or Imhoff tank treatment, and lath trickling filters following Imhoff tank treatment and succeeded by secondary Imhoff tanks.

Disposal of Kitchen and Other Liquid Wastes.—One factor in the use of septic tanks, cesspools, and other rural sewage disposal methods that is generally neglected is the great extent to which the presence of grease in sewage interferes with any treatment process. Grease decomposes slowly under anaërobic conditions. It also coats the particles of fecal matter and cellulose and prevents their destruction. In leaching cesspools and subsurface irrigation drains it tends to choke the pores of the ground and prevent percolation.

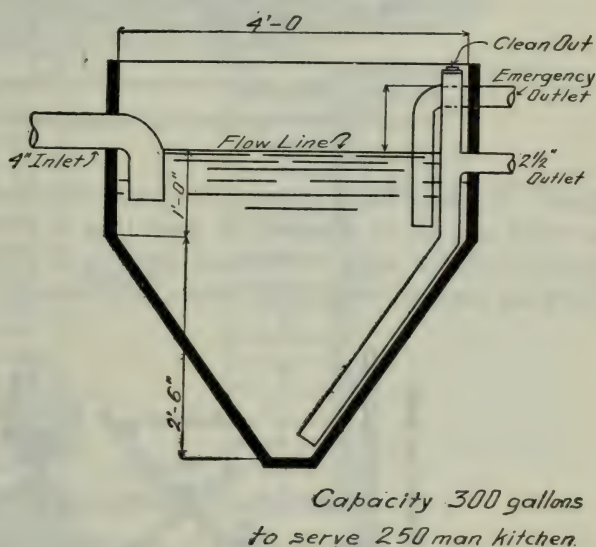


FIG. 102.—UNITED STATES ARMY GREASE TRAP.

Grease and fats decompose with least nuisance in the presence of oxygen, whereas solid fecal matter and cellulose are liquefied best under anaërobic conditions. It follows that the two substances, grease and fecal matter, should be kept apart as far as this can be practically accomplished. Where privies are used, sink wastes are generally put upon the open ground. This may be unsightly, but the danger to health is not very great. When plumbing is introduced, the natural thing for the plumbers to do is to unite all the waste pipes as they do in cities. This brings fats from the kitchen and fecal wastes from the water-closets together and causes the difficulties referred to. It is true that partial separation can be obtained by the use of grease traps, but this has not proved to be very successful in practice. With greasy matters kept out, cesspools

can be used for years without cleaning and effluents from septic tanks can be dissipated through underground tile drains without clogging.

Grease Traps and Brush Filters.—If greasy wastes are kept separate from fecal wastes, they are readily disposed of by the use of grease tanks or brush

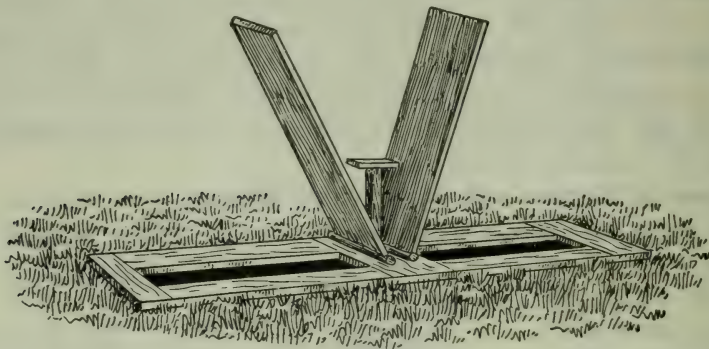


FIG. 103.—STRADDLE PIT COVER.

(Wilson's *Field Sanitation*, George Banta Pub. Co.)

filters. In *grease tanks* or *traps* the grease rises to the surface as the liquid is cooled and can be skimmed off from time to time. A tank used in connection with the army cantonment kitchens is shown in Fig. 102. *Brush filters* are made of twigs, small sticks, and branches commonly heaped along

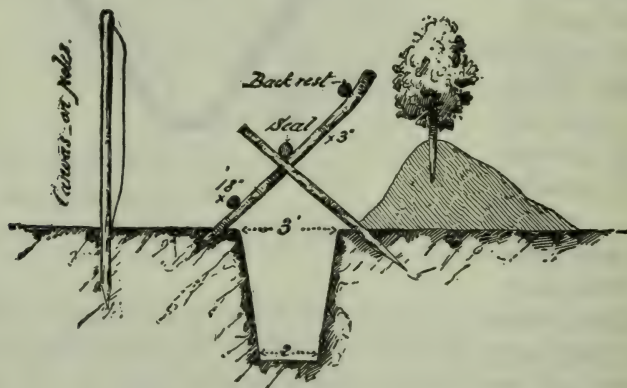


FIG. 104.—CONSTRUCTION OF PIT LATRINE.

(Munson's *Military Hygiene*, Wm. Wood & Co.)

a natural bank. The greasy liquid is permitted to discharge on to the brush and trickles through it. The grease adheres to the twigs, and decomposes in the presence of air without giving off offensive odors. The degreased waste liquor is discharged over the ground. Brush filters must be renewed after they have become loaded with grease, the greasy brush removed burning readily.

Hygienic Results of Rural Sanitation.—As is to be expected, the introduction of sanitary privies is commonly followed by a reduction in intestinal diseases. Thus Hardenbergh⁴ reports that typhoid fever deaths in Richmond, Virginia, were reduced from 57 in 1908 to 28 in 1909 following the installation of sanitary privies in all unsewered homes; further, that the introduction of sanitary privies in Birmingham, Alabama, decreased the typhoid fever death rate of 65.5 per 100,000 in 1917 to 17.8 in 1919. In Birmingham deaths from diarrhea and enteritis among children under two years of age dropped 50 per cent during the same period. Experience has furthermore

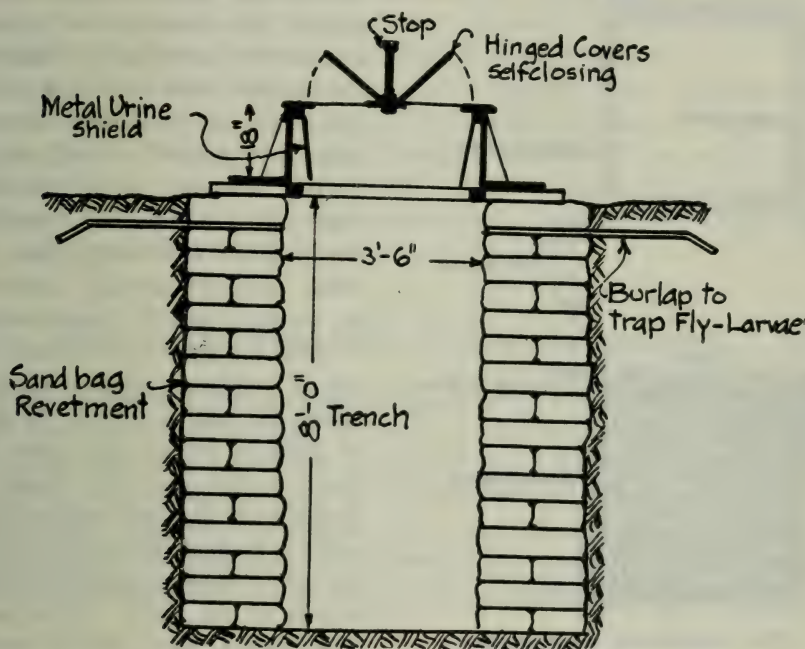


FIG. 103.—DEEP TRENCH FLY-PROOF LATRINE.

(After Lelean.)

shown that hookworm can be stamped out by similar measures and that the incidence of other intestinal diseases can be materially reduced.

Camp Sanitation.—The disposal of human wastes from camps—that is, from temporary shelters—presents a number of sanitary features not encountered under ordinary urban or rural conditions. We may distinguish between military camps in which rigid discipline permits thorough enforcement of sanitary regulations, laborers' camps in which intelligent supervision can ensure safe methods of excreta disposal, and tourists' camps which provide good sanitary facilities when properly controlled but more often leave hygienically important requirements to be decided upon by the individual

⁴ W. A. Hardenbergh, *Home Sewage Disposal*, J. B. Lippincott Co.

who knows little about sanitation and more often than not is unacquainted even with the requirements of the Pentateuch.⁵ The education of the touring public to an appreciation of the sanitary hazards involved in the indiscriminate disposal of human excrement is an important problem of the times.

The methods of excreta disposal that should be employed are similar to the rural methods described above. The structures built, however, are of

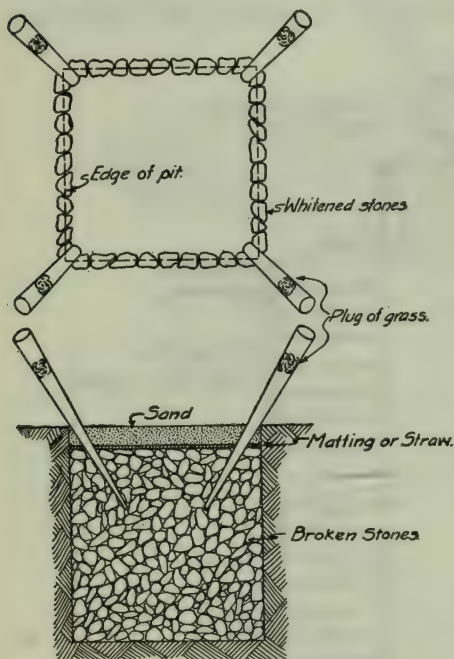


FIG. 106.—URINE SOAKAGE PIT.

than 2 feet) permits rapid disintegration of the excreta. When trenches of this type are to be used for a longer period of time, they are made deeper and are then called straddle pits. These are often provided with covers as shown in Fig. 103. Straddle trenches can be adapted to other temporary encampments as well as to military ones.

Latrines.—When the camp site is to be occupied for several days or weeks, more permanent facilities must be provided. These commonly take the form of latrines. A simple latrine, known as the pit latrine, is shown in Fig. 104. It differs from the straddle trench or pit in being wider and deeper and in providing rough seating facilities in the form of a pole and back rest supported on cross pieces. Latrines can be screened from view by brush or tent canvas stretched between upright poles. Excreta should be covered

necessity even more primitive. It is, nevertheless, essential that they should yield the same hygienic safeguards while caring for a large number of individuals. Some of the methods used by armies on the march or in temporary encampment are applicable to all camps. In the following paragraphs only those are mentioned, however, which differ sufficiently from the rural methods already described to warrant special consideration.

Straddle Trenches.—These consist of relatively shallow trenches which the user straddles. A layer of earth is placed daily upon the fecal matter and the trench is finally back filled and marked when camp is broken. The shallow straddle trench is recommended only for camps of one night. It is easily and quickly made, and by reason of its slight depth (commonly less

⁵ See Deuteronomy 23: 12-13.

with earth by means of a scoop and the trench should be burned out daily with straw and coal oil. When the contents reach 3 feet from the surface, the latrine should be filled, covered with oil and sacking, earthed over, and the site marked. Even when carefully operated, open pits permit the access of flies to the excreta. For this reason the deep trench fly-proof latrine shown in Fig. 105 has been devised. Seats are arranged in single or double rows. The latrine is housed or otherwise screened. A urinal trough draining into the pit or into a separate soakage pit is sometimes provided against one wall of the latrine shelter. Treatment of the pit contents is the same as in the open latrine. Accommodations should be provided for about 5 per cent of the command. During the Great War pail latrines, consisting of pails encased in boxes, were used extensively in the trenches.

Urinals.—Military camps are commonly equipped with galvanized iron cans which are placed in the camp streets at night. These are emptied into soakage pits in the morning and thoroughly cleaned. The ground where the cans stand is burned over each day. Urine soakage pits are sometimes used instead of cans. A common form is shown in Fig. 106. A pit 4 feet cube will serve from 200 to 250 men.

THE URBAN PROBLEM OF SEWAGE DISPOSAL

Although the problem of sewage disposal in urban communities has sometimes been solved satisfactorily by some of the methods just outlined, the natural tendency of community sanitation has been away from the individualistic methods of the rural householder towards collective sanitation of a surer and more effective nature. The problem naturally falls into two parts—namely, the collection of the sewage by what is called the sewerage system, and the disposal of the sewage with or without treatment.

SEWERAGE

A great many different methods have been advocated in the past for the removal of fecal matter from urban communities. Aside from the pail, scavenger, or dry-earth system discussed above, a pneumatic method was at one time seriously considered. In many of the larger continental cities of Europe house wastes are even to-day stored in underground vaults somewhat like cesspools. These are pumped out from time to time into tank cars which carry the wastes to disposal fields on the outskirts of the city. In the progressive civilized world, however, the water-carriage or sewerage system is now the standard method of municipal excreta removal. It has no superior.

The Water Carriage System.—So accustomed are we to present-day methods of urban sewerage that it is hard to realize that the system of water carriage of fecal matter is less than a century old. Up to 1815 the

public drains of London were not permitted to receive excreta; in Boston fecal matter was rigidly excluded from the sewers until 1833; and in Paris this was the case even up to 1880.

Following the report of the Health of Towns Commission in England in 1844, water-closets were rapidly introduced, and in 1847 their connection with the sewers was required by law. The modern sewerage system, therefore, dates from about the middle of the last century. Chesbrough designed a general sewerage system in Chicago in 1855. Boston's first sewerage commission was appointed in 1875. Baltimore was without a sewerage system until a few years ago; even as late as 1922, the system was not fully completed and over 20,000 houses remained unconnected.

The introduction of the water carriage system accomplished its purpose and effectually did away with the offensive accumulations of filth around city dwellings, but it gave rise to a series of other problems that sanitarians are now endeavoring to solve. The first sewers, like the ground and storm water drains from which they evolved or after which they were patterned, were naturally built to discharge their contents into the nearest available body of water—into river, lake, or harbor, according to the situation of the city. Where the streams were relatively large, no nuisance was caused by doing this, but where the streams were relatively small, foul conditions were soon manifested. Water supplies also became infected and in some instances great epidemics followed, while infection was spread in other minor ways. Thus the problem of the removal of fecal matter was sometimes solved at one place only to reappear elsewhere. Litigation also arose between riparian owners along the water courses, involving damages caused by the pollution of the water.

House Drainage or Plumbing.—The house plumbing is the beginning of the town drainage system. It collects the waste materials from toilet, wash bowl, bath tub, kitchen sink, laundry tray, and other fixtures. In combined systems it also carries away the rain water from roof and yard areas. The modern tendency is towards simplified systems. It pays to use good material and workmanship.

Sanitary Plumbing.—The main features of sanitary plumbing are one or more vertical *drainage stacks*—these rise from cellar to roof and collect the wastes from plumbing fixtures—and the *house drain* which runs horizontally under the cellar and receives the discharge from the drainage stack, conveying it to the *house sewer*. The latter extends outside of the building to the street sewer or house disposal plant. Pipes carrying the discharge of water-closets or urinals are known as *soil pipes*; those carrying the discharge of any fixture, except water-closets or urinals, are called *waste pipes*. Thus we may also distinguish between *soil stacks* and *waste stacks*.

Each fixture is equipped with a *trap*—that is, a fitting so constructed as to prevent the passage of air or gas from the drainage system through the fixture into the house. Traps usually contain a *water seal*. In order to prevent the unsealing of traps by syphonic action or back pressure as a

result of water rushing through them or past them, they are commonly vented by means of *vent pipes* which carry air into the house drainage system. Vent pipes from individual fixtures unite in the *vent stack* which rises vertically through the building from the lowest to the highest fixture. The vent stack parallels the soil or waste stack that it serves and is commonly connected with its drainage stack above the highest fixture. The combined stack passes through the roof into the atmosphere. A typical house plumbing system is illustrated in Fig. 107. A house trap which seals the entire house drainage system against the air in the street sewer was formerly required.

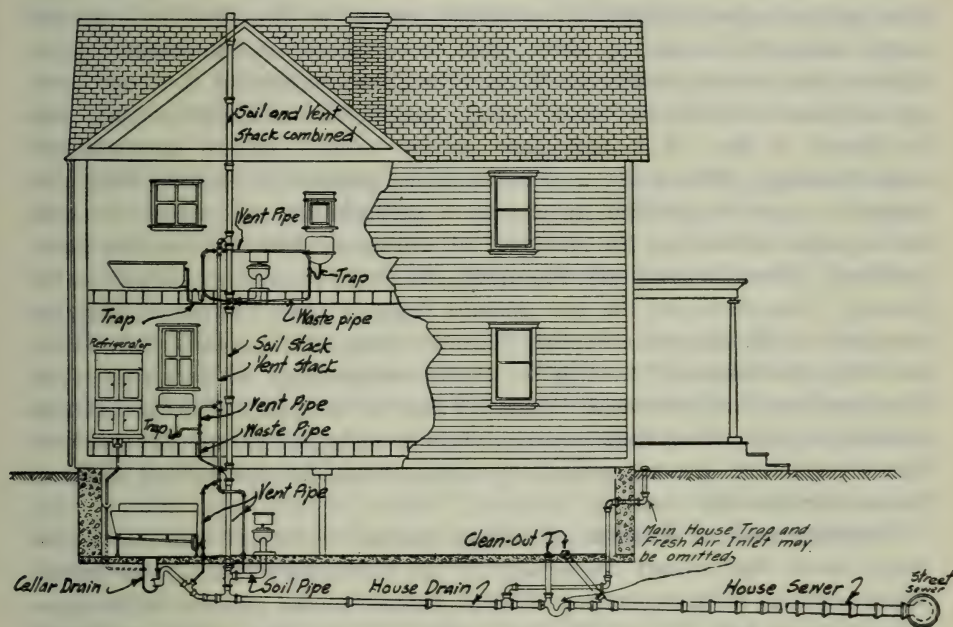


FIG. 107.—TYPICAL HOUSE PLUMBING SYSTEM.

(Adapted from Wisconsin State Plumbing Code)

To-day it is commonly omitted except in cold climates where cold air entering the sewer through the many house vents might result in frozen sewers.

Sewer-Gas.—The old bugaboo of sewer-gas that frightened our fathers before the days of bacteriology is no longer feared by sanitarians, although its influence is still firmly established in the popular mind and continues to pervade plumbing regulations. It is indeed desirable to keep the air of sewers from mixing with the air we breathe—the influence of all impure air should be avoided; but the danger of any one's becoming infected with the germs of disease by breathing sewer-gas is negligible. For sanitary reasons the plumbing system, besides being pneumatically tight, should also be tight against leakage of waste liquors and egress of cockroaches and other vermin that may find their way into the drainage system.

Sewerage Systems.—The wastes collected by the plumbing systems of houses are discharged into the city sewers. The sewers and drains of a city are used for various purposes, the two most important ones being the removal of domestic house sewage and the carrying away of rain water that falls on roofs, yards, sidewalks, and streets. Sometimes the same system of sewers is used to carry both domestic sewage and storm water. Then it is called a *combined system*. Sometimes the storm water is carried in relatively large drains or allowed to flow along in the street gutters, while the domestic sewage is carried in a *separate system* of sewers of smaller size.

Choice of System.—As a result of the evolution of sanitary sewerage from ground water drains, the combined system is the older and the one more commonly in use in older cities and crowded communities. It is cheaper than a dual system consisting of sanitary sewers for the house sewage and storm drains for storm water, except where the storm water can be allowed to flow off in the street gutters without serious inconvenience from flooding. Where the sewage must be pumped or carried long distances in pipes or purified by expensive methods the advantages lie with the separate system, as the quantity of sewage is less and its flow more constant. From the sanitary standpoint the separate system is more satisfactory. The choice of the two systems depends upon various engineering questions involving cost. It also depends upon certain sanitary questions involving the volume of flow in the stream courses into which excess storm flow from combined sewers is discharged and the uses to which these stream courses are put. Most state boards of health no longer permit the construction of combined systems without special investigation and serious study of the sanitary hazards involved.

Arrangement of System.—Sewerage systems consist of *house sewers* or house drains that convey the sewage to the street sewers or *lateral sewers*. These unite in what are termed *main* or *district sewers*, and the latter sometimes combine into one or more *trunk sewers* of large size. *Relief sewers* are sometimes built parallel to old sewers of inadequate capacity, and *underdrains* may be used in connection with the separate system to remove some of the ground water. *Intercepting sewers* are built parallel to streams in order to intercept the sewage and convey it to a safer point of discharge. In the combined system they are not designed to carry all of the flow at times of storm, but are provided with overflows, so that the excess storm water is discharged into the river at various points of overflow. They commonly carry about three times the dry weather flow. This is a matter of importance and one to be remembered in connection with the treatment of sewage, for the quantity of sewage that passes these overflows at times of heavy rain may exceed 50 per cent of the sewage, and during the course of the year may amount to 5 per cent, or even more, of the entire sewage of the city. Such overflow water is almost never treated before discharge and constitutes a serious danger to the users of water from the stream into which the mixed sewage and storm water overflows.

Quantity of Sewage.—The volume of sewage flowing in a separate system, or in a combined system during dry weather, does not differ materially from the water consumption of the city. In small towns it may be as low as 40 or 50 gallons per capita daily, although ordinarily it is more. In large cities it may amount to 100 to 200 gallons per capita or more. The amount of ground water entering the sewers depends upon the tightness of the sewer joints and varies from 5,000 to 100,000 gallons per day per mile of sewer.

Intercepting sewers are commonly designed to provide for a flow of 300 to 400 gallons per capita daily. The amount of storm water depends upon climatic conditions, and engineering treatises should be consulted on this subject. The flow of sewage fluctuates hourly, the maximum being from 50 to 100 per cent of the daily average. Greater fluctuations are sometimes found, especially in cities where large quantities of water are used for manufacturing purposes.

Ventilation and Flushing of Sewers.—The water carriage system offers practically no danger to the public health during the transmission of sewage. In many cities the sewers are ventilated by allowing a free flow of air from the sewers through the house drains, the individual house fixtures only being trapped.

The *catch-basins*, through which the street wash enters the sewers, are trapped against the egress of sewer air. The water that stands in them is a prolific breeding place for mosquitoes. Unless catch-basins are frequently cleaned, the accumulating organic matter putrefies and the resulting odor may be worse than that of the sewer air. Catch-basins are being omitted from some of the best designed modern sewerage systems. In separate storm sewers untrapped *street inlets* leading directly to the sewers are used rather than trapped catch-basins.

Combined sewers are sufficiently flushed by the storms. Separate sewers, if laid on proper grades, need little or no flushing. It has been common in the past to employ automatic flush tanks at the end of lateral sewers, but these are troublesome and waste much water. Hand flushing of sewers with fire hose is therefore frequently practiced.

Hygienic Results of Sewerage.—Statistics show that the abandonment of privies and the substitution of sewerage have reduced the general death rate in many a city. Thus Dr. Boobyer has reported that at Nottingham, England, in a period covering ten years typhoid fever cases occurred in 2.7 per cent of the houses that were provided with privies, in 0.83 per cent of the houses where pail closets were used, and in only 0.18 per cent of the houses that had water-closets connected with the sewers. Similarly, Dr. Porter has stated that in Stockport, England, during the years 1893-7 typhoid fever occurred in 3.4 per cent of the houses where there were privies, but in only 1.2 per cent of the houses that had sewer connections, these figures being based on a study of over 18,000 houses. In Munich, when sewers were constructed in 1856-1869 the typhoid fever death rate fell from 242 to 166 per

100,000; later, after an improved water supply and other sanitary reforms had been brought about, it fell to a much lower figure.

SEWAGE DISPOSAL

The sewage collected by sewerage systems is concentrated at one or more points depending upon the topography of the community and the method of disposal adopted. About 80 per cent of the sewered population of the United States discharges sewage into lakes, streams, or tidal waters in the crude state; the remainder subjects it to some form of treatment before discharging it into water or disposes of it on land. The problem of sewage disposal, therefore, divides into two parts: namely, disposal methods and treatment methods. Both involve a knowledge of the physical, chemical, and biological characteristics of sewage and the changes to which they are subjected.

Composition of Sewage.—A city's sewage consists of the public water supply soiled with the waste products of human life and refuse from household and factory, increased by a certain amount of ground water which leaks into the sewers, and, in the combined system, by varying quantities of rain water and street wash. Disintegrating and decomposing as it flows, the sewage gradually becomes a more or less homogeneous suspension of fine particles in water, together with organic and mineral matter in solution. The longer the sewage flows or stands, the more its constituents become disintegrated; fecal matter and paper become unrecognizable as such; bacteria increase enormously and assist in breaking down the complex organic compounds. The oxygen originally present in the water is reduced and finally disappears, so that from a fresh condition the sewage becomes first stale and then "septic." Mixed with the putrefying organic matter and the swarming hosts of bacteria harmlessly engaged in their beneficent work of destroying the organic matter, there may also be bacteria that have come from persons sick with typhoid fever, dysentery, cholera, or other diseases.

Sewage is obnoxious to the senses because of its decomposing organic matter, but it is dangerous to health because of the possible presence of pathogenic organisms.

The most important constituents of sewage from the standpoint of treatment are the nitrogenous and carboniferous organic materials carried either in suspension or solution, such as fecal matter, urine, cellulose, fats, and soaps. The concentration of these substances, that is, the amount present in a given volume of sewage, depends upon the per capita volume of the sewage and varies widely in different places. Somewhat more constant, however, are these constituents when compared with the number of persons living in houses connected with the sewers.

The following figures show the approximate constituents of sewage expressed in terms of grams per capita daily and in parts per million when the volume of sewage amounts to 100 gallons per capita daily.

ESTIMATED CONSTITUENTS OF AVERAGE SEWAGE

(After Fuller)

	Grams per Capita Daily *	Parts per Million †
Oxygen consumed (Permanganate test)		
2-minute boiling	15.0	39.6
5-minute boiling	22.0	58.0
Nitrogen		
Free ammonia	7.0	18.5
Albuminoid ammonia	2.5	6.6
Organic	8.0	21.1
Total	15.0	39.6
Chlorids	19.0	50.2
Fats	19.0	50.2
Dissolved matters		
Total	136.0	359.0
Mineral	99.0	261.0
Organic and volatile	37.0	98.0
Suspended matters		
Total	93.0	246.0
Mineral	53.0	140.0
Organic and volatile	40.0	106.0
Total Solids		
Total	229.0	605.0
Mineral	152.0	402.0
Organic and volatile	77.0	203.0
Bacteria—322 billion per capita daily		

* These figures also indicate parts per million if the per capita volume of sewage is 264 gallons per day.

† Assuming a per capita volume of 100 gallons per day.

The methods of sewage analysis are practically the same as those of water analysis (page 959). They are not in all respects satisfactory.

DISPOSAL METHODS

There are but two outlets for the sewage collected from cities and towns, namely, disposal upon land or into water. The first is known as irrigation; the latter, as dilution.

Irrigation.—The use of sewage for irrigating dry lands is more a treatment method than a disposal method and will be discussed again later. There are but few soils and climates that permit the use of sewage to irrigate crops under such conditions that all of the liquid is used up and does not at least in part pass from the soil into water courses. In arid regions with open soils, however, large volumes of sewage can be disposed of in this way. In wetter climates the sewage may first be discharged upon the ground but passes through it and is commonly collected in underdrains that lead into nearby water courses. Irrigation then becomes a treatment method and the disposal of the drainage from the irrigation fields must be considered further as a problem of dilution.

Dilution.—The discharge of sewage whether treated or not into streams, lakes, and harbors is by far the most common method of sewage disposal

throughout the world. It can be applied satisfactorily whenever the water receiving the sewage is capable of bearing the load placed upon it. When this is the case, the heavy particles settle to the bottom, the organic matter is oxidized by the oxygen dissolved in the water, and the bacteria are gradually dispersed, consumed by other organisms, killed by sunlight, or otherwise destroyed. These agencies bring about the phenomenon known as the self-purification of streams. (See page 1007.) When, however, the burden placed upon the water is too great, self-purification is retarded, water supplies may become polluted, oyster beds may become infected, and in severe cases streams may be so overloaded with sewage as to become offensive to sight and smell.

Dilution Required.—The minimum amount of water required to dilute raw sewage in streams is usually considered to be from 2.5 to 4 cubic feet per second for the sewage of one thousand people. The Chicago Drainage Canal was designed on the basis of 3.3 cubic feet per second for one thousand people. Rapidly flowing streams require less than this, as much oxygen is absorbed from the air; stagnant streams may need considerably more. The presence of trade wastes in the sewage may materially increase the dilution required. Oily wastes float on the surface and form scums that may interfere with the absorption of oxygen from the air. Wastes rich in organic matter, such as packing house or tannery wastes, consume much oxygen.

In this country no general dilution requirements governing the discharge of treated sewage into water courses have as yet been formulated. Some years ago the British Royal Commission on Sewage Disposal adopted the following figures for English practice:

<i>Condition of Sewage</i>	<i>Required Dilution (Volumes of Water to One Volume of Sewage)</i>
Crude	More than 500
Settled	300-500
Chemically precipitated	150-300

For lower dilutions the treatment plant effluent was required to contain less than 30 parts per million of suspended matter and have a biochemical oxygen demand of less than 20 parts per million in 5 days at 65° F. In studying these requirements, it must be remembered that English sewage is much stronger than American sewage.

The International Joint Commission on the Pollution of Boundary Waters between Canada and the United States based the permissible pollution of boundary waters upon their use for water supply purposes and adopted a standard of less than 500 *Bact. coli* per 100 c.c. as a yearly average. This corresponds to a required dilution of 4 cubic feet per second per capita, a figure about 1,000 times as large as that needed to prevent a nuisance.

Outfall Works.—The location and construction of sewer outfalls has an important bearing upon the success of sewage disposal by dilution. In order to be effective it is necessary to have the sewage thoroughly and quickly

diffused through the water. In lakes, furthermore, the relation between the sewer outfall and the intake of the waterworks must be carefully considered, and the dispersion of bacteria by currents induced by the wind and temperature must be studied. In coastal wastes the effects of the tides must be taken into account.

The change in practice governing sewage disposal by dilution is well illustrated in the three outfalls of the sewerage systems of Metropolitan Boston. The oldest system, built in 1884, discharges the sewage on the surface of the water during ebb tide. The sewage, which is warmer and lighter than the sea water, rides on top of it, does not mix readily, and forms a "sleek" which can be noticed for many miles, and becomes quite offensive during hot weather. Sea gulls feed upon the waste materials and may possibly fly from the floating sewage fields to the water reservoirs of nearby towns. No direct contamination of the latter, however, is in evidence.

The South Metropolitan outfall, built in 1905, discharges through two submerged outlets at a depth of 24 to 30 feet below mean low water. The sewage tends to rise and is distributed through the water. It is difficult to determine the location of the outfall by casual observation of the water surface. This becomes even more so at the North Metropolitan outfall, completed in 1918, which discharges through 14 outlets at a depth of 30 to 50 feet below mean low water. The dilution thus secured is very great and discharge is continuous instead of being intermittent as in the case of the oldest outfall. Even chemical tests indicate but slight traces of sewage in the surface layers of water above the disposal grounds.

Recently the outfall of the Passaic Valley Sewerage System in New York Harbor has provided even greater facility for mixing the sewage and sea water. Here the sewage is discharged at a depth of 40 to 50 feet through 150 special distributing nozzles situated on pipes branching over several acres of harbor bottom.

Stream Pollution.—The discharge of sewage into water sets into motion a series of physical, chemical and biological reactions that are more or less interdependent. These must be studied in order to establish the sanitary hazards involved in dilution.

Biological Considerations.—The problem of sewage disposal by dilution is largely a biological one. The decomposition and oxidation of the organic matter in sewage are brought about by bacteria, and the bacteria serve as food for protozoa and other forms of microscopic animal life. The dissolved organic matter in sewage serves as food for algæ. These algæ and protozoa are, in turn, consumed by rotifers and crustacea, while the latter form the basis of the food supply for various aquatic animals and fishes. Thus there is a continuous biological cycle. Again, animal forms require oxygen and produce carbonic acid, while plants consume carbonic acid and produce oxygen.⁶ Where these processes occur normally and with a proper equilibrium

⁶ When fish die in sewage-polluted water, it is usually due to lack of oxygen.

maintained between animal and plant life, oxygen, nitrates, carbonates, and other chemical food substances, offensive conditions do not result, but where abnormal conditions are produced, as, for example, by the discharge of excessive quantities of sewage or trade wastes into a stream, a depletion of the dissolved oxygen may follow, or there may be an overproduction of algæ, so that the conditions become offensive. The chemical and biological changes taking place in a sewage polluted stream are illustrated by the figures in the following table:

CHEMICAL AND BIOLOGICAL CHANGES IN GENESEE RIVER BELOW THE SEWERS OF ROCHESTER

(Summer, 1912)

MILES BELOW SEWERS	ORGANISMS PER C. C.				DIS- SOLVED OXYGEN PER- CENTAGE SATU- RATION	PARTS PER MILLION			
	Bacteria	Protozoa	Algæ	Roti- fera & Crus- tacea		Free N	Nitrites	Ni- trates	Total Org. N
0	1,650,000	209	156	57	27
1.5	1,823,000	1,786	310	67	6
3.5	123,000	206	436	213	21	1.26	.014	.00	.84
5.0	129,000	136	336	422	20
5.5	67,000	77	363	233	37	.78	.006	.05	.23

Dissolved Oxygen.—The amount of oxygen dissolved in water depends largely upon its temperature. Water near the freezing point will hold nearly twice as much oxygen (14.70 p. p. m.) as at prevailing summer temperatures (7.74 p. p. m. at 29° C.). The dilution required in summer is therefore greater than in winter; and in some situations it would be logical to construct treatment works to be operated during the summer only, thus making a material saving in cost. Sea water dissolves about 20 per cent less oxygen than fresh water.

Water exposed to the atmosphere absorbs oxygen readily. The lower the concentration of oxygen in the water the more rapidly does it replenish its supply from the air. This is called re-aëration and progresses at a more rapid rate if new surfaces of water are brought into contact with the source of oxygen. This explains why running streams can absorb more oxygen than quiescent bodies of water.

Hygienic Aspects of Stream Pollution.—Considering the hygienic aspects of stream pollution with special reference to the pollution of water supplies, it is important to remember that typhoid fever bacilli do not multiply in the ordinary water of our streams, but, on the contrary, when discharged into water they diminish in number. After a week not more than 10 per cent may remain alive, and after a month not more than 1 per cent.

It follows that recent pollution is the most dangerous, and that water stored in reservoirs and lakes becomes more and more safe for use as time

of storage increases. The longevity of the typhoid bacillus is much greater in cold water than in warm water. Hence, water-borne typhoid fever epidemics are more common in winter than in summer, and in northern climates than in southern climates.

Legal and Economic Aspects of Stream Pollution.—Whereas it is true that hygienic and sanitary considerations materially affect the use of rivers and waterways as vehicles for the reception, transmission, and ultimate disposal of sewage, the question is primarily an economic one. The power of streams to transport suspended matter and the ability of natural bodies of water to oxidize and destroy offensive substances represent a natural resource that should be utilized just as far as this can be done with safety and without offense. For each river there is a limit to the amount of permissible pollution. The reasons for this limit are not the same in all cases, but vary according to the use that is made of the water of the river, and no universal standard can be wisely set up or maintained. When the extent of the pollution is such as to affect public health in any way by any reasonable use of the river the sanitary aspect of the situation should control.

Protection against Pollution.—Long experience in this country and abroad has demonstrated clearly and unmistakably that it is possible to purify polluted water to such an extent that it will be reliably wholesome. Some waters, however, are so grossly polluted that the load upon the purification works would be too great to produce an hygienically safe water by the ordinary economical treatment methods. When this is the case, it is usually better to seek a less polluted supply or to require treatment of the sewage or industrial wastes that are polluting the supply. This is a problem in coöperative sanitation that will be touched upon later.

TREATMENT METHODS

By appropriate processes sewage can be treated so that the decomposable organic matter is removed or oxidized and the bacteria removed or killed. A complete purification is not attempted even in the best-conducted plants, as the processes demanded would be too elaborate, too expensive, and too uncertain of results. More often the purification is incomplete, the degree of purification secured being adjusted to the particular needs of the situation. In the past sewage treatment works have been built to remove as much of the decomposable organic matter as was necessary to enable the effluent to be discharged into some waterway without causing offensive conditions. This was the case in Europe, and especially in England, where the streams are relatively small, the cities relatively large, and the amounts of trade waste considerable. In some places greater emphasis has been placed on the removal or destruction of pathogenic bacteria, with the object of protecting oyster beds, bathing beaches, or reducing the "load" on water filters. The degree of treatment thus required varies all the way from a nearly complete purification down to a mere straining out of the grosser solids.

Fundamental Principles of Sewage Treatment.—The fundamental processes in sewage treatment are:

1. Separation of the suspended matter from the liquid sewage.
2. Destruction of the putrescible organic matter in the liquid sewage looking to final mineralization by the processes of oxidation and bacterial action.
3. Transformation of the sewage sludge to a condition of stability and inertness by bacterial action, with or without oxidation.
4. Destruction or removal of the bacteria from the liquid effluent.

The processes involved may be classified as follows:

1. Preparatory processes, such as screens, grit chambers, plain settling tanks, septic tanks, digestion tanks, and chemical precipitation tanks.
2. Key processes, such as subsurface irrigation, broad irrigation, intermittent filtration, contact beds, trickling filters, and activated sludge.
3. Finishing processes, such as secondary sedimentation, disinfection, and dilution after treatment.
4. Sludge treatment, such as digestion, air drying, and mechanical dewatering.
5. Sludge disposal.

The processes are by no means clear-cut. They overlap at many points; they are used singly or in all sorts of combinations.

Preparatory Processes.—The object of most preparatory processes of sewage treatment is the removal of those suspended solids that are readily separated from the sewage by mechanical means, namely, screening or settling.

SCREENING.—There are two types of sewage screens: (1) coarse screens and (2) fine screens.

1. *Coarse Screens.*—These consist of gratings of iron bars seldom less than $\frac{1}{2}$ inch apart which hold back only the largest floating objects found in sewage, such as sticks, paper and rags. They are used chiefly in connection with sewage pumping stations in order to prevent injury to the pumps, but are also employed in treatment works. Two types of coarse screens are widely used: racks placed in an inclined position in the channel of sewage flow and cages lowered in duplicate vertically into the sewage, one being removed while the other is being cleaned.

2. *Fine Screens.*—Fine screens are made of wire mesh or perforated metal plates with a clear opening of seldom more than $\frac{1}{4}$ inch by 2 inches in size. They retain many of the smaller sewage particles, including some fecal matter. Fine screens are employed on outfalls discharging untreated sewage into rivers, lakes, and harbors, and prevent the accumulation of unsightly litter on the water and beaches. They are also used in sewage treatment works to remove substances that might interfere with the operation of the other treatment processes or form a scum upon the settling tanks.

Fine screens clog rapidly and require special mechanisms to keep them clean. A great many different types of screens have been devised. The two most widely known in this country are the Riensch-Wurl screen illustrated in Fig. 108 and the Dorr screen. The former consists of a perforated disk surmounted by a truncated cone, which rotates on an inclined axis, the disk being swept by brushes as it emerges from the sewage. The latter consists of a partially submerged perforated cylindrical drum revolving horizontally through the sewage which passes through the openings into the drum and flows out through a discharge opening in one end. The Dorr screen is self-cleansing, the screenings adhering to the outside of the drum being forced into a pit by the difference in water pressure between the inside and outside of the drum. Other types of screens are continually being introduced.

The amount of material screened from sewage varies from 1 to 30 cubic

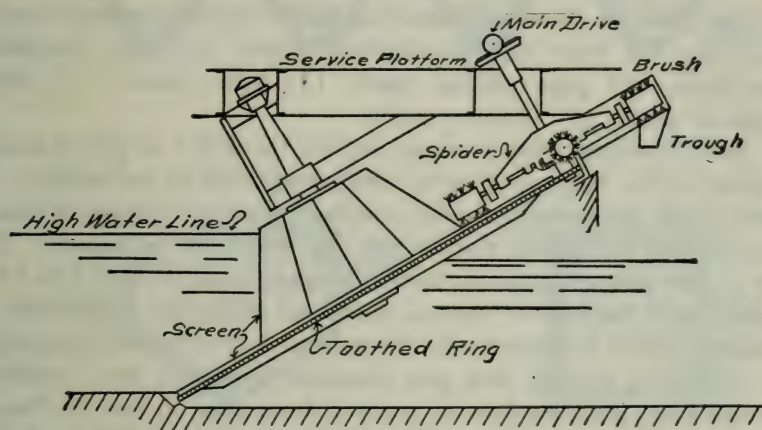


FIG. 108.—RIENSCH-WURL SCREEN.

feet per million gallons of sewage, according to the fineness of the screens and the composition of the sewage. Screenings are pressed, burned under a boiler, buried in land, or turned over to the local garbage and refuse department for disposal.

SEDIMENTATION.—Sedimentation or settling is the most important preparatory process in sewage treatment. Almost 40 per cent of the total solids contained in sewage are carried in suspension; more than half of these will settle out readily when the sewage is allowed to remain quiescent or when it flows at very low velocities. Sedimentation tanks are operated on the fill and draw principle, with horizontal movement of the sewage or vertical flow. They are used in combination with filters to decrease the load on the latter and to prevent the clogging of nozzles or other fine apertures through which the sewage may have to flow. They are also employed after trickling filter or activated sludge treatment to retain the solids unloaded by the filters or activation tanks. Finally, they are often used without other treatment methods to lessen the amount of suspended solids discharged into bodies of water.

The following figures show the approximate percentage of suspended matter removed by sedimentation.

PERCENTAGE REMOVAL OF SUSPENDED MATTER

Type of Sedimentation	Period, Hours	Weak Sewage, per Cent	Medium Sewage, per Cent	Strong Sewage, per Cent
Grit, or detritus tanks	1	10	15	25
Plain sedimentation	2	40	50	65
Also Imhoff tanks	4	45	60	70
Plain or septic sedimentation	12	45	60	75
Septic sedimentation	24	50	65	80
Septic sedimentation	48	50	75	85
Chemical precipitation	4	40	70	95

The sludge obtained from sedimentation tanks requires further treatment.

There are five types of sedimentation basins: (1) grit chambers, or detritus tanks; (2) plain settling tanks; (3) septic tanks; (4) chemical precipitation tanks; (5) two-story tanks.

1. *Grit Chambers*.—As the name implies, the object of grit chambers is the removal of the mineral or gritty solids suspended in the sewage. They are used chiefly in connection with combined sewerage systems where they remove the sand and gravel washed into the sewers during storms. If the velocity of flow through grit chambers is properly controlled (0.5 to 1.0 feet per second) only the heavier mineral substances will settle, but usually sufficient organic matter is trapped with the mineral matter to make the resulting sludge offensive. Vertical flow grit chambers have also been constructed. The sludge from grit chambers is removed from time to time by hand, by mechanical sludge elevators, or by hydraulic means. It is then used for fill or is buried. The amount of sludge collected varies greatly.

2. *Plain Settling Tanks*.—Settling tanks are called "plain" when the force of gravity alone produces the clarifying of the sewage. In order to permit this force to operate the velocity of flow must be checked to reduce the carrying power of the water. Velocities less than two feet per minute are employed, the time of detention varying from thirty minutes to twelve hours. Detention periods greater than four hours are hardly ever economical. Sludge is removed at frequent intervals in order to prevent bacterial decomposition. Typical horizontal and vertical flow tanks are shown in Figure 109 A, B.

3. *Septic Tanks*.—As previously described, the septic process depends upon anaërobic bacterial action, besides physical sedimentation. Septic tanks, therefore, are commonly designed to retain the sewage and the sludge for longer periods than plain settling tanks. The period of detention of the sewage varies from eight to twenty-four hours or longer and the sludge is allowed to remain in the tank for long periods of time, giving opportunity for anaërobic bacterial action after the dissolved oxygen in the sewage has been used up. Septic action liquefies and gasifies some of the organic

matter and the amount of sludge to be handled is reduced. This process is spoken of as digestion. It is accompanied by the presence of a scum on the surface of the tank and a continual rising and falling of sludge through the liquid. The amount of solid organic matter thus digested varies from 10 to 40 per cent, being greatest in strong domestic sewage. Septic action does not materially improve the quality of the effluent. It may in fact make it more objectionable. Septic action cannot be depended upon to render

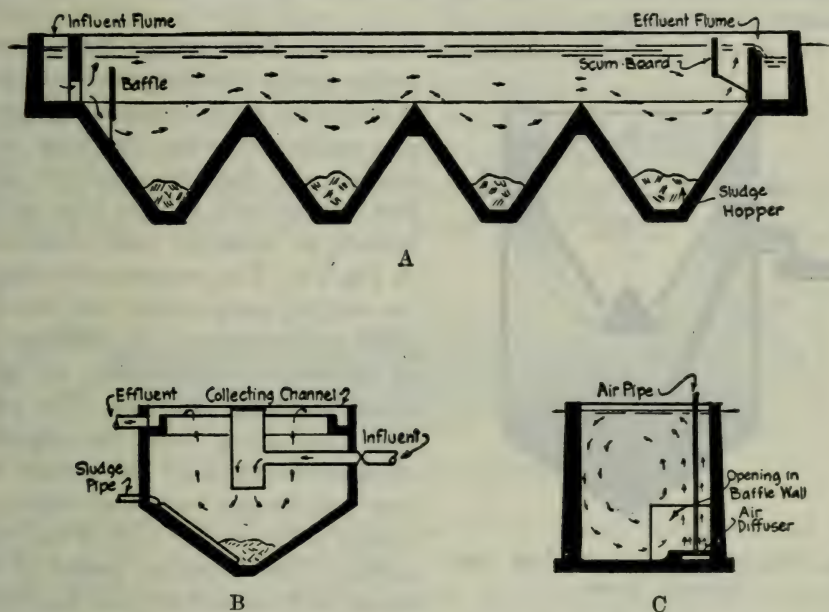


FIG. 109.—A. PLAIN SEDIMENTATION TANK. HORIZONTAL FLOW. B. PLAIN SEDIMENTATION TANK. VERTICAL FLOW. C. ACTIVATED SLUDGE TANK. SPIRAL FLOW.

sewage safe so far as infection is concerned. The structural design of septic tanks does not differ materially from that of plain settling tanks.

4. *Chemical Precipitation.*—Sedimentation may be hastened and increased by the use of chemicals. Lime (calcium oxid), copperas (ferrous sulphate), and alum (aluminum sulphate) are commonly used to coagulate the suspended matter by forming flocculent masses of iron or aluminum hydroxids. The process is similar to that used for the removal of color and turbidity from drinking water. When the sewage itself contains the necessary amount of iron—usually derived from the waste liquors of iron pickling processes—lime only is needed. From 500 to 1,500 pounds of alum are required per million gallons of sewage treated. The amounts of lime and copperas used per million gallons of sewage also vary. There is used at London 500 pounds of lime and 120 pounds of copperas; at Providence, 600 pounds of lime and no copperas; at Glasgow, 600 pounds of lime and 1,000 pounds of copperas.

The acidifying of sewage with sulphuric acid or, better, sulphurous acid fumes obtained by burning sulphur or roasting pyrite and the subsequent precipitation of the solids, known as the *Miles Acid Process*, has attracted attention because the bulk of sludge produced is less than by any other means of precipitation, and furthermore because the sludge lends itself readily to the recovery of grease as a by-product of the process. In England sulphuric acid has been employed successfully for the purpose of recovering grease from the sewage of wool-scouring towns. The process produces a certain measure of disinfection.

5. *Two-Story Tanks*.—In order to separate fresh sewage from decomposing sludge two-story tanks are used in which the upper compartment serves as a settling chamber, the lower as a storage and digestion chamber. The best known tank of this type is the Imhoff, or Emscher tank, illustrated in Fig. 110. The processes obtaining in the settling compartment are those of plain sedimentation; the ones acting in the sludge compartments are those of septic sludge digestion. The gases and scum rising from the digestion chamber do not enter the sewage, but pass into the gas vents that commonly occupy 10 to 20 per cent of the tank surface. The detention period of the sewage is from one to three hours, the velocity of flow being in the neighborhood of 1.5 to 2.5 feet per minute. The sludge storage required varies with the climate from 1 to 2 cubic feet per capita.

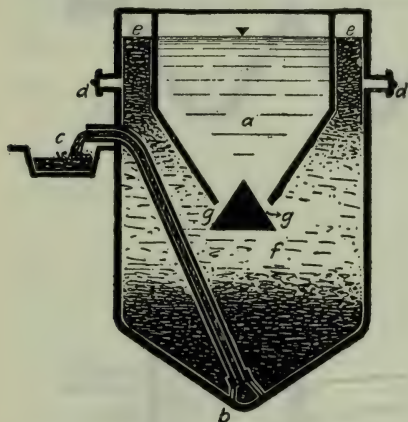


FIG. 110.—TYPICAL SECTION OF AN IMHOFF TANK.

- a. Compartment for flowing sewage.
- f. Sludge digestion compartment.
- g. Baffle to prevent gases and sludge from rising into compartment a, but permitting sediment to fall into the sludge compartment. b-c. Pipe for withdrawing sludge.

The processes obtaining in the settling compartment are those of plain sedimentation; the ones acting in the sludge compartments are those of septic sludge digestion. The gases and scum rising from the digestion chamber do not enter the sewage, but pass into the gas vents that commonly occupy 10 to 20 per cent of the tank surface. The detention period of the sewage is from one to three hours, the velocity of flow being in the neighborhood of 1.5 to 2.5 feet per minute. The sludge storage required varies with the climate from 1 to 2 cubic feet per capita.

Key Processes.—The effluent from sedimentation tanks still contains relatively large quantities of putrescible organic matter in suspension and solution. The transformation of these materials into stable compounds that will not exceed in their demand for oxygen the supply provided in the water courses into which they are discharged, is the object of the key processes of sewage treatment. The methods developed to accomplish this rely chiefly upon oxidation and filtration in which biological processes play a significant part. Historically they can be arranged in the following order: broad irrigation; subsurface irrigation; intermittent sand filtration; contact beds; trickling filters; activated sludge: each of which has its uses.

Broad Irrigation.—Broad irrigation, also known as “sewage farming,” consists in the application of crude or settled sewage to land, making it

serve as food for crops, the principal value, however, being in the water itself. It is distributed by means of ditches and other channels as in ordinary irrigation. The sewage farms of Berlin and Paris are very extensive, the Berlin farms covering over 20,000 acres. The rate of application varies from 3,000 to 15,000 gallons per acre daily, an acre serving for the sewage of from 100 to 300 persons. The crops raised on sewage farms frequently pay the expenses of operation, but seldom pay the interest on the investment except in arid regions, where irrigation is profitable. Broad irrigation cannot be successfully used with clayey soils. The purification obtained is usually very satisfactory, both chemically and bacteriologically.

Subsurface Irrigation.—For small installations a satisfactory method of disposing of sewage after sedimentation is to discharge it through 3-inch or 4-inch tile pipes laid in the ground 10 to 18 inches deep in rows $2\frac{1}{2}$ to 3 feet apart. In sandy soils this method gives satisfaction, and under favorable conditions the sewage of 150 to 250 people can be applied to an acre, the

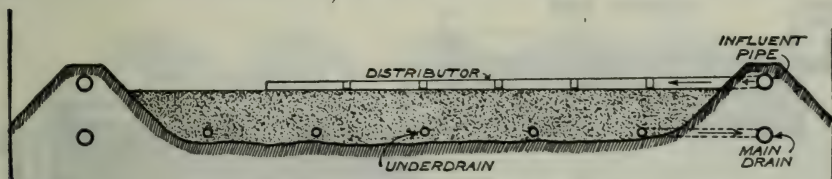


FIG. 111.—CROSS SECTION OF INTERMITTENT SAND FILTER.

rate of application being commonly one to two gallons per lineal foot, or 20,000 to 30,000 gallons per acre daily. With tight soils larger areas are required. With clay soils the method cannot be used. This method of sewage disposal is particularly applicable to suburban and rural conditions. (See Fig. 101.)

Intermittent Sand Filtration.—With this method the sewage is applied intermittently to beds of sand, especially prepared for the purpose, in such quantities that it quickly soaks away, leaving the bed exposed to the air for a period of several hours or several days, thus giving opportunity for aëration and oxidation of the organic matter. The results obtained are usually very satisfactory, provided that the filters are not overloaded. When raw sewage is applied directly to the beds the rates of application vary from 50,000 to 150,000 gallons per acre daily, the population served per acre being from 300 to 1,200. With preliminary treatment higher rates may be used and the sewage of 1,500 to 2,000 people applied per acre. The filtration area is usually divided into beds by means of earth embankments which cover the distributing pipes. Often they are underdrained with tiles laid 20 to 30 feet apart in fine material, or 100 feet apart in coarse material, their depth below the surface varying from 3 to 8 feet. After a few weeks or months the beds become clogged and it is necessary to rake the surface. At intervals the

accumulated deposit on the sand has to be scraped off. In winter the beds are plowed into ridges or the sludge is collected into piles so that ice may form and be supported upon them, leaving channels beneath the ice by which the sewage can be distributed.

The efficiency of intermittent sand filtration is higher than that of any other process. Well-operated plants are capable of removing from 95 to 98 per cent of the suspended matter and bacteria and the effluent is quite clear and non-putrescible. The method is limited, however, to regions where suitable and convenient areas of sandy soil exist.

Contact Beds.—Contact beds are water-tight compartments filled with porous material, such as broken stone or coke, and operated as follows: The bed is slowly filled with sewage, which has previously passed through a settling tank. It is then allowed to remain full for a brief period, after which it is emptied and allowed to remain empty for a longer period. A

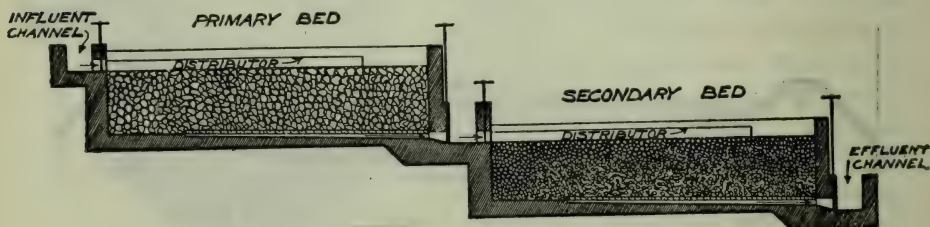


FIG. 112.—CROSS SECTION OF CONTACT BED.

cycle commonly employed is to allow one hour for filling, two hours for contact, one hour for emptying, and four hours for rest. During the period of contact the suspended matter tends to settle upon and adhere to the exposed surfaces of the broken stone or coke, thus forming a film. While standing full septic action occurs and organic matter is absorbed by the film. During the resting period oxidation of this organic matter takes place. The purification obtained in this way is partial. Commonly, two or three contact beds are used in series, the effluent from the first passing to the second, and that of the second to the third. The depth of contact beds varies from 2 to 6 or 8 feet, the broken stone or coke being from $\frac{1}{2}$ inch to 2 inches in size. The rate of application is usually between 300,000 and 800,000 gallons per acre daily, one acre serving a population of about 5,000. When properly operated with settled sewage, contact beds are capable of removing about 65 to 70 per cent of the organic matter, 80 to 85 per cent of the bacteria, and 85 to 90 per cent of the suspended matter. Contact beds become clogged with use, and after periods varying from five to eight years it is necessary to remove the stone or coke and clean them. This method is no longer being used for larger communities but is well adapted to treat the sewage from villages, institutions, and other large-size isolated buildings.

Trickling Filters.—Trickling filters, otherwise called “sprinkling filters” or “percolating filters,” consist of beds of porous material such as broken stone, coke, or clinkers, upon which the sewage is sprinkled and through which it percolates to underdrains laid on a tight floor beneath. The entire bed is arranged with reference to complete aëration throughout, in order that the organic matter of the sewage may become thoroughly oxidized. The suspended matter of the sewage is not permanently retained in the beds, but is carried out in the effluent, which is turbid and requires subsequent clarification. This is called the “unloading” of the bed. The object of the trickling filter is to change the character of the organic matter so as to render it non-putrescible. The sewage is applied to the beds by sprinkling through fixed sprinklers or by use of traveling sprinklers, rotary or rectangular, operated by the discharging sewage or by power. The rate of application varies from 0.5 to 2.0 million gallons per acre daily, one acre of trickling filter serving a population of 10,000 or more. The beds vary in depth from 5 to 10 feet, coarser material being used for the deeper beds. Sprinkling filters

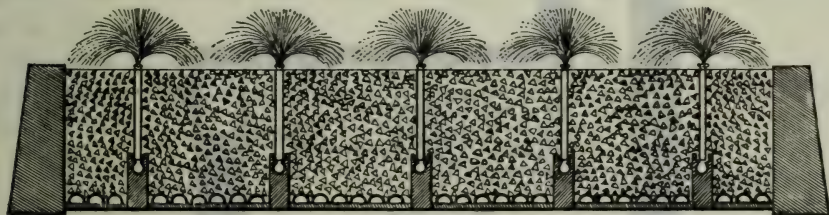


FIG. 113.—TYPICAL SECTION OF A SPRINKLING FILTER.

are commonly operated intermittently in order to permit thorough aëration of the bed. They require specially designed dosing chambers or mechanical devices to secure intermittent dosage. The cycle used varies greatly, a dosing period of several minutes alternating with a resting period of equal or greater length of time. Well-operated sprinkling filters receiving the effluent from plain sedimentation or septic tanks are capable of removing from 85 to 90 per cent of the suspended matter and from 90 to 95 per cent of bacteria, yielding an effluent that is non-putrescible. The effluent from trickling filters is not always clear and should be passed through settling basins before discharge. These basins are known as “humus tanks.”

Activated Sludge.—If sewage standing in a tank is brought into intimate contact with air so that aërobic conditions are maintained throughout the liquid, the particles of suspended matter after a time flocculate into masses of sludge swarming with microscopic life and capable of oxidizing organic matter readily. This sludge is known as “activated sludge.” After this condition has been reached, sewage can be passed through the tank and a surprising clarification, nitrification, and reduction in bacterial content is obtained providing aërobic conditions are maintained and sufficient quantities of activated sludge are intimately mixed with the traveling sewage. The bacterial reduction lies between 90 and 98 per cent. In principle the process

resembles that of aëration and filtration, the sand grains or stones of sewage filters being replaced by sludge particles suspended in the liquid.

Aërobic conditions and mixture can be obtained in two ways. Finely divided air can be forced through the tank by the use of perforated pipes, diffusers, and other devices, or the sewage can be agitated mechanically, new surfaces of liquid being brought into contact with the atmosphere from which they absorb the required amount of oxygen. The first is termed *aëration*; the latter has been called *bio-aëration*. In both cases the sludge is kept in suspension throughout the liquid. The volume of activated sludge required is about 20 per cent of the volume of sewage. The time of retention varies

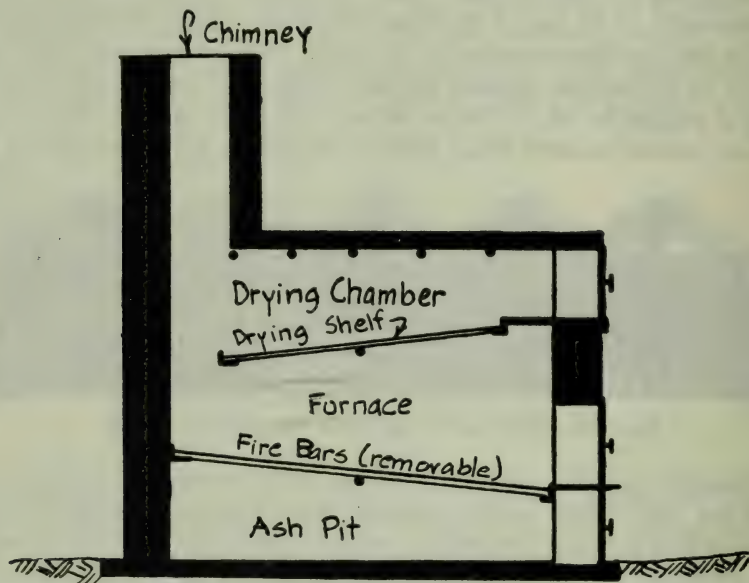


FIG. 114.—DESTRUCTOR SUITABLE FOR CAMPS, ISOLATED DWELLINGS AND SMALL COMMUNITIES.

from four to six hours. An aëration tank is shown in Fig. 108. In activation by aëration the ratio of diffuser area to tank area varies from 1:4 to 1:10, the amount of air used from 1 to 2 cubic feet per gallon of sewage. In bio-aëration tanks a velocity of 1.5 feet per second is sufficient to keep the sludge in suspension and will permit surface aëration providing the tanks are so designed that the sewage is well mixed during travel. The excess sludge is permitted to pass out of the tanks together with the treated sewage and is then removed by sedimentation, vertical settling tanks being used quite commonly. (See Fig. 109 (b).)

Finishing Processes.—As pointed out above, some of the key processes of sewage treatment produce an effluent that still contains much suspended matter even if in well-oxidized form. They require therefore subsequent sedimentation in what are termed *secondary sedimentation tanks*. These are

commonly plain settling tanks of the horizontal or vertical flow variety. Since the material that reaches these tanks is well flocculated, the time of retention required is small.

None of the processes so far described can be relied upon to furnish an effluent free from pathogenic organisms, despite the relatively high degree of purification obtained by the best processes. In order to protect oyster beds, waterworks interests, or bathing beaches, therefore, sewage is sometimes disinfected.

Disinfection is obtained by means similar to those used in water purification. Liquid chlorin and chlorid of lime are the most common chemicals used. The quantities of liquid chlorin required per million gallons of sewage are 40 pounds for intermittent sand filter effluents, 80 pounds for contact bed and sprinkling filter effluents, 120 pounds for settling tank effluents, and 160 pounds for raw sewage. The time of contact that should be permitted in order to allow the disinfectant to take effect varies from one-half hour to two or more hours. From 95 to 99 per cent of the bacteria can be destroyed.

Relative Bacterial Efficiency of Different Processes.—By way of recapitulation the following figures are given to show the relative sanitary efficiency of various processes employed in sewage treatment:

<i>Process</i>	<i>Percentage Removal of Bacteria</i>
Coarse screens	0 to 5
Fine screens	10 to 20
Grit chambers	10 to 25
Sedimentation	25 to 75
Septic sedimentation	25 to 75
Chemical precipitation	40 to 80
Contact beds	80 to 80
Trickling filters	90 to 95
Activated sludge process.....	90 to 98
Intermittent sand filters.....	95 to 98
Broad irrigation	97 to 99
Disinfection of raw or settled sewage.....	90 to 95
Disinfection of filter effluents.....	98 to 99

These figures are mere approximations, but they serve to show how some forms of treatment, very desirable from many points of view, have a low hygienic efficiency. Septic treatment, for example, does not greatly reduce the number of bacteria in sewage; in fact, if the period of detention of the sewage in the tank is long the number of bacteria in the effluent may be greater than those in the raw sewage.

Choice of Sewage Treatment Methods.—The choice of treatment methods to be used in any given case depends upon various considerations, such as the nature of the sewage to be treated, the allowable character of the effluent considered with reference to the use made of the water into which it

is to be discharged, the availability of suitable areas of land at proper elevation, and finally the cost of construction and operation.

The most common combinations of sewage treatment methods employed in the large cities of the United States are shown in the following schedule:

UNITED STATES SEWAGE TREATMENT PLANTS IN CITIES OVER 100,000

(After Pearse)*

DISTRICT	TYPE OF PLANT		
	Screen	Tank	Filter
Sanitary District of Chicago			
Des Plaines	Bar and Fine, G †	Activated sludge	Trickling
Morton Grove	Imhoff
Calumet	Bar, G	Imhoff
Philadelphia, Pennypack Creek	Bar	Imhoff	Trickling
Cleveland			
Westerly	Bar, G	Imhoff
Easterly	Bar, G
Baltimore	Bar	Hydrolytic	Trickling
Rochester			
Irondequoit	Bar and Fine, G	Imhoff
Brighton	Bar, G	Imhoff	Trickling
Charlotte	Bar, G	Imhoff
Providence	Bar	Chemical pre- cipitation ‡
Columbus	Bar	Imhoff	Trickling
Akron	Bar, G	Imhoff	Trickling
Atlanta			
Proctor Creek	Bar, G	Imhoff	Trickling
Intrenchment Creek	Bar, G	Imhoff	Trickling
Peachtree Creek	Bar, G	Imhoff	Trickling
Worcester (old plant)	Chemical pre- cipitation Sedimentation	Intermittent sand
Birmingham			
Valley Creek	Septic contact	Septic contact
Ensley	Sedimentation
San Antonio §
Dallas	Bar, G	Imhoff
Bridgeport	Fine screen
Houston			
North Side	Bar, G	Activated sludge
South Side	Bar	Activated sludge
Albany	Bar, G	Imhoff
Reading	Septic	Trickling

* From Part II, *Hearings on the Improvement of the Illinois and Mississippi Rivers*.

† "G" under screen column indicates grit chamber.

‡ Disinfected.

§ Irrigation by contract.

SEWAGE SLUDGE

Sludge is the principal by-product of sewage treatment. Excepting screenings and the deposits obtained in grit chambers, which are not commonly called sludge, it consists of the solids removed by the different treatment processes. The sanitary disposal or utilization of sludge presents one of the most troublesome problems associated with the management of sewage works.

The sludge obtained by different sewage treatment methods varies greatly in bulk, chemical composition, and physical characteristics. The following table ⁷ may serve for general comparison.

NORMAL VOLUME OF SLUDGE OBTAINED BY DIFFERENT PROCESSES OF SEWAGE TREATMENT

Treatment Process	Normal Volume of Sludge per Million Gallons of Sewage Treated, Gallons	Percentage of Solids in Sludge	Specific Gravity of Sludge	Weight of Dry Solids in Sludge per Million Gallons of Sewage Treated, Pounds
Activated sludge	10,000	2.00	1.005	1,675
Chemical precipitation	5,000	7.50	1.040	3,250
Sedimentation	2,500	5.00	1.020	1,060
Septic tank	500	5.00	1.040	220
Imhoff tank	500	15.00	1.070	670
Trickling filter, humus tank.	500	7.50	1.025	320

The relatively large volumes of sludge produced by the activated sludge process are due to its extreme watery consistency. The high weight of chemically precipitated sludge is accounted for by the presence of the precipitated chemicals.

Sludge Treatment.—Some of the sewage treatment methods incorporate integrally certain processes of sludge treatment. Others require special provisions in order to turn out materials that can be disposed of economically. There are a number of treatment processes which can be grouped as follows: digestion; thickening; drying; mechanical drying without heat; heat drying.

Digestion.—The sludge precipitated by sedimentation in septic tanks and two-story tanks undergoes anaërobic decomposition before removal. In large septic tanks the period of decomposition allowed is relatively short; in small septic tanks of the home sewage disposal variety and in Imhoff tanks it is much longer. The degree of digestion depends upon the length of storage and the temperatures to which the sludge is exposed. The optimum temperature is in the vicinity of 25° C. At lower temperatures digestion is much slower, being inhibited at about 6° C. For this reason digestion compartments of Imhoff tanks commonly provide storage capacity for the sludge accumulated during the winter months. Well-digested sludge is inoffensive and dries rapidly. The gases trapped in the sludge assist in drying. The storage capacity required in northern climates is stated as 1 cubic foot per capita in separate systems and 2 cubic feet in combined systems. In southern climates this volume can be reduced.

Fresh sewage sludge is very offensive and does not dry quickly. Plain sedimentation tanks therefore are sometimes provided with *separate sludge chambers* into which the settled sludge is drawn from time to time. The sludge tanks, which lie next to the settling tanks instead of beneath them

⁷ H. P. Eddy, *Public Works*, Vol. 51, pp. 112-115, 130-133.

as is the case in two-story tanks, are sometimes divided into two compartments. In the first the sludge undergoes fermentation; in the second, digestion. The final product is in all ways similar to Imhoff tank sludge. The sludge from trickling filter humus tanks is sometimes lifted into sludge digestion tanks for further treatment.

A relatively new development in sludge digestion is the utilization of the gases produced during digestion. The gases contain much methane. This combustible gas is used in some cases to produce part or all of the power and heat needed in the treatment works; in others it is conducted into the gas mains of the community.

Thickening.—All sludges are extremely watery. As shown in the above table, the most concentrated of them (Imhoff sludge) contains 75 per cent of water, and the most watery (activated sludge) 98 per cent. In order to thicken the watery sludges mechanical thickeners are sometimes employed. They commonly consist of helical scrapers or plows that revolve about a vertical arm and move the settling sludge towards the center of the tank, compacting it during travel. The dewatering accomplished, although relatively small, is often sufficient to make the process economical, for it must be remembered that the reduction of a 98 per cent sludge containing but 2 per cent of solid matter to a 96 per cent sludge containing 4 per cent of solid matter signifies that the volume of sludge to be handled has been cut in two.

Air Drying.—The most common method of dewatering sludge is air drying. This is accomplished by spreading the sludge in relatively thin layers on underdrained sand, gravel, or cinder beds. Part of the moisture retained in the sludge is evaporated and part filters through the bed into the underdrains. The time required for drying varies with climatic conditions and the character of the sludge. Well-digested sludge is less offensive and dries more readily than relatively fresh sludge in which a tough, greasy scum excludes penetration of the air into the sludge. In northern climates the drying area required varies from 2 to 5 square feet per capita for fresh and activated sludge; for well-digested sludge it is about 1 square foot, and for trickling filter humus tanks, 0.5 square foot per capita. In southern climates the area required can be reduced. Glass-covered drying beds require but half the area of open beds. When the sludge has dried sufficiently to contain but 50 per cent of moisture, it becomes spadable and is removed from the beds.

Mechanical Drying without Heat.—A number of different methods have been devised to dewater sludge more rapidly than is possible on drying beds. Mechanical driers take the form of filter presses, centrifuges, and vacuum driers in which the excess liquid is either pressed, thrown, or sucked out of the sludge. The sludge is thus compacted to sludge cake. The waste liquor is highly offensive. Sometimes the sludge is treated with acid, alum, or other chemicals in order to permit more rapid dewatering.

Heat Drying.—The sludge cake produced by air or mechanical drying is sometimes further dewatered by placing it upon roasting pans or into

rotary kilns in which the remaining water is driven off by heat. This is an expensive process. The dry sludge is commonly ground and sold as fertilizer.

Sludge Disposal.—There are a number of ways for the final disposal of sludge: namely, dumping, lagooning and trenching, filling, fertilizing.

Dumping.—Some of the larger sea-coast towns discharge the wet or dewatered sludge from sedimentation tanks onto barges which are towed out to sea. The sludge is then dumped in deep water. This method of sludge disposal is also practiced in some of the cities situated on great lakes or large rivers.

Lagooning and Trenching.—Where there is much low-lying land in the vicinity of the disposal plant, it is a relatively simple matter to throw up dikes or utilize natural depressions in the ground to receive the wet sludge, which then forms a sludge lagoon. The sludge dries slowly under the influence of evaporation and digestion. Where much land is available, it is left in place until the lagoon is filled; where land is scarce, the sludge is removed after drying and disposed of in other ways. The odors rising from sludge lagoons are troublesome. Sometimes the sludge is run into trenches which are then covered with earth. After the sludge dries it is worked into the soil by plowing. The disposal area required is naturally large.

Filling.—Dried sludge is well adapted for use in filling low-lying land and much waste land can be reclaimed in the neighborhood of treatment works by careful filling.

SLUDGE USED IN THE UNITED STATES

CITIES 100,000 AND OVER

(After Pearse) *

City	Hauled by Farmers	Used Year After Year	Money Received
Philadelphia †	Yes	Yes	No
Baltimore ‡	Yes	Some do and some do not	25 cents per load
Rochester	Yes	Yes	75 cents per load of 2 cubic yards
Providence	Small amount	No	No
Columbus	Yes	Yes	No
Akron
Atlanta	Yes	Yes	Contract
Worcester	Some	Some	No
Dallas	No
Bridgeport
Houston	No
Albany	No
Reading	(?)

* From Part II, *Hearings on the Improvement of the Illinois and Mississippi Rivers.*

† 9-year average.

‡ Received from \$374.47 to \$2,689.94 per year from farmers and from sale to fertilizer company.

Use as Fertilizer.—The use of sewage sludge as a fertilizer continues to be advocated as a measure of conservation. Municipalities are sometimes

able to dispose of wet sludge or sludge cake to farmers who use it for fertilizing purposes. In some cases a nominal charge is made for the material. In the densely populated districts surrounding large cities, it is frequently impossible to get rid of the sludge produced and projects are now under way to manufacture dry sludge for fertilizing purposes. Dried activated sludge has excellent fertilizing qualities and is especially adapted to use on lawns and golf courses. It is hoped that sufficient revenue will be derived from the sale of the sludge to offset some of the expense of sewage and sludge treatment. The use of sludge produced by the treatment works of the large cities of the United States is shown in the preceding table.

Hygienic Aspects of Sludge Utilization.—A number of sanitary hazards are involved in the use of sewage sludge for the fertilizing of crops. In the North these are probably restricted to the transmission of bacterial diseases, such as typhoid fever and dysentery by means of vegetables or other agricultural products that are consumed raw. In the South the sludge may contribute besides to the pollution of the soil and the transmission of hookworm infection and amebic dysentery. Recent studies show that the viability of the bacilli of the typhoid-dysentery group in digesting sewage sludge is a matter of days rather than, as formerly believed, weeks or months, and that the longevity of the organisms of the cholera group is even shorter. The typhoid fever bacillus probably never survives as long as ten days in digesting sewage sludge.

Choice of Sludge Disposal Methods.—The choice of sludge disposal methods like that of sewage disposal depends upon numerous factors, such as the general location of the community, the demand for sludge as a fer-

SLUDGE DISPOSAL IN THE UNITED STATES

CITIES OVER 100,000

(After Pearse)*

City	Dump at Sea	Lagoon	Air Dry and Fill	Run Out and Flow In	Into Stream at Flood	Dry
Philadelphia	Yes
Cleveland †	Yes
Baltimore	Yes	Yes
Rochester	Yes
Providence	Yes
Columbus	Yes	Yes	Yes
Akron	Yes	Yes ‡
Atlanta	Yes	Yes	Yes
Worcester §	Yes
San Antonio	Yes
Dallas	Yes
Bridgeport
Houston	Yes	Yes	Yes
Albany	Yes
Reading	Yes

* From Part II, *Hearings on the Improvement of the Illinois and Mississippi Rivers.*

† Dumped into Lake Erie.

‡ Major portion of sludge discharged into river.

§ No money appropriated to operate sludge presses since April, 1917.

tilizer, the economics of sludge treatment, and the availability of land for disposal. Some of the methods practiced in large American cities are classified in the preceding table.

Management of Sewage-Treatment Works.—Proper management of sewage-treatment works is as important as proper design, and is more difficult to secure. It is a most regrettable fact that many treatment works in the United States have been badly neglected, and, in consequence, have given inefficient service. Neglect not only results in making the effluent unsatisfactory but leaves the works themselves in an offensive condition. Neglect of small plants is more common than of plants large enough to require the entire time of one or more attendants.

Another frequent cause of failure is that treatment works are allowed to become outgrown, so that the plant becomes overloaded and the process becomes inefficient. The sewers of a city are usually designed for a long period in advance—forty or fifty years—but this is not the case with treatment works, for the reason that such works can ordinarily be enlarged when necessary. This is sound policy, for the reason that the methods of treatment are constantly improving, and it is desirable to take advantage of these improvements as far as possible whenever enlargement is necessary. But, if the works are to operate satisfactorily, the enlargement must be made as the tributary population increases, taking advantage of the methods and improvements developed up to the time.

The treatment of sewage is so largely a chemical and biological matter that it is desirable to have the works in charge of men trained in sanitary engineering, with a laboratory at their disposal. Tests of the sewage before and after treatment should be made regularly in order to ascertain the efficiency of the process. Tests should also be made of the water into which the sewage is discharged. In the case of plants of large size, provided with laboratories, such tests are made daily, but in the case of plants too small for constant employment of a chemist, tests should be made regularly by some controlling authority. Herein lies one of the functions of the state board of health.

Treatment Plants as Nuisances.—If sewage treatment works are properly designed and carefully operated, and if they are enlarged from time to time to meet the needs of the growing community, they need not be the cause of offensive conditions, but often they are, as a matter of fact, a source of nuisance in themselves. There is a natural opprobrium attached to a region where such works exist that results in a recognized deterioration of property values. The processes used for the treatment of sewage not infrequently result in odors that may be objectionable over considerable areas. Where the treatment works are entirely covered, as some kinds of works may be, little or no nuisance may result, but where, for example, the sewage is first submitted to putrefaction in a septic tank and the septic effluent is then sprayed into open air upon the surface of sprinkling filters, this exposure of the atomized liquid results in the liberation of odors that may reach distances up to

perhaps half a mile from the plant, depending upon the amount and character of sewage treatment, the local topography, prevailing direction of the wind, humidity in the atmosphere, and other conditions. Recent experiments show that the judicious use of chlorin compounds as deodorants will eliminate complaints in many cases.

Frequently high winds will carry the spray itself for several hundred feet with inevitable bacterial pollution of the air. In the operation of sprinkling filters also it has been found that at certain seasons of the year swarms of minute filter flies (*psychoda*) breed in the porous beds. These are very troublesome, if not dangerous, in the immediate vicinity of such works, but their radius of flight is extremely small. These flies are now being combatted by the use of chlorin or by the cultivation of certain inimical insects (*Achorutus viaticus*). In considering the need of sewage treatment it is proper to balance these possible nuisances against those resulting from the discharge of untreated sewage into a body of water. The situation must be studied for the installation of sewage treatment works sometimes merely substitutes one nuisance for another.

Nuisances Caused by Trade Wastes.—It happens not infrequently that the greatest nuisance in streams is due not so much to domestic sewage as to the presence of trade wastes that are discharged into the stream directly, or that are allowed to flow into the stream through the sewers. For example, the discharge of spent dye liquors may color the water of a stream for many miles; petroleum wastes from gas works may cause iridescent films to form upon the surface of the water, producing an unsightly appearance and increasing the odor directly, as well as indirectly, by excluding air from the water; the acid iron wastes from galvanizing works may cause a rusty discoloration that not only imparts a brown color to the water, but paints the rocks and submerged stumps along the shores for many miles. When nuisances of this character arise it is wise and proper to install clarification plants, and sometimes more elaborate works, for such nuisances cause real damage to property and to personal comfort. Trade waste pollution may interfere with the filtration of water even more than sewage itself. Illustrations of this are the paper-mill pollutions in New York State and the acid-iron wastes in Pennsylvania.

COÖPERATIVE SANITATION

What appears to be needed at the present time is some method of co-operation by which needed sanitary reforms can be brought about at least expense. It is unbusinesslike to compel the treatment of the sewage of a large upstream city in order to protect the water supply of a small city lower down, if pure water can be furnished the latter in some better and cheaper way. Legislation that clothes the state authorities with power to prevent the pollution of streams by sewage, but does not give them power to compel the purification of water or to control pollution by trade wastes, is

unfortunate. It naturally leads to litigation rather than coöperation, and may retard rather than hasten necessary sanitary reforms. Some of our state authorities have already been entrusted with this matter and our national government as well has taken a hand. In England and Germany district boards have been given jurisdiction over particular areas. In some respects these natural hydrographic boundaries have advantages over artificial state boundaries. In whatever form the authority may be constituted the idea of coöperation should prevail. Ironclad rules against stream pollution should give way to a rational distribution of the burden of water purification and sewage treatment, and an equitable adjustment of cost made between the parties interested, thus decreasing the total expense of sanitary measures required and utilizing natural resources for the purification of sewage in water as far as this is safe.

If the system of water carriage of sewage continues in use, the time will some day come when the sewage of all of our cities will be treated, partially or completely, and all surface water supplies purified. It is proper to anticipate this consummation as far as our means permit, but in the meantime it is good business and sound common sense to spend our money first where it will go farthest and do the most good, building water purification plants and sewage treatment works, sometimes one, sometimes both, as they may be needed.

Adequate remedies against stream pollution from the standpoint of nuisance have been usually obtained by an appeal to the principles of common law. Cases involving bacterial pollution by sewage have been thus far too few to establish definite precedents. It will be interesting to see whether, in view of our increasing population and especially the increasing growth of our cities, the courts will ultimately decide that the use of unpurified river water as a source of water supply by riparian owners is a reasonable use of the water.

REFERENCES

- Annual Report of the Massachusetts State Board of Health, 1890-1910.* (Summary of results obtained at the Lawrence Experiment Station during twenty-one years is given in the *Report of 1908.*)
- Contributions from the Sanitary Research Laboratory of the Massachusetts Institute of Technology*, eight volumes, containing papers by Sedgwick, Winslow, Phelps, and others, 1905-1910.
- DUNBAR. *Principles of Sewage Treatment*, Translated by H. T. Calvert. Charles Griffin & Co., Ltd., London, 1908.
- FULLER, G. W. *Sewage Disposal*. McGraw-Hill Book Co., New York, 1912.
- METCALF and EDDY. *American Sewerage Practice*. Three volumes. McGraw-Hill Book Co., New York, 1915. The third volume treats of sewage disposal.
- LELEAN, P. S. *Sanitation in War*. J. & A. Churchill, London, 1917, 2nd Ed.
- FORD, JOSEPH H. *Elements of Field Hygiene and Sanitation*. P. Blakiston's Son & Co., Philadelphia, 1917.
- FOLWELL, A. PRESCOTT. *Sewerage*. Wiley & Sons, New York, 1918, 8th Ed.

- KINNICUTT, WINSLOW, and PRATT. *Sewage Disposal*. Wiley & Sons, New York, 1919, 2nd Ed.
- METCALF and EDDY. *Sewerage and Sewage Disposal*. McGraw-Hill Book Co., New York, 1922.
- HARDENBERG, W. A. *Home Sewage Disposal*. J. B. Lippincott Co., Philadelphia, 1924.
- BABBITT, HAROLD E. *Sewerage and Sewage Treatment*. Wiley & Sons, New York, 1925, 2nd Ed.
- KERSHAW, G. BERTRAM. *Sewage Purification and Disposal*. Cambridge University Press, Cambridge, England, 1926, 2nd Ed.
- FULLER, G. W., and MCCLINTOCK, J. R. *Solving Sewage Problems*. McGraw-Hill Book Co., New York, 1926.
- For references to recent works for the treatment of sewage, see files of *Engineering News-Record*.

SECTION XI

REFUSE DISPOSAL

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The refuse disposal problem is only to a slight extent an hygienic one; it is more a problem of economy, convenience, and general cleanliness. Odors rising from fermenting garbage or the processes connected with garbage disposal do not injure the public health directly, yet they are offensive to the sense of smell and interfere with the full enjoyment of an otherwise healthy environment. From this standpoint their elimination is an important matter. Garbage also attracts flies, which breed in unprotected garbage receptacles and sometimes rise in swarms from dumps in which garbage, manure, or other organic wastes are permitted to accumulate. These materials, furthermore, provide food for rats and often support a large rodent population.

Accumulating rubbish creates a fire hazard and is not merely offensive to the æsthetic senses, but affords an opportunity for mosquito breeding in tin cans and other discarded receptacles that become filled with rain water and house the insect larvæ and pupæ until maturity. Ashes and street dust are easily moved by the wind and when blown about irritate eyes, nose, and throat and predispose to bacterial infection. The indirect relations of refuse disposal to the public health are therefore many and the sanitary disposal of wastes, more particularly garbage, is quite commonly a function of the local boards of health, although this function is more and more being delegated to other municipal departments. Its connection with boards of health is easily explainable in the light of the developmental history of sanitary science where it occupies a position similar to that of plumbing.

Composition and Quantities of Refuse.—The general term “refuse” is applied to all of the solid waste material from human habitations not carried by the sewers. As such it includes ashes, rubbish, garbage, street sweepings, and dead animals, as shown in the classification on the following page. Excreta, which form part of the solid wastes of a community, although included in this classification, are considered in other sections of this book, for the reason that they are the most serious sanitary menace and need separate treatment.

¹This section is based upon material prepared by the late Professor George C. Whipple for previous editions of this book.

CLASSIFICATION OF MUNICIPAL REFUSE MATERIALS

After Hering and Greeley

- PUBLIC REFUSE:** Street manure and litter; sweepings and dust; leaves; droppings from carts; large dead animals; snow; cleanings from public catch-basins.
- TRADE REFUSE:** Steam ashes; dry factory wastes; slaughterhouse waste (offal); rubbish from office buildings and factories; cleanings from private catch-basins.
- MARKET REFUSE:** Garbage from markets; rubbish and cleanings from markets; old boxes and barrels.
- STABLE REFUSE:** Manure; straw; cleanings from stables.
- HOUSE REFUSE:**
- Garbage:* Animal matter; vegetable matter; tin cans; small dead animals.
 - Ashes:* Dust; glass; crockery; brick and stone; metal fragments.
 - Rubbish:* Sweepings from buildings; boxes and barrels; wood; paper; rags; excelsior; straw; leather; rubber; metal ware; bedding; old furniture.
 - Night Soil:* Contents of privies.

The quantity of waste material that must be removed from cities is very large. In the Borough of Manhattan of the City of New York, for example, ashes amount to about 1,300 pounds per capita per year; rubbish, 100 pounds; street sweepings, 300 pounds; and garbage, 200 pounds; the total amount of refuse being, in round numbers, a ton per capita per year. In smaller cities the per capita quantities of collected refuse are less than half of this, sometimes considerably less. The amount of garbage alone varies from less than 100 to upward of 200 pounds per capita per year. Both the quantity and character of the refuse vary with the seasons, the maximum amount of ashes being produced in the winter and the maximum amount of garbage in the summer.

Ashes weigh from 900 to 1,200 pounds per cubic yard; garbage, from 900 to 1,100 pounds; street sweepings, from 700 to 1,800 pounds; and rubbish, from 150 to 250 pounds. The following figures serve to indicate approximately the constituents of the principal classes of refuse.

CONSTITUENTS OF CITY REFUSE

Refuse	Water, per Cent	Volatile Matter, per Cent	Ash, per Cent	Carbon, per Cent	Heat Units per Pound of Refuse, B. T. U.
Ashes	7-25	8-10	50-60	18-25	3,700
Garbage	70-80	15-25	5-15	4- 8	2,000
Rubbish	5-15	40-65	5-15	15-40	6,000
Street sweepings	35-45	20-30	25-95	18-25	4,000

Collection of Refuse.—There are two general methods of refuse collection: the mixed system and the separate system. The choice of the system of collection depends to a large degree upon economic considerations and the type of ultimate disposal practiced. With the *mixed system*, which is the one most generally used in Europe, all of the wastes are placed in a single receptacle by the householder. They are then collected in single conveyances and transported to the point of disposal where they are disposed of jointly. With the *separate system* garbage, ashes, rubbish, and other wastes are placed in separate containers by the householder and collected in separate wagons or trucks to be separately disposed of, usually in different ways. The separate system is commonly used in America, but with numerous combinations of processes of collection and disposal. It requires a good deal of intelligent coöperation on the part of the householder but is probably more sanitary in operation than the mixed system.

The choice of proper receptacles for the storage of the various types of refuse largely determines the amount of nuisance that waste materials will cause in the neighborhood of human habitations. Garbage should be placed



FIG. 115.—A ROCK PILE CREMATORY.

in covered metallic containers that render the contents inaccessible to dogs and other animals and do not permit the breeding of flies. Some cities require that all garbage shall be wrapped in paper before being deposited in the container. This keeps the cans clean and prevents rapid fermentation in summer and freezing in winter. Garbage cans should be thoroughly scoured from time to time. Ashes are commonly placed in covered metal containers, and rubbish is usually permitted to be stored in barrels or tied into bundles. Because of its high market value as a fertilizer, stable manure is usually removed by private individuals. It is a favorite breeding medium for flies, however, and should be stored in well-drained receptacles that are as nearly fly-tight as possible. The use of insecticides for the destruction of fly maggots is not very efficient because of the large quantities of organic matter in the manure.

The frequency of refuse collection varies with the season of the year. Garbage is produced in greater bulk during the summer and ferments more rapidly at this season of the year. It is therefore commonly collected at shorter intervals of time during the summer months (1-3 days) than in winter (3-7 days). Ash collection is more frequent in winter.

Disposal of Refuse.—In rural or sparsely settled communities the disposal of refuse is a simple matter. Garbage is fed to domestic animals,

notably chickens and pigs. Failing this, it is either buried in field or garden or burned together with rubbish. Incineration is readily accomplished in specially constructed wire baskets. For large dwellings, isolated hotels, or camps, stone or brick incinerators are sometimes built.

A type of incinerator much used in army camps during the Great War is shown in Figure 116. In this incinerator the damp refuse is thrown onto the drying shelf, whence it slides or is pushed into the fire. If fly proof, this pattern may be used for the incineration of feces. In temporary camps it is impossible to build incinerators of this type and the refuse is therefore either burned in open pits lined with rubble, as shown in Figure 115,

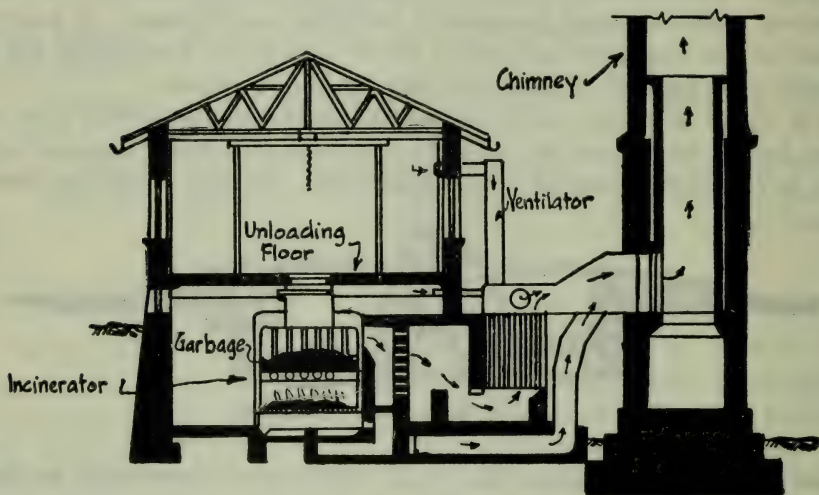


FIG. 116.—CROSS SECTION OF A DECARIE INCINERATOR.

or buried. In the circular rock pile crematory the fire is built in one quadrant, and the solid wastes are thrown into it. The central pile of rocks assists in creating a draft and offers a large surface for the evaporation of liquids which are poured into neighboring quadrants. When fuel is scarce and stones few, the Caldwell or English crematory can be used. It consists of a trench 10 feet long and 1 foot wide. It slopes from the surface at either end to the center where it is about 15 inches deep. A barrel is placed over the deep portion and a chimney of clay, earth, or sand properly tamped is built around it. A fire is made in the pit and the barrel is burned out, after which the solid cone of earth remains. Fuel and garbage are dropped through the chimney. The leeward opening is closed and a bed of tin cans is provided to take the place of a grate.

In urban communities refuse disposal becomes an important and sometimes vexing sanitary problem. A number of different methods are in use as illustrated in the following classification in which the applicability of the disposal process to each class of waste is indicated.

CLASSIFICATION OF METHODS OF REFUSE DISPOSAL

Adapted from Crohurst

Dumping	Filling	Burial	Incineration	Feeding	Reduction	Fermentation	Sewerage
Garbage Animals Ashes Rubbish Excreta Street sweepings	Garbage Ashes Rubbish Street sweepings	Garbage Animals Excreta Street sweepings	Garbage Animals Ashes Rubbish Excreta Street sweepings	Garbage	Garbage Animals	Garbage Animals	Garbage

The choice of the system of disposal depends upon local conditions and must be decided by economic as well as sanitary considerations. Reasonable sanitation at a reasonable cost is the most that can be expected.

Dumping.—In some of the large sea-coast towns of the world, notably New York City and Liverpool, England, some of the municipal refuse, particularly garbage, is disposed of by being placed on scows, towed out to sea, and dumped into the ocean. This practice of depositing waste materials in deep water was also in vogue during the earlier days of the cities in the Mississippi Valley and along the Great Lakes, but is no longer countenanced. It is probably the cheapest method of refuse disposal, but results in the ultimate stranding of sometimes putrid waste materials along the water-line of beaches and banks.

Filling.—Some of the refuse materials, notably ashes, are well adapted for filling low-lying lands, abandoned clay pits, and tidal marshes. Many communities make profitable use of this method for the disposal of their mineral, non-putrefying wastes, and large tracts of so-called “made land” have thus been created. The only objection to this method is the blowing about of fine ash dust during windy weather. In the “sanitary fill” a certain amount of decomposable organic material is mixed with the ashes and, if properly proportioned and well placed, is slowly destroyed without causing a nuisance. When, however, too large quantities of garbage and other organic wastes, such as manure, are mixed with the mineral refuse, a nuisance is sure to occur, odors arise, flies are bred, and rats and other animals scavenge about the dumps. If combustible materials as contained in rubbish are added, “dump fires” occur and sometimes continue to burn or smolder for years, producing smoke and foul stench that are carried long distances by the winds.

Burial.—Burial in long trenches under a layer of earth can be successfully employed in disposing of organic refuse material. Waste materials of this type can also be scattered upon the ground and plowed under. Decomposition takes place very slowly under the earth cover and relatively large tracts of land are therefore required. This makes the use of burial expensive for large communities but offers a safe and sanitary method for smaller municipalities. For successful operation it naturally requires rigid

separation of the refuse. Careless covering of the wastes permits fly breeding and gives rise to other nuisances.

Incineration.—Incineration is the destruction of wastes by fire. The mixed refuse of cities and towns contains much combustible material and is readily disposed of by incineration. In the United States it is customary to exclude all or most of the ashes, although the latter sometimes contain as much as 30 per cent of unburned coal. Ashes, however, are large in bulk and increase the operation difficulties of incinerator plants.

From the sanitary standpoint incineration affords an excellent method of refuse destruction as the solids remaining after incineration are stable materials that can be used to fill lands adjacent to the incinerator. In order to prevent odors from the flue gases in the vicinity of incinerator plants, the temperature of the gases must be raised above 1200° F. When these temperatures are not reached, the vapors driven off from the wastes are distinctly offensive. Auxiliary fuel is frequently required to start the furnace. The steam generated by incineration can sometimes be usefully employed in operating the plant equipment and producing electric power.

Many different types of incineration plants have been developed. Most of them possess certain patented features that are intended to make the process more economical or to secure speedier or surer operation. In some plants the refuse is first dried or heated before passing onto the incineration grates; in others it is deposited directly in the furnace. The amount of mixed refuse burned varies from 50 to 100 pounds per square foot of grate area. The Decarie incinerator, illustrated in Figure 117, has been used successfully in a large number of American municipalities. Attempts have recently been made to utilize the gases and heat of fermenting garbage in aiding the incineration process and one plant constructed upon this principle is now in operation.

Feeding.—Incineration of garbage as well as the other methods of garbage disposal that have been discussed above are objected to because of the loss of large quantities of food material contained in garbage. This food may be conserved by feeding it to hogs, special piggeries being sometimes established for this purpose. Feeding requires frequent collection and careful management at the piggery, but presents a sanitary and often profitable method of garbage disposal. Many small communities and even some of the larger cities dispose of their garbage in this way, which, if carefully controlled, does not create any nuisances. Comparisons of garbage-fed hogs with grain-fed animals result in a slight advantage of the latter, but present no hygienic factors detrimental to the interests of public health. Garbage from hotels and restaurants which usually contains larger amounts of valuable foodstuffs is quite commonly collected privately by piggeries in the vicinity of cities and towns irrespective of the disposal system employed by the municipality.

Reduction.—The reduction process is suitable to the disposal of garbage and dead animals, which contain much grease and fertilizing material. The

raw materials, however, require special treatment to extract the grease and produce dry substances that can serve as a filler or fertilizer base. When the garbage reaches the common type of reduction plant, it is sorted to remove foreign substances, such as tin cans, glass bottles, and rags. It is then conveyed to a series of tanks known as digesters in which it is cooked from six to ten hours under a pressure of about 60 pounds. It then passes through presses which separate the water and fats from the solid material that

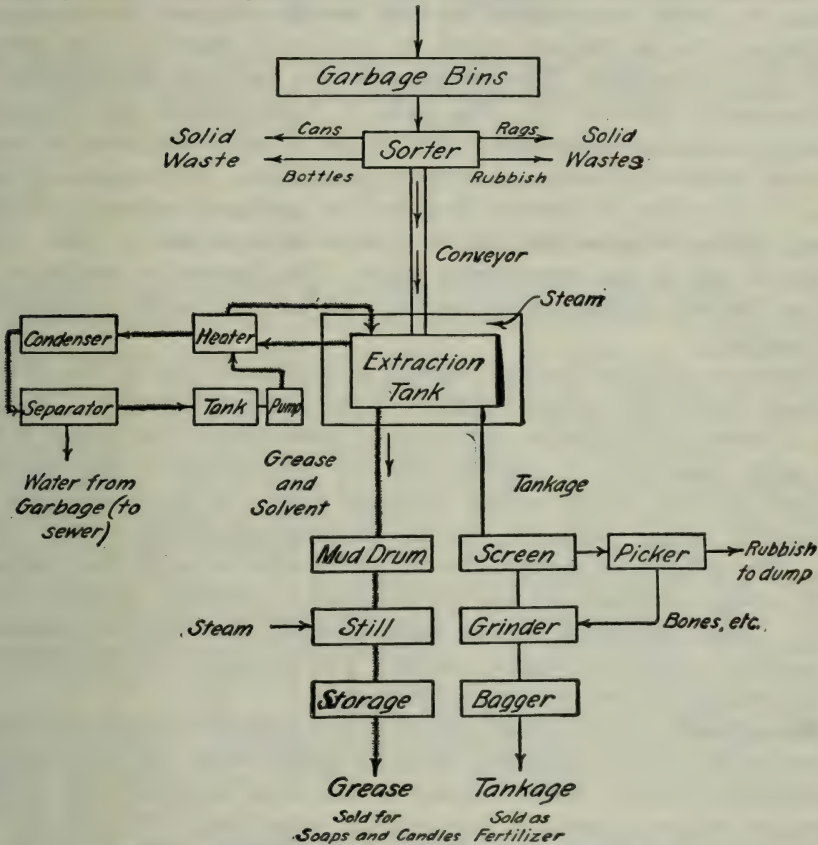


FIG. 117.—COBWELL PROCESS OF GARBAGE REDUCTION.

is known as tankage. The water and grease pass through settling tanks where the grease is skimmed off. The water is discharged or evaporated; in the latter case the solids are added to the tankage, which is sometimes treated with a solvent, such as naphtha, for the further extraction of grease, the solvent being recovered by distillation. The tankage is dried and ground, the marketable tankage amounting to about 20 per cent of the garbage. From 1 to 3 per cent of grease is recovered and sold for the manufacture of soap and grease compounds. The Cobwell process, outlined diagrammatically in Figure 117, differs in operation from the one described above in the fact that the solvent is added to the green garbage at the first cooking instead of after the first extraction of grease.

The design and sanitary operation of garbage reduction plants presents many difficulties. Unless carefully managed, offensive odors are given off which are sometimes carried great distances and explain the common prejudice to this type of plant. If proper precautions are taken, however, nuisances arising from the operation of reduction plants can be done away with. Scrubbing of the waste gases and chlorination will control the odors.

Fermentation.—The conversion of garbage and other organic refuse into humus by suitable fermentation has only recently attracted the attention of sanitarians. In the Beccari system the garbage is placed in cells through which air is circulated. The garbage ferments and rises spontaneously in temperature to 140-150° F. The gases of decomposition pass through a chimney containing diaphragms on which earth or other substances are placed to render odorless the volatile products of fermentation. Fermentation is completed in thirty-five to forty-five days and the garbage is reduced to a dark brown, inoffensive mass that can serve as a fertilizer base or filler. The process is still in the experimental stage. The destructive distillation of garbage has long challenged engineering skill but still awaits a satisfactory development.

Sewerage.—A novel garbage disposal method makes use of the existing sewerage system and sewage treatment works of the community. The garbage is transported to one or more disposal points where it is ground into a fine pulpy mass which is dumped into the trunk sewers of the city and conveyed by them to the sewage treatment plant. It then undergoes the processes of sewage treatment, such as screening, sedimentation, and oxidation. The addition of garbage is said not to interfere with the treatment of the sewage, although the load on the plant is naturally increased materially. Proper provisions must be made to handle the additional quantities of suspended solids and organic matter. The economics and practicability of this process have not yet been established.

Management.—The sanitary collection and disposal of refuse is a proper governmental function justifiable under the police power. In many smaller communities, however, refuse disposal is still being undertaken by private enterprise without systematic governmental supervision. In some of the larger communities both the collection and disposal are entirely in the hands of the local authorities. In others the refuse is collected by the municipality but disposed of by private parties. In still others collection as well as disposal is contracted for. It is possible to obtain satisfactory results both with the municipal system and the contract system, but the latter is apt to be the more economical one.

REFERENCES

- LELEAN, P. S. *Sanitation in War*. J. & A. Churchill, London, 1917, 2nd Ed.
FORD, JOSEPH H. *Field Hygiene and Sanitation*. Blakiston, 1917.
CROHURST, H. R. "Municipal Wastes." *Pub. Health Bulletin No. 107*, United States Public Health Service, 1920.
HERING, R., and GREELEY, S. A. *Collection and Disposal of Municipal Refuse*. McGraw-Hill Book Co., New York, 1921.

SECTION XII

VITAL STATISTICS

The Registration of Births, Deaths, and Marriages, and the Reporting of Notifiable Diseases; the Resulting Records and Derived Statistics; and Their Legal, Social, and Public Health Uses.

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Statistics have suffered in reputation because of the seeming truth of the trite statement that one can prove anything by figures. In reality figures are but evidence upon which conclusions may be based. If the evidence is faulty and the faults are not perceived, errors in judgment may result. But this is true of all evidence upon which opinions are based and is no more true of figures and statistics than it is of other kinds of evidence.

Statistics are derived from the collection and numerical classification of observations relating to certain facts or events. They are usually limited to the systematic collection and classification of data relating to relatively large classes of events. In the making of statistics the first and essential step is the recording of observations. After the observations have been noted a numerical compilation of their frequency or of the frequency of certain of their conditions or attributes is possible. The derived statistics, being but a numerical classification or analysis of the recorded events, depend primarily for their usefulness upon the accuracy of the original records of facts. They depend secondarily upon the accuracy of the statistical classification, compilation and analysis.

Recording and Compilation of Facts.—The original notation of facts and of the occurrence of events is usually secured in one of two ways, by enumeration or by registration. In the United States, for example, observations relating to the population are made by enumeration at the decennial censuses. The census enumerators go from house to house and secure certain information regarding each individual. The enumerators are the observers who secure the original data. Statistics of the population are made by the classification of the information thus obtained and the numerical compilation of the frequency of certain attributes.

On the other hand, the notation of facts relating to deaths is secured by registration. For each individual who dies there is registered with an official known as a registrar of deaths certain information regarding the deceased and the cause and time of death. Here the observers who record

the original data are the physicians, members of families, and undertakers. From the classification and compilation of the information thus recorded mortality statistics are derived. Statistics of population depend for their accuracy upon the correctness of the records made by the enumerators and mortality statistics upon the accuracy of the information registered in death certificates.

Need for Knowing the Dependability of Basic Data.—The statistical method is in itself dependable, although it is true that statistics may be vitiated by the use of inaccurate or incomplete data as a basis or of faulty methods in classification and compilation. Conclusions drawn from statistics by those who attempt to use them may be quite erroneous, but this is as often due to the limitations of the user as to the limitations of the statistics. A common error in the use of vital statistics is the comparing of numerical statements or ratios which are too dissimilar to allow of comparison.

One of the dangers in drawing conclusions from statistics is that the statistics may have been compiled and published by statisticians who were unfamiliar with the accuracy or inaccuracy of the data which they have compiled. The data collected by census enumerators may be incomplete or fictitious as they have been at times for certain communities. Under such circumstances the derived population statistics must necessarily be undependable.

Vital statisticians may compile the data of deaths as recorded in death certificates without knowing that the causes of death given may be erroneous in a large proportion of the cases. In such instances the statistics of causes of death must be equally erroneous.

Statistics of birth may be compiled and published and accepted by the unknowing as representing actual conditions. The fact may be that in the state or city under consideration only 80 per cent or maybe only 60 per cent or 50 per cent of all births were registered during the period under consideration. The derived statistics may therefore be worse than useless, for, while they have the appearance of giving information, what they actually give is misinformation.

Morbidity statistics, statistics of the incidence of disease, may be compiled and published without a clear explanation that the statistics are of the reported cases but that there were many cases that were not reported, the proportion of unreported cases depending upon the disease, the community and the time.

Statistics, When Used, to Be Studied Critically.—There is a tendency for the statistician to accept the data given to him as being accurate, and to compile, tabulate and analyze them as though they in all instances represented facts possible of such handling. The final statistical results are likely to be accepted by the student without a thought of the nature or source or possible inaccuracies of the basic data.

Often the statistician understands the incompleteness and inaccuracies full well and in footnotes and explanatory text explains precisely the nature

and extent of dependability of published statistical tables. The reader, however, is prone to pay little attention to explanatory text and frequently to ignore footnotes. A statistical table should always be studied with care and caution and at times critically to an extent bordering on suspicion.

Persons unacquainted with statistical methods are likely to consider statistics as a subject beyond their comprehension. This is a mistaken idea. Most statistical tables are the result of but the simplest of mathematical computations, and are derived usually by nothing more intricate than addition, multiplication and division. Anyone who can comprehend that 350 births during a year in a population of 35,000 persons would be at the rate of 10 births for each thousand of the population can understand most ordinary statistical statements. Familiarity with the meaning of certain of the terms in common use is, however, essential.

To make dependable statistics the original observations and records from which they are derived must be true and accurate, and the classification, compilation, and analysis must be done by competent individuals. The value of statistics when thus handled is daily demonstrated in various social, economic and commercial activities.

VITAL STATISTICS

Definition.¹—Vital statistics may be defined as statistics relating to the life histories of communities or nations. They pertain to those events which have to do with the origin, continuation, and termination of the lives of the inhabitants. They commonly include statistics of population, births, marriages, deaths, and the occurrence of disease, and the conditions attending these events.

Development.—Vital statistics are not a thing of recent origin. Their development to their present form, however, is comparatively modern. The Egyptians, Greeks, and Romans made census enumerations. Some of the ancients, notably the Romans, required also the registration of births and deaths. The statistical treatment of the records was, however, comparatively limited. During the last century and a half, and more particularly the last fifty years, the treatment of vital statistics has been undergoing a rapid evolution. In their present developed form they give a fund of useful information otherwise unobtainable. They have become an essential to every well-organized community and nation. They give a composite picture of the life history of a people which can be secured in no other way. They furnish a means of comparing the life history of one community or people with that of others and of the present with the past.

Based upon Population.—All vital statistics are based upon the population. The frequency of births, marriages, sickness, and deaths is expressed in terms of the population, usually as rates giving the number for each 1,000 inhabitants or class of inhabitants. In comparing different communities or

¹ For statistical methods and definitions of terms, see page 1177.

different periods, births, marriages, deaths, and the incidence of disease must be based upon a common unit of population. The first requisite to useful vital statistics is statistics of population showing the number of inhabitants, classified according to age, sex, nativity, race, and occupation. It would be desirable, if possible, to have also a classification according to economic status, as birth, sickness, marriage, and mortality rates frequently vary with the incomes of individuals or households. An understanding of population statistics is therefore the primary essential to the comprehension or use of vital statistics.

POPULATION STATISTICS

Source of Data.—The principal source of information regarding population under existing conditions is a census enumeration. For the United States these enumerations are made every ten years. The last census was taken as of January 1, 1920. In the United States a census has been taken every ten years since 1790, in Great Britain every ten years since 1801. In taking a census it is desirable so far as possible to take it at a time when the greatest number of people will be at their usual homes. In the United States a midwinter census finds many people absent from the northern states and an unusual number in southern winter resorts. A midsummer census finds an unusual number at the seashore and at other summer resorts. A number of the states take a census midway between the United States decennial censuses, so that they have an enumeration of the population every five years. Many large cities count their population every year.

As the only source of definite information as to population is the census enumeration, and as the population is continually changing, in most cases increasing, it is necessary to make estimates of the population for the periods between the census enumerations upon which to base rates for the various vital events, and especially for the accurate computation and expression of marriage, birth, death, and sickness rates.

Nature of Census Information.—The taking of a census consists usually of more than a mere enumeration of all persons living at the time the census is taken. It includes the recording of certain information regarding each individual. In taking the 1920 United States census the following information relating to each individual was recorded by states, counties, and townships, villages or cities: Name; address; sex; color or race; age at last birthday; whether single, married, widowed, or divorced; birth and "mother tongue" of the individual and place of birth of his father and mother; year of migration to the United States; whether naturalized or alien; whether able to speak English; the individual's occupation, the kind of work done and the industry or business in which employed; whether an employer, employee, or working on own account; whether able to read and write; whether attending school; whether he owns the home in which he lives.

From the information thus obtained the statistics of population are made. By the classification and numerical compilation of these data it is possible

to ascertain the composition and distribution of the population as to sex, color or race, age, marital status, nativity, occupation and literacy.

Sources of Error in Census Enumerations.—A certain number of individuals will be enumerated both at the place where they happen to be and at their proper residences. A few will be missed entirely. However, the degree of error thus caused will not be great.

The margin of error in the securing of ages is greater. The age record is customarily intended to be the age in years at the last birthday. The ages given for children under 5 years old are likely not to be accurate due to the tendency to give the age of a child between 6 and 12 months of age as 1 year old and that of a child between 1 and 2 years old as 2 years of age. This tendency to give the age at the next birthday persists up to about the fifth year, although it is perhaps greatest during the first and second years. To avoid the error thus arising, the United States census records the ages of children under two years of age in years and months. For example, a child 6 months of age is recorded as six-twelfths of a year old and a child of 17 months of age as $1\frac{5}{12}$ years old.

Women 15 to 20 years of age are prone to give their ages as between 20 and 25 years. Adults over 25 years of age frequently do not know their exact ages and are likely to approximate their ages as being 30 or 40 or 50 years, and to a less extent as 35 or 45 or 55 years. The result is that there is at each census an exaggerated number of ages of 30, 40, 50 years, and also a lesser exaggeration of ages 35, 45, 55, and 65 years. Individuals over 80 years of age have a tendency to give their ages as greater than they really are.

There is also a considerable margin of error in the recording of occupation. This is due largely to an imperfect understanding of what is wanted and to the multiplicity of occupations and a lack of knowledge as to their proper designation.

Fluctuation in Population.—Populations are constantly changing. Individuals are continually being added by immigration. In the United States, and more particularly in some sections of the United States, considerable numbers are annually being added in this way. Immigration is also an important factor in the growth of population in certain South American countries, South Africa, New Zealand, Australia, and Canada.

Populations suffer a continuous diminution by reason of emigration. This is especially true of some European countries.

Migrations may not only affect the population of a country as a whole, but may also alter the distribution of people within a country. There is in many countries a constant movement of people from rural localities to the cities and from one locality to another.

All populations are also being increased by births and suffering losses by deaths. The rate of change, however, resulting from births and deaths is usually comparatively constant or alters gradually, while the changes due to migrations may be exceedingly irregular. The increase in the population

caused by the excess of births over deaths is known as the natural increase. A country in which the birth and death rates are equal and in which the factor of migration is negligible will have a fixed population.

The increase of population in certain countries is shown by the following table:

SHOWING GROWTH OF POPULATION OF CERTAIN COUNTRIES IN MILLIONS, 1800 TO 1910

	1800	1830	1860	1890	1910
France	27	32	36	38	39
Great Britain and Ireland...	16	24	29	38	44
Russia in Europe	35	45	68	92	..
Austria	25	29	34	40	49
Italy	17	21	25	30	34
Spain	10	11	15	17	19
Belgium	3	4	6	7
Sweden	2	2	3	4	5
United States	5	12	31	62	92

Estimates of Population.—The frequency of births, marriages, or deaths is usually expressed as the number occurring during the calendar year per 1,000 population. The figures thus given are known as the birth, marriage, or death rates, and are computed upon the mean population—that is, the number of inhabitants estimated to have existed at the middle of the year, July 1. These estimates are necessary for all dates except those on which census enumerations are made. For the making of estimates there are two principal methods commonly used, known, respectively, as the arithmetical and the geometrical methods. In each method the populations at the last two census enumerations form the known quantities from which the estimates are derived.

Arithmetical Method.—In the arithmetical method it is assumed that the increase or decrease in population which occurred between the last two census enumerations took place in equal amounts during each intercensal year (the years between two census enumerations) and will continue to take place annually in like numbers until the next census shall have been taken. Thus, given a city which had a population of 50,000 at the 1900 census (June 1, 1900) and one of 61,850 at the 1910 census (April 15, 1910), the increase during the intercensal period (9 years and 10½ months) would be 11,850, and the annual increase according to the arithmetical method would be

$$\frac{61,850 - 50,000}{9\frac{21}{24}}, \text{ or } 1,200$$

If it is desired to estimate the population as of July 1, 1906, for the purpose of calculating annual rates, this is done by adding to the population as it existed June 1, 1900, the sum of 1,200 for each year intervening between the date of enumeration (June 1, 1900) and the date for which the estimate

is to be made (July 1, 1906). There being 6 years and 1 month between these dates, the calculation would be

$$50,000 + (6\frac{1}{12} \times 1,200) = 57,300$$

This method assumes the same amount of increase each year and is analogous to the calculation of simple interest. It does not take into account the fact that with the annual increase in population the number of persons of marriageable age and therefore the number of married persons will be greater each year and consequently the number of births. The growth due to natural increases (the excess of births over deaths) is analogous to the increment of compound interest, and where this factor (the natural increase) is the principal one affecting the population growth estimates of population made by the arithmetical method are unsatisfactory, and especially so where the estimate is made for a date several years away from a census enumeration. Where the excess of births over deaths is the controlling factor in population growth the geometrical method of making estimates, being based on the principle of compound interest, is more accurate. Where migration is an important factor in population change, the arithmetical method may be the more accurate. The arithmetical method has been the one found most reliable in the United States and is the method used most commonly in the past by the Bureau of the Census. The method best adapted to a given population can be ascertained by taking the last two intercensal periods and finding whether the rate of increase during the last intercensal period was, when based upon the increase during the preceding intercensal period, at the rate indicated by the arithmetical or the geometrical method.

Geometrical Method.—As previously stated, the geometrical method is based upon the principle of compound interest.

Assuming a decennial census, let

P = population in 1900.

P' = population in 1910.

r = the annual increase per unit of population.

Then the population would be—

$$\text{In 1901} = P(1 + r)$$

$$\text{In 1902} = P(1 + r)^2$$

$$\text{In 1903} = P(1 + r)^3$$

$$\text{In 1910 } (P') = P(1 + r)^{10}$$

$$\frac{P'}{P} = (1 + r)^{10}$$

$$\sqrt[10]{\frac{P'}{P}} = 1 + r \text{ and } r = \sqrt[10]{\frac{P'}{P}} - 1$$

In practice the calculation would be made with the aid of a table of

logarithms, and given the value of r the estimated population for any intercensal or postcensal date is readily obtained. For postcensal dates the estimated population would be—

$$\text{In 1911} = P' (1 + r)$$

$$\text{In 1912} = P' (1 + r)^2$$

$$\text{In 1913} = P' (1 + r)^3$$

$$n^{\text{th}} \text{ year} = P' (1 + r)^n$$

The registrar general of England and Wales has used the geometrical method for England and Wales as a whole and a modified method for lesser subdivisions.

However, estimates are after all but estimates and nothing more. For large populations as of entire countries, and for smaller populations not much affected by migration, they are usually sufficiently dependable. In the United States they have been satisfactory for most of the older states and for many of the larger cities. Illustrations of where they have at times not been dependable are the State of Washington and the City of Detroit. For example, the State of Washington had a much more rapid growth in population between the census years 1900 and 1910 than it had between 1910 and 1920; therefore estimates made in the usual way, subsequent to the census of 1910 and previous to that of 1920, overstated the population. The percentage of error would be greater for each succeeding year. In Detroit the population increase was at a much greater annual rate between 1910 and 1920 than it was between 1900 and 1910. Therefore, estimates based on either the arithmetical or the geometrical method of calculation would have indicated a lesser population than was actually present in the city.

There are other states than Washington where the rate of growth has changed, although probably few where the change has been so great. There are many cities where the change in rate of growth has been as great as in Detroit although this is probably not often true of cities of the size of Detroit.

The estimates of the population for the United States are made by the Bureau of the Census. The task is a trying and in a measure an unsatisfactory one, dealing as it does with populations which ebb and flow in unrestricted movement throughout extensive areas. It is only by keeping in touch with the local factors which affect population that the work is possible. For cities, in addition to the actual movements of population, there must be considered the frequent changes in area and boundaries. American cities are continually changing their corporate limits, most of these changes are of the nature of extensions in area and annex populations which had been previously outside the cities.

Estimates of population for intercensal and postcensal years for states and cities of the United States can usually be obtained from the Director of the Census. If they cannot be thus obtained it is probably because the data are not available from which a reliable estimate can be computed.

MARRIAGE STATISTICS

Marriage statistics are of interest because of the information they give regarding the social life of the people and the establishment of families and households, and because of the relation of marriages to population growth through their influence on the birth rate. Their consideration naturally precedes that of birth statistics.

The data for marriage statistics are obtained by the registration of marriages. The common custom in the United States is to require persons desiring to marry to obtain first a license from a designated official. This license is presented to whomever performs the marriage ceremony. The person officiating is required to register the marriage. Those responsible for the completeness of marriage records are therefore in this country usually the clergy and justices of the peace. There is seldom much difficulty in securing complete records of marriages, and the amount and value of the information given by marriage statistics depend upon the nature and extent of the facts recorded relating to the contracting parties.

In England and Wales marriage statistics are compiled by the registrar general of marriages, births, and deaths. In this country the official responsible for the compilation of marriage records varies in the several states. The United States Bureau of the Census compiled and published in 1909 statistics of the number of marriages and divorces in the United States from 1867 to 1906. It has also compiled the number for the year 1916 and for the year 1922.

Marriage Rates.—Marriage rates may be expressed as the number of marriages for each 1,000 population. While this method gives certain information of a definite character and is useful for comparing different years of the same community and different communities of similar population composition, it is not useful in comparing populations in which the proportion of single persons of marriageable age is not the same. For the purpose of comparing marriage rates, therefore, the more exact method is to express the rate as the number of marriages or persons married for each 1,000 unmarried, divorced, and widowed, of marriageable age, usually those over fifteen years of age.

Factors Influencing Marriage Rates.—Marriage rates are usually influenced by economic conditions. National prosperity increases the rate, economic depression reduces it. For the same reasons it is influenced by the demand for labor and the rate of wages. The relation of the adopted standard of living to the average wage has a similar effect. In the absence of other factors, the marriage rate is usually a fair index of the relation of average income to standard of living.

The marriage rate may also be affected by the frequency of divorce and remarriage. A high birth rate tends to increase the marriage rate in succeeding years. In communities such as mining towns and new industrial

centers the marriage rate may be limited by the presence of a relatively small number of marriageable women.

The marriage rate in a city may be fictitiously high, due to the fact that many couples from the surrounding country and small towns may go there for the purpose of being married, returning then to their homes. In a country affected by emigration a relatively large proportion of the emigrants are likely to be young men and women, the women frequently following after the men have become located. This naturally affects the marriage rate of the home country.

Uses of Marriage Registration.—The purpose of the registration of a marriage is largely to protect the home and family. It furnishes reliable evidence upon which to base the legitimacy of children and the dower rights of women.

BIRTH STATISTICS

Statistics of births are of interest mainly because of their relation to population growth, the excess of births over deaths being known as the "natural increase." Growth of population has been the object of concern to nations largely because of its effect in determining the future military strength and the number of men available for purposes of offense and defense. The practically stationary population of France has for some time been the subject of comment, but with her limited territory it is a question whether the people as a whole are not better off with the present population than they would be with a larger one. More people mean greater congestion and more intense competition. During the last century Great Britain, Germany, Austria, and Russia have trebled in population. Had France done the same, she would now have nearly 80 millions of people, and it is doubtful whether this would have added to the happiness and welfare of the race except that it would probably have been of advantage to her in the late World War and would be an added factor of security for the future.

It is undoubtedly better to have a people proportionate in number to land area and natural resources than to have a teeming population with the consequent economic problems. It would seem more in keeping with modern ethics to strive for a people composed of intelligent, physically sound individuals free from disease and properly housed, fed and clothed, whose days furnished time for both labor and recreation under conditions which conduced to physical and mental welfare and not to deterioration, rather than to strive for mere numbers.

To the health officer and sanitarian birth statistics have only casual interest. Birth registration, however, which furnishes the data from which the statistics are made, is important not only in public health work but in other ways as well.

Registration in the United States.—In legislation the registration of births, marriages, and deaths was formerly usually associated and provided for by the same laws. Since 1900, however, this has not been generally true

in the United States, where the practice has developed of providing separately for the registration of births and deaths.

A model bill for the registration of births and deaths recommended for enactment by the several state legislatures has been drafted and indorsed by the American Medical Association in consultation with representatives of the Bureau of the Census, the Children's Bureau, the American Public Health Association, the American Bar Association, and a number of other organizations and societies national in scope. The essential features of this law have been adopted by a number of states. It is important that other states should also enact it, for it is without question as effective a law as any that has been proposed for adoption in this country. It is also highly desirable that the laws of the several states on the subject be uniform, if the Bureau of the Census is to compile the records for statistical purposes. The power to legislate on such matters resides with the individual states. The only means the Bureau of the Census has of preparing national birth and death statistics is to compile the records registered in the several states under state laws. This is done by making copies of the birth and death certificates registered in the various states and from these copies taking the data for statistical tabulations. The adoption of a uniform law would therefore have distinct advantages, even if it were possible for state legislatures individually to draft better ones.

United States Registration Area for Births.—A registration area for births was designated by the Bureau of the Census, beginning with the year 1915. The area in April, 1925, included 33 states and the District of Columbia, comprising in all 76.1 per cent of the total population of the United States. The statistics of births have been compiled for this area from transcripts made of the birth certificates filed in the respective states in the same manner that mortality statistics are compiled from state records.

Source of Data.—While the data from which population statistics are derived are obtained by direct enumeration, the data from which birth statistics are compiled are gotten by registration. The usual requirement is that whenever a child is born either the attending physician or midwife, or, in their absence, the parents or the head of the household in which the birth occurred, shall register with an official designated for the purpose certain information regarding the child and its parents.

Nature of Information Secured by Registration.—The information required to be registered concerning each child born usually includes certain facts relating to the child and the circumstances of its birth, together with certain items concerning the parents. The essential facts are the name of the child, its sex, date and place of birth, and whether born alive or stillborn, and the names and residence of the parents. There are many other items of information concerning births which are of the greatest value and serve various purposes, such as the age, color, nativity, and occupation of the parents, whether the child is a single birth, a twin, or a triplet, and whether legitimate or illegitimate. These facts are usually required to be stated. Copies of the

standard certificate of birth for the United States may be obtained from the Superintendent of Documents, Government Printing Office, Washington, D. C.

The items registered serve two principal purposes. They serve, first, to identify the child and to establish its age and parentage, and second, to furnish statistical data.

While in the enumeration of the population the original observer, upon the accuracy of whose work population statistics largely depend, is the census enumerator, in birth registration the original observer, upon whom dependence must be placed, is usually the physician attending at the birth, sometimes the midwife, and in the absence of these the parents.

Births are usually required to be registered with an official appointed for the purpose and known as a registrar. Customarily it is the same official with whom deaths are registered. At times a small fee has been paid to the person making the registration or filling out the certificate. This custom, however, is likely to create in the minds of many the idea that the registration is a matter of discretion—that if the fee is not wanted there is no compulsion to file the certificate and that the forfeiting of the fee annuls the obligation. This is especially true in some parts of the United States, where physicians and midwives have in many instances not yet come to realize that the importance of proper registration may mean so much to the child and its parents that no accoucheur has completed his task nor fulfilled his obligations to the child and its mother until an accurately filled out certificate has been filed with the registrar. The failure to file such a certificate is such a neglect of the interests of both patients, the child and the mother, that it would seem proper to class it with malpractice.

Birth Rates.—There are several ways of expressing the birth rate. Each method of statement gives information not given by the others.

Rate per 1,000 Population.—The birth rate may be expressed as the number of births occurring during a year for each 1,000 of the population. This is known as the crude birth rate, and is based upon the total estimated mean population for the year—that is, for the calendar year, the population estimated as of July 1. The crude birth rate shows the net result to the community of the several factors governing reproduction—the number of women of child-bearing age, the number of those who are married, the frequency of illegitimacy, etc. In conjunction with the crude death rate it shows the ratio at which the community is reproducing itself by natural increase. It is a quite satisfactory basis for comparing the birth rate of different years for the same community or that of different communities having populations of similar composition. It is unsatisfactory for the comparison of populations having different proportions of females of child-bearing age or of married women—a mining town or new industrial center may have comparatively few women; a fashionable residential district may have a relatively large female population, most of which consists of unmarried servants.

Rate per 1,000 Women of Child-bearing Age.—Birth rates may be ex-

pressed as the number of births occurring during the year per 1,000 women of child-bearing age. For this purpose the female population between the ages of fifteen and forty-five years as determined by census enumeration, or by estimation for intercensal and postcensal years, is taken. The proportion of women of these ages in the population having been ascertained by a census, the same relative proportion is assumed to be maintained until a succeeding census shows a change.

This method gives rates that furnish a much better basis for the comparison of different communities, inasmuch as it gives the births in proportion to the number of potential mothers. It is not, however, satisfactory under all conditions, and the method next described yields more useful information.

Rate of Legitimate Births per 1,000 Married Women of Child-bearing

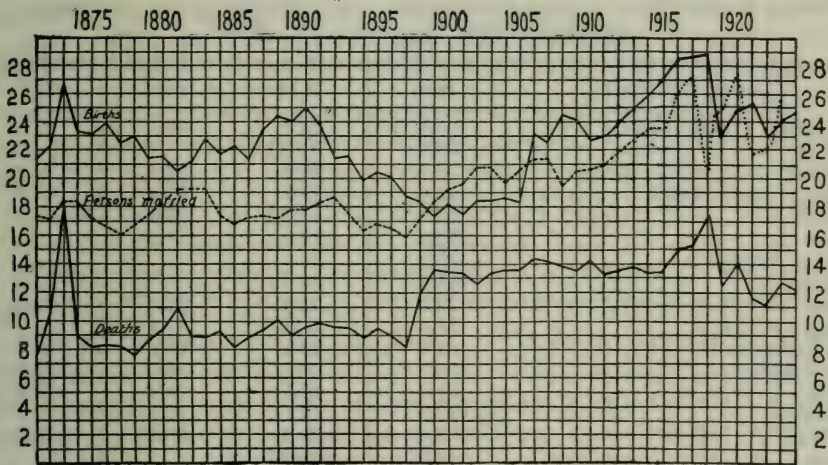


FIG. 118.—BIRTHS (INCLUDING STILLBIRTHS), PERSONS MARRIED, AND DEATHS (EXCLUDING STILLBIRTHS) REGISTERED PER 1,000 POPULATION PER ANNUM IN MICHIGAN, 1871-1924.

Age (15 to 44 or 15 to 49 Years of Age) and of Illegitimate Births per 1,000 Unmarried Women of Child-bearing Age.—In different communities the proportion of married and single women may differ considerably and consequently comparison of their crude birth rates or of rates based on the number of women of child-bearing age would yield comparatively little useful information. The proportion of married women in industrial communities is usually considerably larger than it is in residential suburbs, where there are greater numbers of female servants. To make allowance for these differences in population composition the most useful method of stating the birth rate is in terms of the number of legitimate births per 1,000 married women of child-bearing age (15 to 44 years or 15 to 49 years) and the number of illegitimate births per 1,000 unmarried women of this age.

Sources of Error in Birth Statistics.—The principal sources of error in birth statistics are to be found in defective registration. There is no reliable

check by which the failure to register births can in all cases be detected. In many foreign countries the people have become accustomed to register births and apparently their returns are quite complete. The registration of illegitimate births, however, is always less complete than that of the legitimate. In the United States the people, as a whole, have in many sections not become accustomed to the registration of births. This is undoubtedly due in some instances to a rapidly changing local population continually receiving large numbers of immigrants from various foreign countries—immigrants who are ignorant of our registration laws and may have little opportunity of learning their requirements—and at times possibly to the absence of effort by the authorities to enforce the laws.

As checks upon the completeness of birth registration registrars frequently

BIRTH RATES (EXCLUSIVE OF STILLBIRTHS) PER 1,000 POPULATION IN CERTAIN COUNTRIES, 1886, 1913, 1914 and 1915 *

Country or State	1886	1913	1914	1915
Australian Commonwealth	35.4	28.3	28.1	27.3
Austria	38.3	31.3 ‡
Denmark	32.4	25.6	25.6	24.2
England and Wales	32.8	24.1	23.8	22.0
Finland	35.3	27.1 ‡	26.9
France	23.9	19.0	18.0
German Empire	37.0	27.5
Hungary	45.6	36.3 ‡
Ireland	23.2	22.8	22.6	22.0
Italy	37.0	31.7	31.1
The Netherlands	34.6	28.1	28.2	26.2
New Zealand	33.1	26.1	26.0	25.3
Norway	31.2	25.3	25.2	23.8
Roumania	42.2	42.1	42.5
Scotland	32.9	25.5	26.1	23.9
Servia	42.0	38.0 ‡
Spain	36.7	30.4	29.8
Sweden	29.8	23.2	22.9	21.6
United States				
Connecticut	22.2 †	25.6	26.5
Michigan	21.3 †	24.8	25.6	26.6

* Taken from the Annual Reports of the Registrar General of Births, Deaths, and Marriages in England and Wales, 1913, 1914 and 1915, except the rates for Connecticut and Michigan, which were taken from the State reports.

† Includes stillbirths.

‡ Year 1912.

use the death returns of young children and especially of infants, checking up each recorded death with the birth records to see whether the birth of the child had been registered. The notices of births appearing in newspapers are also often used for the same purpose. If christenings were required to be reported by those officiating, this too would be of assistance.

Uses of Birth Registration and Statistics.—Birth statistics are of use in ascertaining the natural increase of the population (excess of births over deaths). They also give valuable information regarding the effective fertility or fecundity of the race and of the frequency of illegitimacy. These matters are of interest to the economist and the statesman. The possession

of birth statistics also furnishes the basis for the present accepted means of stating the infant mortality rate, as will be explained later. The data from which the statistics are made, the registered births, are, on the other hand, of value to the community in many ways, and to the health officer among others may be especially useful. Some of the uses will be enumerated.

Legal Record.—The registration of a child's birth forms a legal record that is frequently useful and may be of the greatest importance. It establishes the date of birth and the child's parentage and legitimacy. It may be required to establish the child's age for attendance at public schools, for permission to work in states where children below a certain age are not allowed by law to be employed; to show whether a girl has reached the age of consent, whether individuals have attained the age when they may marry without the parents' permission; to establish age in connection with the granting of pensions, military and jury duty, and voting. It may be necessary in connection with the bequeathing and inheritance of property or to furnish acceptable evidence of genealogy, and in fact may be important and useful in possible events too numerous to mention. During the period of mobilization for the late war, there were many instances where the interests of individuals were curiously affected because of there being no dependable record of the time and place of their births.

Uses in Public Health Administration.—Registration of births shows where the babies are and makes possible such observance and protection as the health department desires to extend. With birth registration it would be possible for the health authorities to see that the babies are vaccinated against smallpox. This is one of the uses made of registration in England. It would also be possible to see that the babies in poor families have proper food and adequate attention. The observation of infants under two weeks of age would bring to light some cases of ophthalmia which otherwise might cause serious injury to vision and at times total blindness.

Factors Influencing Birth Rates.—Birth rates are directly influenced by the number of women, and particularly of married women, of child-bearing age in the population. The child-bearing period of life for women may be considered as that between the ages of fifteen and forty-nine years; the ages between twenty-five and forty-four years are for most races of the north temperate zones, however, those mainly productive.

The economic and social status of the population may also affect the birth rate. In many countries at present the poor families have considerably more children per family than have the well-to-do; in fact to some extent the number of children per marriage seems to be in inverse ratio to the family income. On the other hand, to a degree, poor economic conditions are liable to discourage or delay marriage, so that married couples are relatively fewer and older when married, with fewer resulting offspring. The adoption of a more expensive standard of living may produce the same results as depressed economic conditions, fewer and delayed marriages.

The birth rate is also affected by the habits and customs of the people,

by their desire to have children or their desire not to have them. Also a high infant death rate is usually accompanied by a high birth rate and, conversely, a low infant death rate by a low birth rate.

MORBIDITY STATISTICS

Morbidity statistics are the statistics of sickness and disease. They show the occurrence of diseases and their relative prevalence in different localities and at different times. They differ from mortality statistics in that as relates to disease, mortality statistics are the statistics of fatal cases only, while morbidity statistics include all cases. For example, if in a city there were 500 cases of typhoid fever, of which 50 terminated fatally, mortality statistics would deal with the facts relating to the 50 fatal cases, while morbidity statistics would deal with the entire 500.

In the life of the individual, after birth, the next event included in vital statistics which usually occurs is sickness. Disease has perhaps a greater influence in determining the happiness and efficiency of the individual and of the community than any other factor. It also has a direct bearing on the individual's longevity even when in itself not fatal, for every attack of sickness probably does some injury and leaves the human machine impaired to a degree, and an illness occurring a number of years before death may have a far greater influence in determining the duration of life than the terminal illness.

Morbidity statistics have not evolved apace with those of births, marriages and deaths. This is due to the different purposes they serve. The branches which have to do directly with the growth of population were first developed, probably because of the need of the information which they gave in connection with taxation and military enlistment. Morbidity statistics, on the other hand, are contemporary with our comparatively recently acquired knowledge of the causes of diseases and their manner of spread. Their need has been felt only with the advent of present-day public health administration, which in turn has been activated in large measure by the story of the causes of death told by mortality statistics.

Morbidity statistics had their origin in the requirement of the notification of cases of certain dreaded diseases, notably smallpox. With the appointment of health officers and the establishment of health departments the notification of other diseases has been required. As knowledge of the causes of diseases and their manner of spread has been obtained and health departments have been faced with the responsibility of controlling maladies found to be preventable, the list of notifiable diseases has grown, for those responsible for public health administration have found that it is impossible effectively to control a disease without prompt information of when, where, and under what conditions cases of the disease are occurring. No epidemiologist would think of attempting to control an outbreak of smallpox or plague without inaugurating a dependable system whereby he would receive

prompt and accurate information of the occurrence of cases. It is just as impossible effectively to control tuberculosis, typhoid fever, scarlet fever, industrial lead poisoning, or any other preventable disease without a knowledge of the occurrence of cases.

The requirements for notification of the preventable diseases and the extent of their enforcement may be taken as one index of the intelligence and efficiency of health administration in a community.

Morbidity Statistics in the United States.—*Present Status.*—In the United States the authority to require the notification of cases of sickness resides in the respective state legislatures. In some of the states authority has been given to the state boards of health to cover the subject by regulations. In most instances local authorities have the right to supplement the state requirements by such additional ones as may be needed. The laws and regulations of the several states differ widely, as do also the efforts made to enforce them.

The common and most general plan is to require that the original report be made by the physician to the local health officer immediately on diagnosis of the case. The local health officer forwards to the state health department, either immediately or at intervals, a transcript or a summary of the notifications received by him. In a number of states these reports by the local health departments are made to the state authorities daily, in some weekly, in several states monthly, and in a few states at longer intervals. In the states in which the reports are made daily the state health department is in a position to keep constantly informed regarding the prevalence of the notifiable diseases. The same is in less measure true when the reports are made weekly. When the reports are made at longer intervals the current value of the information to the state department is largely lost.

In certain states physicians have been required to report the notifiable diseases directly to the state health department. This, in fact, makes the state health officer also the local health officer and responsible for the control of the notifiable diseases, the control of disease and the notification of cases being inseparable, the latter giving the necessary information by which to direct action in the former.

In some states the laws relating to morbidity reports specify that cases of certain classes of disease shall be notifiable. These classes have been variously stated, the wording being in some instances that "all cases of contagious or infectious diseases dangerous to the public health shall be reported," in others "all communicable diseases," or "all contagious diseases," or "all diseases dangerous to the public health." When the requirements have been stated in general terms in this way their enforcement has been especially difficult unless the diseases included have been specifically enumerated.

The Model State Law for Morbidity Reports.—Since each state has exclusive authority within its jurisdiction over the requirements for the notification of disease, any comprehensive plan that may be developed for morbidity reports and morbidity statistics must be the result of combined

effort and coöperation and the enactment by the several states of similar requirements. It implies also an adequate enforcement of these requirements. The question of state morbidity reports is one of the most difficult problems to be solved by the state authorities. A number of states have been endeavoring earnestly to solve the problem within their respective jurisdictions. Considerable progress has been made in several instances. The question is an important one, and is bound to receive much consideration in the future. The state health authorities in conference with the Public Health Service had the matter under consideration for some time and in June, 1913, approved a model state law for morbidity reports. The model law as originally drafted made the occurrence of cases of the following-named diseases and disabilities notifiable:

GROUP 1.—*Communicable Diseases*

Actinomycosis	Ophthalmia neonatorum (conjunctivitis of newborn infants)
Anthrax	Paragonimiasis (endemic hemoptysis)
Chickenpox	Paratyphoid fever
Cholera, Asiatic (also cholera nostras when Asiatic cholera is present or its importation threatened)	Plague
Dengue	Pneumonia (acute)
Diphtheria	Poliomyelitis (acute infectious)
Dysentery:	Rabies
(a) Amebic	Rocky Mountain spotted or tick fever
(b) Bacillary	Scarlet fever
Favus	Septic sore throat
German measles	Smallpox
Glanders	Syphilis
Gonococcus infection	Tetanus
Hookworm disease	Trachoma
Leprosy	Trichinosis
Malaria	Tuberculosis (all forms, the organ or part affected in each case to be specified)
Measles	Typhoid fever
Meningitis:	Typhus fever
(a) Epidemic cerebrospinal	Whooping-cough
(b) Tuberculous	Yellow fever
Mumps	

GROUP 2.—*Occupational Diseases and Injuries*

Arsenic poisoning	Naphtha poisoning
Brass poisoning	Bisulphid of carbon poisoning
Carbon monoxid poisoning	Dinitrobenzine poisoning
Lead poisoning	Caisson disease (compressed-air illness)
Mercury poisoning	Any other disease or disability contracted as a result of the nature of the person's employment
Natural-gas poisoning	
Phosphorus poisoning	
Wood alcohol poisoning	

GROUP 3.—*Miscellaneous Diseases*

Beriberi	Drug addictions or habits
Cancer	Pellagra
Continued fever lasting seven days	

The Results of Notification in Certain States and Cities.—The completeness of the reports of the notifiable diseases in states and cities in which there is registration of deaths may be estimated with some degree of accuracy by comparing the number of cases reported with the number of deaths registered as due to the same cause. In doing this, however, it must be borne in mind that we do not know the fatality rates of many diseases, for up to the present time there have seldom been satisfactory morbidity records of sufficiently broad application to permit of the determination of such rates, and it must also be remembered that the fatality rates of many diseases

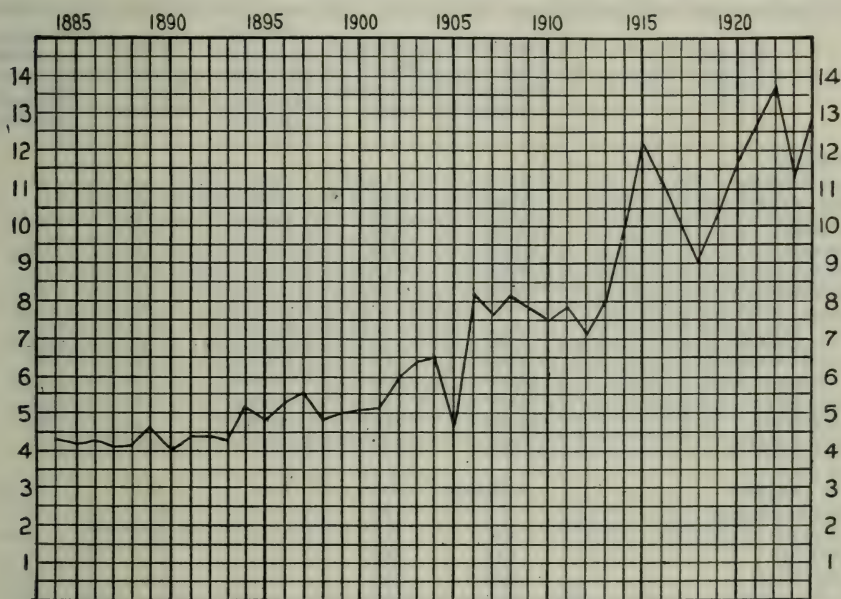


FIG. 119.—DIPHTHERIA.

Number of cases notified per annum for each death registered in Michigan, 1884-1924.

vary in different epidemics, and from year to year, and with the season and geographic location. See also table, page 1150.

Source of Statistical Data.—The manner of collecting the data from which morbidity statistics are compiled is closely allied to the registration method used for births. The data consist of the reports of cases of disease made usually by physicians and in some instances by the heads of families and households. The original observers then, upon whom morbidity statistics depend chiefly for their completeness, are the practicing physicians. This is necessarily so, for neither the health department nor any other branch of government can keep in such close touch with the lives of the people as to be in a position to know of the occurrence of disease. The physician is the one who, because of the very nature of his work and his relation to the community, is best able to have this information and furnish it. He comes in

contact with the sick to a degree that others do not. The health officer cannot know of the presence of disease except as it is reported to him by physicians. Experience has shown that there may be hundreds of cases of a dangerous infection in a city and the health officer not know of its presence, in the absence of notification.

DIPHTHERIA, MEASLES AND TYPHOID FEVER

(Cases Reported, Deaths Registered, Indicated Case Rates per 1,000 Population, Indicated Fatality Rates per 100 Cases, and Number of Cases Reported for Each Fatality Registered, in Certain Cities, 1923)

Diseases and Cities	Number Cases Reported 1923	Number Deaths Registered 1923	Indicated Case Rate per 1,000 Inhabitants	Indicated Fatality Rate per 100 Cases	Number of Cases Reported for Each Fatality
Diphtheria					
Los Angeles	2515	119	3.77	4.7	21
San Francisco	1708	148	3.17	8.7	11
Chicago	5836	365	2.02	6.3	16
Detroit	2424	205	2.43	8.5	12
New York	8050	553	1.36	6.9	15
Philadelphia	3310	270	1.72	8.2	12
Measles					
Los Angeles	3688	21	5.53	0.6	176
San Francisco	3908	21	7.25	0.5	186
Chicago	15168	206	5.26	1.4	73
Detroit	4597	85	4.62	1.8	54
New York	13999	245	2.36	1.8	57
Philadelphia	7997	223	4.16	2.8	36
Typhoid					
Los Angeles	171	22	0.26	12.9	8 —
San Francisco	73	17	0.14	23.3	4 +
Chicago	473	57	0.16	12.1	8 +
Detroit	134	42	0.13	31.3	3 +
New York	890	140	0.15	15.7	6 +
Philadelphia	271	29	0.14	10.7	9 +

Unfortunately many practicing physicians have little knowledge of the methods of health administration and in common with people in general frequently expect the health department in some mysterious manner to control disease without placing upon them the burden and privilege of co-operating by the notification of the occurrence of cases. The practicing physician, whether he recognizes it or not, or is so recognized by the community, is essentially an adjunct of the health department, for, unless he performs his part, the health department is in large measure helpless.

Among practicing physicians, at least in the United States, there has at times been the feeling that the knowledge of a disease in a patient is privileged information which they should not be called upon to impart. In communities where the laws require the notification of the disease this feeling has no legal basis and the physician who does not make report is not a law-abiding citizen. But aside from the legal aspects of the matter there would seem to be little justification for such a course. Every physician has a

number of individuals or families who look to him, and properly so, not only for treatment, but also for such reasonable protection from disease as he is able to give. The failure to report the occurrence of a case of communicable disease in one patient may lead to its spread to others among his clientele whose rights he has ignored. He therefore violates the intent and spirit of the ethical principle of the protection of patients among whom must be considered the well together with the sick. The notification of disease is in the interests and for the protection of the community, and as his patients are members of the community their interests are ignored and, because of the anti-social whim or supposed convenience of the individual affected with a notifiable disease, they are deprived of the protection they have a right to expect. It would seem that the physician who fails to report his cases of preventable diseases required to be notified may properly be considered as actively obstructing public health administration.

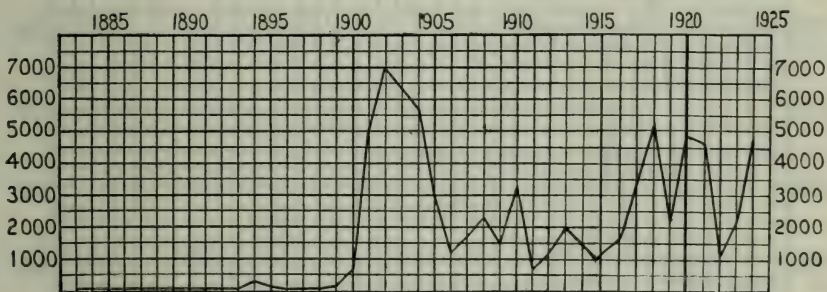


FIG. 120.—SMALLPOX.

Number of cases notified per annum in Michigan, 1883-1924.

The health department laboratory may be, and in many places is, an important factor in giving information of the occurrence of cases and prevalence of certain diseases. By having a diagnostic laboratory with a trained personnel at the service of the practicing physician the health department becomes not only a consultant performing gratuitous service for the physician but at the same time secures early and accurate information of many cases which otherwise might not be properly diagnosed and therefore not reported. A record of every positive diagnosis made by the laboratory should be sent to the epidemiological bureau or other division of the health department responsible for the control of disease and should for purposes of morbidity records constitute notification of the case when accompanied by such necessary information as the name, age, sex, and address of the patient. There would seem to be no good reason why the services of the health department should not be at the disposal of the community for the diagnosis of all diseases.

Nature of Information Secured by Morbidity Notification.—It is the practice for health departments to furnish to physicians notification blanks

upon which the reports are to be made. In some instances these are in the form of post cards, which have proper spaces indicated for notation of the required information. These cards require the physician to affix a stamp before mailing them to the health department. A far better practice is that employed by many states and cities, through coöperation with the U. S. Public Health Service, of supplying physicians with postal card forms which do not require postage before mailing.

The information relating to the reported cases which physicians are required to give varies in the several states. It has been customary to require the physician, in making his report, to include all the data regarding the



FIG. 121.—MEASLES.

Number of cases notified per annum for each death registered in Michigan, 1890-1924.

case desired by the health department. In the majority of instances no further data regarding these cases are secured by the health officials. While it may be impracticable in most instances to change this practice at the present time, it must be recognized that a local health department should prefer to collect its data regarding each case itself, and should not be willing to depend upon the physician's report for its epidemiologic information. Logically, the only information which the physician should be depended upon to give in his report is the occurrence of a case, or a suspected case, of a given disease in such and such a person at such and such an address. He might properly be required to add to this such data as are matters of record or easily verified, such as the age, color, and sex of the patient, and similar information. The local health department, however, should be reluctant to depend upon the diagnosis of the practicing physician, unless the diagnosis has been veri-

fied by a trained diagnostician in the service of the department itself. This has been the practice during recent outbreaks of such diseases as yellow fever and plague. It is also the practice in certain other instances. It must necessarily become the practice whenever a determined effort is to be made in the control of any preventable disease.

The Standard Notification Blank.—The standard notification blank approved by the state and territorial health authorities of the United States in conference with the Public Health Service at their tenth annual conference in June, 1913, calls for the following information:

1. Date.
2. Name of disease or suspected disease.
3. Patient's name, age, sex, color, and address. (This is largely for purposes of identification and location.)
4. Patient's occupation. (This serves to show both the possible origin of the disease and the probability that others have been or may be exposed.)
5. School attended by or place of employment of patient. (Serves same purpose as the preceding.)
6. Number of persons in the household, number of adults and number of children. (To indicate the nature of the household and the probable danger of the spread of the disease.)
7. The physician's opinion of the probable source of infection or origin of the disease. (This gives important information and frequently reveals unreported cases. It is of particular value in occupational diseases.)
8. If the disease is smallpox, the type (whether the mild or virulent strain) and the number of times the patient has been successfully vaccinated, and the approximate dates. (This gives the vaccination status and history.)
9. If the disease is typhoid fever, scarlet fever, diphtheria, or septic sore throat, whether the patient had been or whether any member of the household is engaged in the production or handling of milk. (These diseases being frequently spread through milk, this information is important to indicate measures to prevent further spread.)
10. Address and signature of the physician making the report.

These reports are to be made on postal cards furnished for the purpose and mailed immediately to the local health department, so that proper measures can be taken to prevent the spread of the disease or to find the focus or source from which the case originated, that the occurrence of additional cases may be prevented. These reports are then to be forwarded to the state department of health; but before being forwarded the local health department is to note thereon:

1. Whether the case was investigated by the local health department.
2. Whether the nature of the disease was verified.

3. What measures were taken by the local health department to prevent the spread of the disease or the occurrence of additional cases from the same origin.

Sources of Error in Morbidity Statistics.—The errors in morbidity statistics of civil populations are due principally to incomplete notification—that is, to the failure of physicians to report all cases of the notifiable diseases. More cases of disease usually occur than are reported. This can never be entirely overcome, for many diseases vary in severity under different conditions, and some cases are so mild that their true nature is not recognized, and frequently they do not come to the attention of physicians.

The cases notified are usually correctly diagnosed, for physicians do not generally report cases until they are practically sure of the diagnosis, as

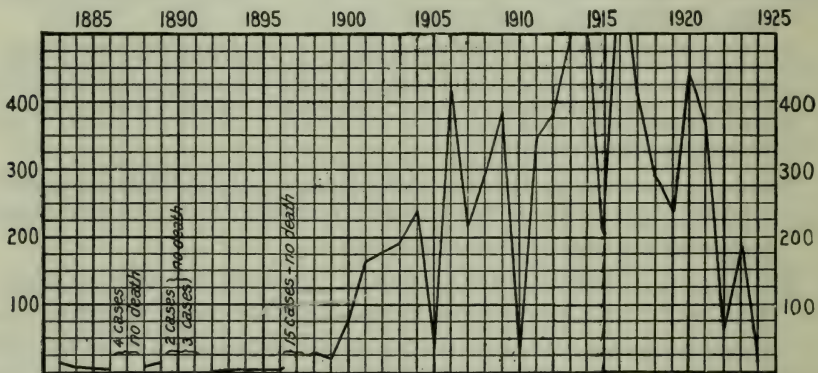


FIG. 122.—SMALLPOX.

Number of cases notified per annum for each death registered in Michigan, 1883-1924.

In 1914 there were 1,527 cases notified and only one death registered.

the case remains an evidence of faulty diagnosis if a mistake is made. Then, too, physicians naturally wish to report only those cases required and to know whether a given case is one of these he must first be reasonably sure of his diagnosis.

The errors in morbidity statistics are therefore chiefly those of incompleteness. In this they resemble birth statistics, although the degree of incompleteness, due to the difference in the nature of the two, is usually greater in morbidity statistics.

They differ from mortality statistics, in which the principal source of error is incorrect statements of cause of death. Due to the control possible over the disposal of bodies of the dead, it is not difficult in most communities to obtain practically complete registration of deaths. It is, however, exceedingly difficult to secure correct statements of the causes of death. The physician feels compelled to give a diagnosis in each death certificate and usually does so even when he is uncertain of the nature of the malady.

The tendency is then in morbidity reports for the diagnoses to be cor-

rectly given, but not all cases reported, while in the registration of deaths the tendency is for the recording of practically all deaths but the filing of many incorrect statements of the causes of death.

Uses of Morbidity Reports and Statistics.—In health administration, morbidity reports—that is, reports of cases of sickness—serve several purposes, which may be briefly stated to be as follows:

1. In the communicable diseases morbidity reports show the occurrence of cases which constitute foci from which the disease may spread to others, as in scarlet fever, typhoid fever, tuberculosis, or yellow fever, and make it possible to take proper precautions to protect the family of the patient, his associates, or the community at large.

2. In some diseases morbidity reports make it possible to see that the sick receive proper treatment, as in ophthalmia neonatorum, diphtheria, and,

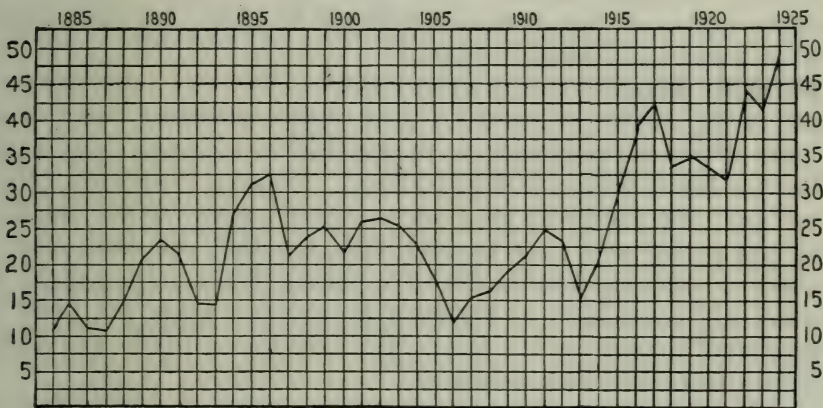


FIG. 123.—SCARLET FEVER.

Number of cases notified per annum for each death registered in Michigan, 1884-1924.

in certain cities, tuberculosis. The reporting of cases of ophthalmia in the newborn makes it possible to save the sight of some infants who would otherwise not receive adequate treatment until after much damage had been done. In diphtheria the health department can be of service in furnishing antitoxin. Some cities furnish hospital or other relief to consumptives who would otherwise be without proper treatment.

3. In diseases that are not communicable, such as those due to occupation or environment, reported cases show the location of conditions which are causing illness or injury. This makes it possible to remedy the faulty conditions, so that others may not be similarly injured.

4. In certain diseases, of which the cause or means of spread is unknown, morbidity reports show their geographic distribution and varying prevalence and the conditions under which cases occur. This information has great potential value in attempts to ascertain their causes and means of spread.

5. Reports of the occurrence of disease are necessary to show the need

of certain sanitary measures or works and to control and check the efficiency of such measures or works when put into operation. In pulmonary tuberculosis such reports show the number of consumptives in the community and the need of sanatoria. In malaria they show the prevalence of the disease, the need for drainage and other anti-mosquito work, the efficiency of such work when in operation, and when a change in the prophylactic measures or additional ones are necessary. In typhoid fever they show faults in the water supply or in the control of the production and distribution of milk or in the disposal of excreta in special localities.

6. Morbidity reports when recorded over a period of time and properly compiled become a record of the past occurrence of disease. They show the relative prevalence of disease from year to year and under varying conditions. They show the effect of the introduction of public health measures and of sanitary works. They give a history of disease not obtainable in their absence. Morbidity reports are very much more difficult to collect, but when accurate are more useful than mortality figures.

Morbidity Rates.—*Crude Morbidity Rates.*—Morbidity rates may be expressed as the number of cases of a given disease occurring during a year per 1,000 of the total population, or the rate may be expressed as the number of cases per 10,000 or per 100,000 population. These are crude rates. Giving the rate per 1,000 population has the advantage of employing the same population unit as that used for expressing birth, marriage, and death rates. It has, however, what has been considered by some a disadvantage, namely, that the rates will occasionally be expressed by fractions where the 1,000 unit of population is taken as the basis. For this reason 10,000 and 100,000 population units have often been used.

Specific Morbidity Rates.—Diseases limited entirely or principally to certain ages or to certain classes of the population should be expressed also in rates of the number of cases per 1,000 persons in the population of that age or class. Diseases limited to childhood should be expressed as rates per 1,000 children; diseases limited to women should be expressed as rates per 1,000 women. Occupational disease rates should be expressed in terms of the number of cases per 1,000 persons employed. These are specific rates.

Specific morbidity rates showing the incidence of disease by age groups, sex, occupation, and economic or social condition will be possible with the improved notification methods which are being gradually adopted.

Case Fatality Rates.—The case fatality rate of a disease is usually expressed in terms of the number of deaths per 100 cases; that is, as the percentage of cases which terminate fatally. For example, if out of 100 cases of typhoid fever 10 die, the case fatality rate is 10 per hundred or 10 per cent. In calculating fatality rates it is to be borne in mind that among cases reported during one week, month, or year, all or part of the fatal terminations may occur during a succeeding week, month, or year.

Hospital Statistics and Sickness Insurance Records.—In a number of foreign countries much valuable information regarding sickness rates, aside

from that of the commonly notifiable diseases, is being secured from the workingmen's sickness insurance records. In some countries hospital statistics are compiled and furnish data of much value. Bolduan¹ suggested a plan for compiling hospital morbidity statistics in this country. The method is especially applicable to the hospitals of a large city, but might be used for the hospitals of an entire state and is capable of being made nation-wide in scope. The essential feature of the plan is the filling out of "discharge certificates," analogous to ordinary death certificates, on the discharge of each patient from a hospital. These discharge certificates are then to be sent to a central filing bureau, preferably the health department, and there classified and analyzed.

It is also especially desirable to have statistics of the insane and mentally defective. New Jersey has enacted a law requiring the notification of cases of mental deficiency and of epilepsy.

Factors Influencing Morbidity Rates.—The factors which influence morbidity rates and the prevalence of sickness are the manifold direct and indirect causes of disease. There are certain widely acting indirect factors which increase morbidity by lessening individual resistance. There are other factors which are specific for individual diseases. In malaria the direct cause is infectious anopheline mosquitoes, and the indirect cause swamps and stagnant water in which the mosquitoes breed. The factors influencing typhoid fever rates are commonly the milk supply, the water supply, the manner of disposal of excreta, presence of flies, the extent to which houses are screened, personal and social habits, etc. In an industrial community the morbidity from occupational diseases and from diseases caused indirectly by the conditions attending certain kinds of labor constitutes a factor the importance of which is beginning to be realized.

Notification of Occupational Diseases.—Most civilized nations have during the last hundred years undergone an industrial evolution. It has been within this period that the large factory with its hundreds or thousands of workers has had its development and that many of our present industries and the majority of our industrial processes have been developed. So great has been this change in the industrial life of the people that there has been developed a new and important branch of hygiene and sanitation which is properly termed industrial hygiene. With this industrial development there have evolved new diseases and disabilities due to the nature of the individual's work or to the conditions incident to the work. Not only have new diseases in a sense been evolved, but a number of diseases previously rare have become much more common. Under existing social conditions a large proportion of the people are engaged in some occupation, and the diseases of occupation merit the attention and consideration of the community. See section on Industrial Hygiene and Diseases of Occupation.

¹ Charles F. Bolduan, "Hospital Morbidity Statistics," *N. York M. J.*, March, 1913, p. 643.

The control of occupational diseases has during the last few years been receiving much consideration. Naturally the first step in the control of the industrial diseases was the securing of a means by which the occurrence and prevalence of these diseases might be known to those whose duty it would be to control them. For this purpose, and largely because of the activities of the American Association for Labor Legislation, a number of states have since 1911 enacted laws requiring the notification of certain occupational diseases.

A number of state laws require cases of occupational diseases to be notified to the state health department, and others require the notifications to be made to the state labor office. The results of notification have not been as yet satisfactory. This may be due to the newness of the idea to the physician of considering whether a disease is occupational in origin. The medical schools have given little attention to the subject. It is highly important to the practicing physician that he have a knowledge of the industries of his community and of the diseases and disabilities they are likely to cause. The proper and successful treatment of patients necessarily depends upon a knowledge of the direct or indirect cause of the individual's ailment, and in an industrial community this will depend frequently upon a knowledge of occupational diseases.

A number of states have enacted laws which should in a way be much more successful in bringing to light the occurrence of these diseases (Illinois, Missouri, Ohio, and Pennsylvania). The plan referred to is that of requiring certain industries to have their employees examined physically by competent physicians at stated intervals to ascertain whether there exist in the employees any ailments or disabilities due to the nature of their occupation. The physicians making these examinations naturally become in time expert, if they are not so in the beginning, and the examination of the employees in this way will guarantee the finding of a large proportion of the cases of industrial diseases, in most instances in their earlier stages. If the occupational diseases are to be controlled, it is necessary that the occurrence of cases be ascertained in some way, for the occurrence of each case shows the existence of conditions which have produced disease in one employee and will in all probability produce it in others. Each case notified shows a danger spot. (See also page 1216.)

Morbidity Statistics of Military Populations.—Military organizations offer a much better opportunity for the recording of morbidity data than do civil populations. In military organizations all persons are under constant supervision and all but the most trivial illnesses become a matter of record. This includes not only the cases of those diseases which are ordinarily reportable among civil populations but cases of other illnesses and disabilities as well. The limitations of military morbidity statistics are confined largely to the question of diagnosis, but even the dependability of diagnosis is probably greater in military organizations because the medical officers are a selected group and because of the added measures of control.

Military morbidity statistics may be exceedingly useful in determining the geographic distribution, and in a measure the relative prevalence, of certain diseases throughout the country. They may be of particular value for this purpose in localities where these diseases are not reportable or are at best very incompletely reported among the civil population. Thus records of the incidence of malaria at military posts and camps will give information not otherwise obtainable of the prevalence of this disease. This is particularly true under conditions existing in times of peace.

The value of military morbidity records to show the geographic distribution of disease is limited, however, largely to diseases which are endemic or pertain particularly to localities of which malaria is one of the most typical.

Some of the terms used in stating morbidity data of military groups differ from the corresponding terms used for civil populations. Thus in the navy the population of a vessel or other naval unit is known as the "complement." In the army the military population of a camp or other military group is commonly termed the "strength."

Admission Rate.—Ordinarily in military organizations every person reporting ill or disabled is considered as "admitted" to the sick list and the total number so reporting constitutes the total admissions. For some ailments a soldier or sailor may apparently recover and be discharged from the sick list, but have relapses and be readmitted in some instances several times. To this extent the total admissions do not show the total number of cases of disease or disability. The first admission of an individual for a case of illness constitutes the original admission and the subsequent admissions for the same illness or disability are "readmissions." The number of original admissions therefore gives the number of actual cases. The term "admission rate" corresponds to the term "morbidity rate" as used for civil populations, and is usually expressed as the number of original admissions during a year for each 1,000 of the "complement" or "strength" just as civil morbidity rates are expressed as the number of cases recorded during a year per 1,000 population. Thus an admission rate of 640 would mean that there had been 640 cases of illness or disability during the year for each 1,000 of the military population. This would be the crude admission rate. Admission rates might be given for classes of disease or disabilities or they might be given for special classes of the military population. These would be specific admission rates. This is illustrated by the following three tables, one taken from the annual report of the Surgeon-General of the Navy for the year 1924, the other two from the annual report of the Surgeon-General of the Army for the year 1924.

ADMISSION RATES

(By Age Groups, Enlisted Men of the Navy, for Disease Only, Injuries, Poisoning, and for All Causes Combined During Year 1923)

AGE GROUP	NUMBER IN GROUP	ALL DISEASES		INJURIES		POISONING		ALL CAUSES	
		Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000	Ad- mis- sions	Rate per 1,000	Admis- sions	Rate per 1,000
16-19	10,978	9,976	908.71	803	73.15	14	1.27	10,793	983.13
20-24	44,372	26,742	602.77	2,899	65.34	64	1.44	29,705	669.55
25-29	16,300	978	425.95	947	58.10	31	1.90	7,921	485.95
30-34	8,020	402	299.33	391	48.75	11	1.37	2,803	349.45
35-39	3,685	206	251.83	188	51.02	18	4.88	1,134	307.73
40-44	1,201	67	270.60	61	50.79	6	5.00	392	326.39
45-49	460	23	297.83	22	47.83	1	2.17	160	347.83
50-54	135	7	370.37	7	51.85	0	0	57	422.22
55-59	48	15	312.50	0	0	0	0	15	312.50
60-64	14	4	285.71	0	0	0	0	4	285.71
65-69	2	0	0	0	0	0	0	0	0
70-74	1	0	0	0	0	0	0	0	0
Unknown	1	0	0	0	0	0	0	0	0
TOTAL	85,217	47,521	557.42	5,318	62.38	145	1.70	52,984	621.50

Non-effective Rate.—The members of a military organization who are unable to perform their regular military duties because of illness or disability are known as non-effectives. The average proportion of any organization thus unavailable for duty is known as the non-effective rate, and is expressed as the average daily number non-effective per 100 or per 1,000 of the “complement” or “strength.”

DISEASE AND INJURY, UNITED STATES ARMY

(Influence of Race, Rank, and Sex, Total Officers and Enlisted Men, including Nurses, 1923; Absolute Numbers, Rates per 1,000, and Percentage Rate)

DISEASE OR INJURY	ABSOLUTE NUMBERS				RATIOS PER 1,000				PERCENTAGES		
	Admis- sions	Deaths	Dis- charges for Dis- ability	Days Lost	Admis- sions	Deaths	Dis- charges for Dis- ability	Non- effec- tive	Case Fatal- ity	Per Cent of Cases Dis- charged for Dis- ability	DAYS LOST PER CASE
Disease :											
Officers	4,384	24	24	66,286	391.74	2.14	2.14	16.23	0.55	0.55	15.12
Nurses	285	2	13	5,750	347.56	2.44	15.85	19.21	.70	4.56	20.18
White en- listed	59,730	202	1,708	987,371	564.58	1.91	16.14	25.57	.34	2.86	16.53
Colored en- listed	1,733	16	51	30,694	392.26	3.62	11.54	19.03	.92	2.94	17.71
Filipinos ...	3,026	16	143	41,866	429.22	2.27	20.28	16.27	.53	4.73	13.84
Porto Ricans	1,705	5	14	15,242	718.50	2.11	5.90	17.60	.29	.82	8.94
TOTAL ...	70,863	265	1,953	1,147,209	538.28	2.01	14.68	23.87	.37	2.76	16.19
Injury :											
Officers	692	31	11	18,973	61.84	2.77	0.98	4.64	4.48	1.59	27.42
Nurses	17	276	20.7392	16.24
White en- listed	12,326	198	124	195,272	116.51	1.87	1.17	5.06	1.61	1.01	15.84
Colored en- listed	407	6	5	5,861	92.12	1.36	1.13	3.63	1.47	1.23	14.40
Filipinos ...	471	7	7	6,997	66.81	.99	.99	2.72	1.49	1.49	14.86
Porto Ricans	200	6	2	2,716	44.53	2.53	.84	3.14	3.00	1.00	13.58
TOTAL ..	14,113	248	149	230,095	107.20	1.88	1.13	4.79	1.76	1.06	16.30

DISEASE AND INJURY, UNITED STATES ARMY

(Influence of Country, White Enlisted Men, 1923; Absolute Numbers, Ratios per 1,000 and Percentage Rates)

DISEASE OR INJURY	ABSOLUTE NUMBERS				RATIOS PER 1,000				PERCENTAGES		
	Admissions	Deaths	Discharges for Disability	Days Lost	Admissions	Deaths	Discharges for Disability	Non-effective	Case Fatality	Per Cent of Cases Discharged for Disability	DAYS LOST PER CASE
Disease:											
United States.	43,629	159	1,252	686,094	541.73	1.97	15.55	23.34	0.36	2.87	15.73
Philippine Islands.....	3,361	9	64	74,051	887.04	2.38	16.89	53.54	.27	1.90	22.03
Hawaii	6,568	14	251	136,611	528.57	1.13	20.20	30.12	.21	3.82	20.80
Panama	4,588	10	107	60,746	676.10	1.47	15.77	24.53	.22	2.33	13.24
China	774	7	15	15,335	849.62	7.68	16.47	46.12	.90	1.94	19.81
Transports and Europe	810	3	19	14,534	601.78	2.23	14.12	29.58	.37	2.35	17.94
TOTAL	59,730	202	1,708	987,371	564.58	1.91	16.14	25.57	.34	2.86	16.53
Injury:											
United States.	9,285	156	96	146,227	115.29	1.94	1.19	4.98	1.68	1.03	15.75
Philippine Islands.....	404	9	3	5,670	106.63	2.38	.79	4.10	2.23	.74	14.03
Hawaii	1,646	16	18	30,247	132.46	1.29	1.45	6.67	.97	1.09	18.38
Panama	859	12	4	9,739	126.59	1.77	.59	3.93	1.40	.47	11.34
China	80	3	1	1,113	87.82	3.29	1.10	3.35	3.75	1.25	13.91
Transports and Europe	52	2	2	2,276	38.63	1.49	1.49	4.63	3.85	3.85	43.77
TOTAL	12,326	198	124	195,272	116.51	1.87	1.17	5.06	1.61	1.01	15.84

MORTALITY STATISTICS

Mortality statistics are statistics of deaths. They are of interest primarily because of their relation to changes in population. Aside from the factor of emigration, mortality statistics show the losses in numbers being sustained by the population, just as birth records show the additions. Where migration is a factor having an appreciable effect upon population it likewise merits statistical consideration, for it, too, represents population gains and losses.

Mortality statistics have performed another important service in creating an interest in public health administration and securing support for sanitary measures. They show the extent of the loss by death caused by diseases. In the absence of morbidity records they have also frequently been used as an index of the prevalence of certain infections. It has been possible to use mortality statistics for the latter purpose on the assumption that the fatality rates of disease are fairly constant. However, we should bear in mind what News-holme has said:

"The registration of deaths gives a very imperfect view of the prevalence of disease. . . . It is fallacious to assume any fixed ratio between sickness and mortality. The fatality of a given infectious disease varies greatly in different outbreaks under varying conditions. The highest ratio of sickness is occasionally found associated with a favorable rate of mortality."

This absence of fixed fatality rates is shown by the experience in the

United States with smallpox, in which the ratio of deaths to cases has varied from 1:1,000 to 1:3; measles, in which the ratio of deaths to cases has been from 1:800 to 1:20; typhus fever, including "Brill's disease," in which it has varied from 1:5 to practically no fatality; and typhoid fever, in which the ratio has varied from 1:24 to 1:5.

Registration of Deaths in the United States.—The history of the registration of deaths in England and the United States is coupled with that of marriages and births. In the United States dependable registration was first enforced in Massachusetts and New Jersey. Other states have had laws of various types, mostly inadequate. Only recently have any number of states secured anything like complete registration. The bringing about of accurate

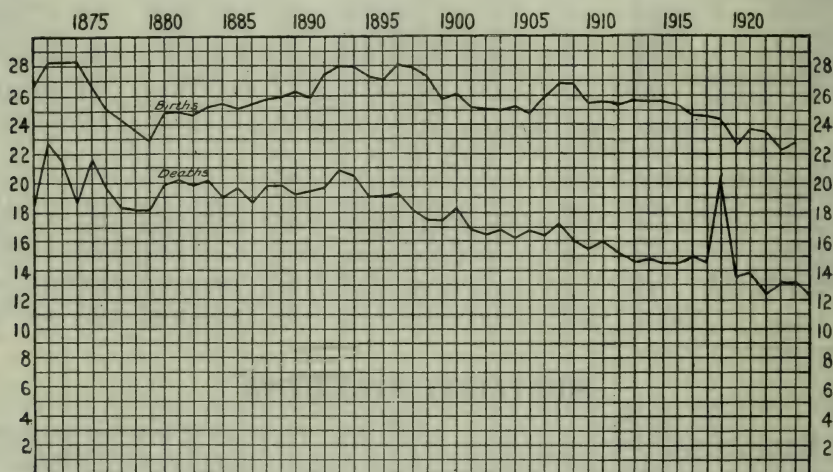


FIG. 124.—BIRTHS AND DEATHS (EXCLUSIVE OF STILLBIRTHS) PER 1,000 POPULATION PER ANNUM, REGISTERED IN MASSACHUSETTS, 1871-1924.

death registration in the United States is due largely to the efforts made by the Bureau of the Census.

United States Registration Area for Deaths.—The registration area for deaths established by the United States Bureau of the Census includes the states, and cities in other states which effectively enforce satisfactory registration laws, and, in the opinion of the Director of the Census, have at least 90 per cent of all deaths registered. This area was first established in 1880 and at that time included Massachusetts, New Jersey, and certain cities in other states. In April, 1925, the area included 39 states, the District of Columbia and 18 cities in other states, comprising in all 88.4 per cent of the total population of the United States.

Source of Data.—The original information from which mortality statistics are derived is obtained by the registration of deaths. This is commonly accomplished by the use of a blank or schedule prepared for the purpose and in this country known as a death certificate. The model law for the registration of

births and deaths provides that no body shall be interred or otherwise disposed of or removed or temporarily held pending further disposition "more than 72 hours after death unless a permit for burial, removal, or other disposition thereof shall have been properly issued by the local registrar of the registration district in which the death occurred or the body was found. And no such burial or removal permit shall be issued by any registrar until, wherever practicable, a complete and satisfactory certificate of death has been filed with him. . . ." This insures the making of a death certificate and its registration in each instance of death unless the body is surreptitiously and illegally disposed of. It therefore guarantees practically complete registration. In the rural districts of some localities bodies are frequently interred in private burial grounds and on farms in some chosen spot on the premises. Under these conditions bodies would occasionally be buried without registration, due to ignorance of the law.

The Standard Death Certificate.—The standard death certificate in use throughout the registration area for deaths calls for the following information:

Place of death.

Name, sex, color, race, conjugal condition, age, date of birth, occupation, and birthplace of decedent, name and birthplace of father, maiden name and birthplace of mother.

Signature and address of informant giving preceding information.

Date and time of death and a statement as to the duration of medical attendance on the decedent, the cause of death, and its duration, and certain other data are to be given by the physician, if any, last in attendance.

The date and intended place of burial and the address of the undertaker are to be given over the undertaker's signature.

The date when the certificate is filed is inserted by the registrar with his signature.

Copies of the standard certificate of death used in the United States may be obtained from the Superintendent of Documents, Government Printing Office, Washington, D. C.

The responsibility of seeing that a certificate is properly made out and filed with the registrar rests primarily upon the undertaker, according to the provisions of the model law.

Sources of Error.—In the use of mortality statistics as well as other statistics erroneous and unwarranted conclusions are sometimes arrived at by attempting to compare incomparable data. Mortality rates secured by lax enforcement or faulty methods of registration cannot properly be compared with those based upon complete registration. Nor can the rates of communities with populations of different sex and age composition be compared unless proper allowances are made and the rates expressed in terms of the same population. For example, it is improper to compare the mortality rate of an aggregation of young men picked for physical fitness, such as an army or navy, with the crude or general mortality rate of a civilian population. The

nearest means of making comparison would be to compare the rate of the picked body of men with the rate among men of the same age groups in the civil population. But even this would be faulty, for the one group would consist of men specially picked for physical fitness, while the other group would include the fit and the unfit, the strong and the weak. Nor is it possible to compare the mortality rate of any special population group with the rate of the population from which it has been derived by intentional or other process of selection unless the differences in population composition are considered. Thus, it would give little information of value regarding the effect of locality and environment upon the duration of life to compare the mortality rate of New York City or the registration area of the United States with that of the Canal Zone during the construction of the canal without taking into account any differences which may have been produced in the age and sex composition of the two populations by the selective process naturally operating in the case of the Canal Zone.

Another possible source of error in mortality statistics which requires to be considered is the original data contained in the death certificates from which the statistics are compiled. The personal and statistical particulars usually furnished by some member of the family are undoubtedly in most instances accurate with the exception of the statement of occupation of the decedent, which offers unusual difficulties, due to the indefiniteness of many of the terms commonly used in so far as showing the exact kind of work is concerned. This is due in some measure to the fact that the nomenclature in common use has not progressed apace with the rapid development of new industries and industrial processes and methods. Whereas fifty years ago the statement of occupation would have been in most cases comparatively simple and easily understood, to-day with changed industrial conditions the matter requires greater precision if useful statistical information is to result.

Perhaps the most common error entering into death registration, and therefore into mortality statistics, is in connection with the statement of cause of death. Aside from the fact that in the instances in which it has been impossible for the attending physician to feel reasonably certain as to the nature of the terminal illness a cause of death is nevertheless usually stated in the certificate, and also the fact that at times the physician knowing the nature of the illness may, in the belief that he is shielding the family from odium or because of their whim, intentionally state an erroneous cause of death, there still remain the many unavoidable errors of mistaken diagnosis. Just how great a factor this last may be it is difficult to estimate.

However, the findings of Richard C. Cabot² give at least a hint of its possible importance and the extent to which it may affect that part of mortality statistics relating to causes of death. In a study of 3,000 autopsies with regard to the relation of the actual cause of death as found post mortem to the clinical

² Richard C. Cabot, "Diagnostic Pitfalls Identified During a Study of 3,000 Autopsies," *J. Am. M. Ass.*, Dec. 28, 1912, p. 2295.

diagnosis Cabot found that the percentage of correct clinical diagnoses in various diseases was as follows:

	<i>Percentage of Correct Diagnoses</i>
Diabetes mellitus.....	95
Typhoid	92
Aortic regurgitation.....	84
Cancer of colon.....	74
Lobar pneumonia.....	74
Chronic glomerulonephritis.....	74
Cerebral tumor.....	72.8
Tuberculous meningitis.....	72
Gastric cancer.....	72
Mitral stenosis.....	69
Brain hemorrhage.....	67
Septic meningitis.....	64
Aortic stenosis.....	61
Phthisis, active.....	59
Miliary tuberculosis.....	52
Chronic interstitial nephritis.....	50
Thoracic aneurism.....	50
Hepatic cirrhosis.....	39
Acute endocarditis.....	39
Peptic ulcer.....	36
Suppurative nephritis.....	35
Renal tuberculosis.....	33.3
Bronchopneumonia	33
Vertebral tuberculosis.....	23
Chronic myocarditis.....	22
Hepatic abscess.....	20
Acute pericarditis.....	20
Acute nephritis.....	16

The cases studied were hospital cases under conditions assumed to be favorable to correct diagnosis. It is quite safe to assume that in medical practice at large the percentages of correct diagnoses would be found lower than those found by Cabot.

McLaughlin and Andrews³ carried on an investigation in Manila into the nature of the diseases from which children were dying. They made post-mortem examinations of children in which certain diseases had been given as the cause of death. The diseases selected were those appearing most frequently in death certificates. The reason for the investigation was to ascertain whether the death certificates showed the real causes of death in children in Manila and if not what the actual causes of death were.

³ Allan J. McLaughlin and Vernon L. Andrews, "Studies on Infant Mortality," *Philippine J. Sc.*, July, 1910, 5, No. 2, p. 149.

A summary of their findings was as follows:

<i>Assigned causes of death</i>		<i>Causes of death ascertained by autopsy</i>	
Meningitis	37	Cholera	40
Enteritis	22	Beriberi	97
Convulsions	40	Pneumonia	14
Beriberi	50	Enterocolitis	7
Bronchitis	27	Meningitis	4
		Nephritis	2
		Empyema	2
		Acute tonsillitis, pharyngitis, and bronchitis	1
		Cerebral hemorrhage.....	1
		Undetermined	8
<hr/> Total.....		176	Total.....
			176

In the registration area of the United States very probably the causes given in death certificates of children correspond more nearly to the actual causes of death than they did in Manila. This, however, should be ascertained by careful studies. Mortality statistics cannot be more accurate than the death certificates from which they are compiled.

For a further discussion of the possible scope of the inaccuracies entering into mortality statistics because of the faulty or incorrect statement of cause of death on death certificates the reader is referred to the Twelfth Annual Report of the Bureau of the Census giving mortality statistics for the year 1911, pages 36 to 38.

Uses of Death Registration.—Death registration serves a number of highly important purposes. Its functions are legal, economic, and social. Death registration is useful in preventing and detecting crime through the restrictions placed upon the disposal of dead bodies. It serves as evidence in the inheritance of property and in the settlement of life insurance contracts and policies. It is only proper that the time, place, and cause of death of each individual should be made a permanent record for both sentimental and legal reasons.

Death registration makes it possible to show by mathematical computations and statistical methods the extent and rate of change in population produced by deaths; the average duration of life; and, to the extent that the certified causes of death have been correctly stated, the relative frequency with which the several causes produce death. Death statistics by comparison with birth statistics give useful information regarding population increase or decrease.

Death Rates.—Death rates may be expressed as the ratio of the total number of deaths, taken as a unit, to the population. For example: 1 in 60. The usual method, however, is to express these rates in terms of the number of deaths per 1,000 population, or in some instances per 10,000 or even 100,000, or 1,000,000.

Crude Death Rates.—The rate which shows the proportion of all deaths to the total population, and which is usually obtained by dividing the total number of deaths by the total population in thousands, is known as the crude death rate; also as the general or central death rate. To compute the crude death rate the total number of deaths during a year and the mean population for the year (estimated population as of the middle of the year, for the calendar year as of July 1) are taken. To illustrate: In a city having a total of 900 deaths during a calendar year, and an estimated population of 60,000 as of July 1 of the year, the crude death rate would be $900 \div \frac{60,000}{1,000} = 15$ and would be expressed as 15 per 1,000 population.

Crude death rates are of value chiefly to show the numerical loss of the population by death. They also serve as a satisfactory basis for the comparison of the death rates of different communities having populations of similar composition as to age and sex. For populations of dissimilar composition they are not suitable as a basis of comparison, for the death rates of women are usually lower than those of men and the death rates of the several age groups vary within wide limits; and the death rate, therefore, depends to a marked degree upon the relative numbers of males and females and the proportion of the population included in the various age groups.

Death Rates for Short Periods.—Death rates for short periods (for a week, month, or quarter) are expressed in terms of annual rates; that is, what the annual rate would be provided deaths occurred throughout the year with the same frequency as during the week or month under consideration. Death rates for short periods are likely to have little significance, as quite accidental causes may affect them to a considerable degree. Taken for a number of years, however, they give useful information regarding seasonal variations. If in a city there were 20 deaths during a given week and the mean population of the city for the year was 60,000, then the crude death rate for the week would be

$$20 \times \frac{365}{7} \left(\frac{\text{days in year}}{\text{days in week}} \right) \div \frac{60,000}{1,000} (\text{population of city in thousands}) = 17.38.$$

The mortality for the week would, therefore, be at the rate of 17.38 per 1,000 population per annum.

Specific Death Rates.—Special or specific death rates are the rates of specified or limited subgroups of the population. These subgroups may be obtained by dividing the population according to sex, age, race, social condition, occupation, and so on. Specific death rates may be stated as the proportion of the number of deaths per annum in the subgroup per 1,000 of the mean annual number of the population in that subgroup. Sometimes specific death rates are given in terms of 10,000, 100,000, or 1,000,000 of the subgroup population.

Among the most important of the specific rates are those relating to age groups. Their significance is shown by the following statement of rates for the registration states of the United States for the year 1911:

<i>Age Group</i>	<i>Death Rate per 1,000</i>
Under 1 year.....	112.9
1 to 4 years.....	11.8
5 to 9 years.....	3.1
10 to 14 years.....	2.2
15 to 19 years.....	3.6
20 to 24 years.....	5.2
25 to 34 years.....	6.4
35 to 44 years.....	8.9
45 to 54 years.....	13.6
55 to 64 years.....	26.2
65 to 74 years.....	55.2
75 years and over.....	138.9
All ages.....	13.9

Specific race group rates are also important. In the registration area for deaths in 1911, the death rate for the white population was 13.7 and that of the colored 23.7 per 1,000, while the rate of the two groups taken together was 14.2 per 1,000. In 1916 the death rates in the city of Washington were 15 per 1,000 among the white population and 25.4 per 1,000 among the colored; in Baltimore 15.9 for white and 30.7 for colored; in New Orleans 14.3 for white and 30 for colored. For the registration area as a whole the death rates in 1916 were for the white population 13.5 per 1,000 and for the colored 20.5 per 1,000.

In considering the separation of deaths into those of white and colored, one must bear in mind the possibility that in many communities such a separation may amount to a classification according to industrial or economic status, the colored deaths being those in households having the smaller incomes. In this connection one is reminded that investigations into the rate of infant mortality and the relative prevalence of certain diseases, such as tuberculosis, have revealed that the infant mortality rate varied usually with the incomes of the population groups and that the relative prevalence of tuberculosis seemed to be largely determined by the same factor. It may be that if in the average community deaths could be classified according to economic status—that is, according to the family or household income, a difference in the mortality rates would be obtained approximately as great as that resulting from a white and colored classification.

That a classification of deaths on a basis other than that of color may show differences in mortality rates fully as great as those produced by a white and colored classification is well known.

In New York City (the old city) for the year 1906 the death rate among that part of the population of Irish nativity was 29.26 per 1,000, and among the population of Swedish nativity 11.21 per 1,000, while the death rate of native-born Americans was 18.49. Classified according to the nativity of the parents of the decedents, the death rate among persons whose parents were

born in Italy was 36.43 per 1,000; of those whose parents were born in Austria-Hungary, 23.4 per 1,000; and of those whose parents were born in Sweden, 10.97 per 1,000; while of those whose parents were born in the United States the rate was 13.98 per 1,000. The death rate of the colored population throughout the whole city (Greater New York) was 27.16. These figures indicate that, in so far as crude death rates are concerned, other groups of the population may have, and in New York City did have, higher rates than the colored population.

The death rate differs also in the two sexes. It is higher for males than for females.

Standardized Death Rates.—Due to the wide variation in the death rates at different ages it is impossible satisfactorily to compare the crude death rates of populations differing in composition as regards the relative number of individuals in the several age groups. The International Statistical Institute recommended (1895) that to facilitate the comparison of death rates the population of Sweden as it existed in 1890 be used as a standard population for the statement of rates. Rates expressed in terms of standard population are known as standardized or corrected rates. The method is as follows: Take the population for which it is desired to state the standardized death rate and ascertain the specific death rates of its several age groups. Now take the corresponding age groups in 1,000,000 of the standard population and compute the number of deaths that would have occurred in each age group at the specific death rate found to exist in the population for which the standard death rate is being computed; add the number of deaths which it is thus found would have occurred in the age groups of the standard population. This gives the standardized rate per 1,000,000. The standardized rate per 1,000 is obtained by moving the decimal point three places to the left.

The standardized death rate is the rate which would have occurred in the standard population if the death rates in its several age groups had been the same as those of the corresponding age groups of the population under consideration.

The registrar general of births, marriages, and deaths of England and Wales has for some years taken for a standard the population composition of England and Wales as shown by the 1901 census. The population of Sweden of 1890 was divided without distinction of sex into the five age groups: Under 12 months of age, over 12 months and under 20 years, 20 to 39 years of age inclusive, 40 to 59 years of age inclusive, and 60 years of age and over. The population of England and Wales is classified separately by sexes in quinquennial age groups and furnishes a much more delicate and exact standard for measurement. The use of the Swedish population standardizes for age; the use of the English standardizes for both age and sex.

Factors Affecting Death Rates.—Death rates are affected not only by the statistical methods used in their preparation and by the age, sex, and race composition of the population, the social, marital, and economic status of the

people, the nature and conditions of employment and the adaptability of a people to their environment, but also in limited areas by a number of other factors, such as the location of hospitals and institutions.

Nonresidents; Hospitals and Institutions.—Frequently a hospital or other institution will be located in one community while its patients or inmates will come largely from other places. The extent to which this is true depends upon the nature or reputation of the hospital or institution. The result may be that the local death rate will be affected to an appreciable extent by deaths of nonresidents in such institutions. In England and Wales an attempt has been made during recent years to overcome this difficulty by the allocation of all deaths in so far as possible to the locality of usual residence. In compiling deaths for a registration district or area for the purpose of showing death rates, erroneous results will be obtained if the deaths of nonresidents are excluded and no additions made for the deaths of residents which are continually occurring and being registered elsewhere.

In the absence of a dependable means of including the deaths of residents occurring in other districts it is, unless under most exceptional circumstances, unsafe to exclude the deaths of nonresidents.

For the public health purposes of mortality statistics nonresident deaths might be considered as those of persons who had been already affected with their fatal illnesses at the time they had come to the locality and who had not developed or contracted the illnesses in the locality.

Migration.—Migration affects death rates by changing the age, sex, or race composition of the population. Migrants are likely to consist more largely of males than of females, of young adults than of the extremes of life. The effect of migration depends upon whether the balance is one of emigration or immigration and the nature of the migrants lost or gained.

The problem of registration of non-resident deaths is perplexing; it requires standard methods and coöperation between registrars.

Birth Rate.—Ignoring the question of migration, a population increases because of the excess of births over deaths, natural increase. In a stationary population the birth rate equals the death rate. As all born must eventually die the birth rate depends for its excess over the death rate upon the ever-increasing number of child-producing elements in the population and the resulting greater numbers in the younger age groups. Other things being equal, a community with a high birth rate will, because of the greater proportion of the population in the younger age groups, have a lower crude death rate than a community with a low birth rate.

Marital Condition.—Mortality in certain countries seems to be more dependent on marital conditions than on sex. This is shown by the following table taken from a paper entitled "Some Researches Concerning the Factors of Mortality," by Lucien March (*Journal of the Royal Statistical Society*, London, March, 1912):

SHOWING FOR THE PERIOD 1886-1895, THE NUMBER OF DEATHS PER 10,000 PERSONS
ACCORDING TO THEIR MARITAL STATUS IN FRANCE, PRUSSIA, AND SWEDEN

MARITAL STATUS	MALES, AGE			FEMALES, AGE		
	20-39	40-59	60 and Over	20-39	40-59	60 and Over
France:						
Married	77	153	583	80	121	456
Single	103	246	794	78	166	730
Widowed or divorced.....	211	293	1,148	145	198	930
Prussia:						
Married	71	175	582	79	128	497
Single	84	231	806	59	179	729
Widowed or divorced.....	201	346	1,091	101	172	805
Sweden:						
Married	53	114	453	66	96	364
Single	83	204	690	61	120	528
Widowed or divorced.....	104	190	856	98	132	698

It will be noted in the following table that there has been a marked fall in the crude death rates throughout the civilized world. A study of the deaths by age groups indicates that in the United States the reduction in death rates has been entirely in the younger age groups and that the rates for ages above forty-five or fifty years have not only not diminished, but have actually increased. This means that a greater proportion of the population than formerly is living to be fifty years of age. The increased death rates among that part of the population over fifty probably means that those saved from earlier death and carried along to the age of fifty succumb rather rapidly to the vicissitudes of life. It would seem that this is to be expected.

DEATH RATES (EXCLUSIVE OF STILLBIRTHS) PER 1,000 POPULATION IN CERTAIN COUNTRIES, 1886, 1913, 1914, 1915 AND 1920.

Country or State	1886	1913	1914	1915	1920
Australian Commonwealth	15.4	10.8	10.5	10.7	10.5
Austria	29.7	20.5 †	21.3
Denmark	18.1	12.5	12.6	12.8	12.9
England and Wales	19.5	13.8	14.0	15.7	12.4
Finland	22.2	16.1	15.6	15.9
France	22.5	17.7	19.6	19.1	17.7
Germany	26.2	15.0 †	21.4	15.1
Hungary	31.7	23.3 †
Ireland	17.8	17.1	16.3	17.6	14.8
Italy	28.7	18.7 †	17.9	20.4
The Netherlands	21.8	12.3	12.4	12.4	12.0
New Zealand	10.5	9.5	9.3	9.1	10.2
Norway	16.2	13.2	13.5	13.3
Roumania	26.7	25.9	23.8
Scotland	18.9	15.5	15.5	17.1	14.0
Servia	29.6	21.1 †
Spain	29.3	22.1	22.1	22.0	23.2
Sweden	16.6 *	13.6	13.8	14.7	13.3
United States (registration area for deaths)	19.8 †	14.1	13.6	13.6	13.1
Connecticut	16.2	15.0	15.1	14.9	13.5
Massachusetts	18.6	15.0	14.7	14.5	13.8
Michigan	8.9	13.9	13.4	13.4	13.8

* Year 1880.

† Year 1912.

The International List of Causes of Death.—Many persons, even among registrars and statisticians, seem to misunderstand the nature of the International List of Causes of Death. It seems frequently to be thought of as being a nomenclature of diseases. It is important that just what it is shall be thoroughly understood.

In the course of the registration of deaths in a population of any size, the names given to the causes of death as written on the death certificates will aggregate in the total many thousands, depending upon the education and training of the physicians signing the death certificates. Several different terms will be used by different physicians for the same disease, or the same name may be used for several different morbid conditions. For instance, typhoid fever will be variously termed abdominal fever, cerebral typhoid, continued fever, enteric fever, gastro-enteric fever, typho-enteritis. Malarial fever will be variously recorded as bilious intermittent fever, chills and fever, dumb ague, fever and ague, gastric remittent fever, intermittent fever, marsh anemia and sometimes as malaria. On the other hand "continued fever," "rheumatism," "pneumonia," "cancer" and "heart disease" are each used as the name of many different pathologic conditions and it is impossible to know what is meant by these terms unless the user gives his definition of their meaning as used by him.

And so it is with all the other diseases and conditions causing death. It will be readily understood that it is impossible for the statistician to give the occurrence of death for each of the multitudinous causes which would thus be found recorded in death certificates. Statistically, it would be an impossible task and from the standpoint of printing and publication and the futility when published, meaningless and of no value. It is therefore necessary for the statistician to take the death certificates and divide them into a practicable number of groups, under which they can be compiled, and for which tables can be published. Every statistical office has done this. The only alternative would have been to give practically a serial statement of deaths and their assigned causes. But where each statistical office determined for itself the groups of causes of death which it would associate together in one table, or, in other words, the groupings under which it would publish its causes of death, no two statistical groupings were the same, and therefore, no two sets of tables were comparable. If the French statistical office made one grouping and published their tables according to their grouping and the English statistical office made another grouping and published their tables according to that grouping, comparative deductions as relating to the mortality data of the French and English peoples would be impossible.

To overcome this difficulty and make mortality statistics of all countries comparable, it was determined about 1893 to decide upon a statistical grouping of causes of death for purposes of tabulation and publication which would be used by the several countries publishing mortality data. As a result of this we have the International List of Causes of Death. After the revision in 1920, the International List contained 205 titles or groupings under which all re-

turns of death may be tabulated. Each title may figuratively be considered as a basket and the 205 titles as the 205 baskets into one of which the statistician drops each death certificate coming to his office. From the assortment of death certificates thus made statistical tables are published. About half of the 205 numbers or titles of the list include but one disease; the others include groups of diseases or death-producing causes.

It will thus be seen that the International List is by no means a nomenclature, nor is it even a classification, unless it be considered a classification for purposes of statistical publication.

Each people speaking a different language has had to determine for itself which names given on death certificates as causes of death in their respective languages should be included under each of the 205 titles in the International List. The only part of the International List which can be international is the 205 numbers and the titles in so far as the titles are descriptive. To make a list entirely possible of international use, each title should have a number and a definition of the disease or of the class of diseases to be included under that number. For instance, Title 17, "Plague" would be truly capable of international use only when the definition of what is meant by "Plague" is given.

INFANT MORTALITY

Infant mortality is the mortality of infants under one year of age. While the specific death rates for other age groups are given as the ratio of the number of deaths to the number of individuals in the age group as ascertained by census enumeration and estimated for intercensal and postcensal years, it is not practicable to do this for the first year of life. There is extreme difficulty in ascertaining by enumeration the infant population. This is due largely to confusion of the current year of age with the completed year of life. Then, too, a census of infants under one year of age would be of value only for the year in which it was taken.

The commonly accepted method of stating infant mortality is as the ratio of deaths of children under one year of age to living births, and is usually expressed as the proportion of deaths during the calendar year to 1,000 living births during the same period. To illustrate: If in a city there were during a year 224 deaths of infants under one year of age, and if during the same year there were 2,000 births, the infant mortality rate would be 112 per 1,000 births per annum.

Infant mortality rates might be based upon the number of births during the preceding year or upon the mean of the number of births of the current year and the preceding year. However, the number of births of the current year has been accepted as the most satisfactory basis in Great Britain and many other countries.

Making the estimation of infant mortality depend upon birth registration is unfortunate in a way. However, there seems to be no other practicable basis for estimation. In the absence of change in other factors an improving com-

pleteness of birth registration would give an apparent decreasing infant mortality rate and might lead to unwarranted deductions. See also page 504.

LIFE TABLES

In theory life tables represent the duration of life of individuals born at the same time. Given a group of individuals born in any one year and a life table will show the number in the group that will still be alive in each succeeding year as long as any remain. It will also show the number who will have died previous to any given year and the number dying during each year. To observe a group of individuals from the cradle to the grave is under most

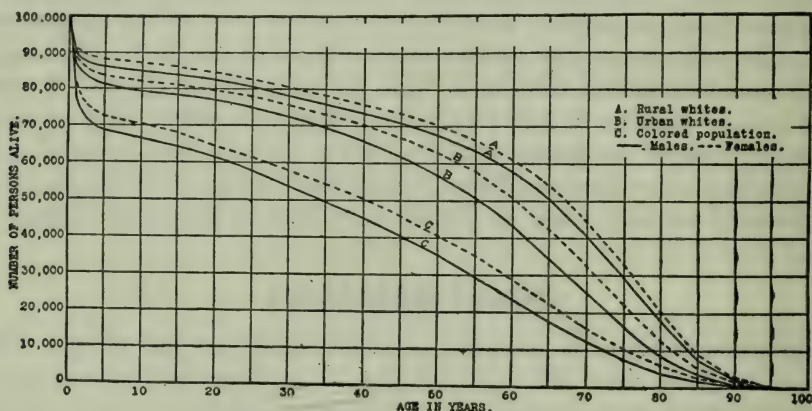


FIG. 125.—CHART SHOWING THE NUMBER OF WHITE MALES AND WHITE FEMALES IN BOTH THE RURAL AND URBAN POPULATIONS, AND OF COLORED (NEGRO) MALES AND FEMALES IN THE TOTAL POPULATION, REMAINING ALIVE AT EACH AGE OUT OF 100,000 BORN ALIVE IN THE "ORIGINAL REGISTRATION STATES."

(Based upon the "United States life tables: 1910," published by the Bureau of the Census. Taken from *Pub. Health Rep.*, April 6, 1917.)

conditions impracticable, and besides yields information the value of which is largely lost before it is obtained, for conditions affecting longevity may change and the life history of one generation may be quite different from that of the next.

Much of the value of a life table consists in showing current conditions as they affect the longevity of the community or race. For this purpose tables are constructed from the information furnished by an enumeration of the population (census) classified by age and sex and the registration of deaths with the decedents classified also by age and sex. The population age and sex groups give the number and proportion remaining alive at each year of age, the deaths show the number dying at each year of age. For the purpose of getting data which show general conditions prevailing during the period, and of avoiding the errors which might arise by using the death records of a year during which unusual mortality conditions prevailed, the death records for a number of consecutive years are usually used.

Given the above data, the expectancy of life or mean after lifetime at a given age is readily obtained. The following table is one prepared under the direction of William H. Guilfoy, registrar of records of the New York City department of health, and published in the monthly bulletin of the department for May, 1913. It compares the expectation of life based on the mortality experience of the three years 1909, 1910, and 1911, with that found by the late John S. Billings based upon the experience of 1879, 1889, and 1881:

APPROXIMATE LIFE TABLES FOR THE CITY OF NEW YORK BASED ON MORTALITY RETURNS FOR THE TRIENNIALS 1879 TO 1881 AND 1909 TO 1911. (*Guilfoy.*)

YEARS OF MOR- TALITY	EXPECTATION OF LIFE, 1879 TO 1881			EXPECTATION OF LIFE, 1909 TO 1911			GAIN (+) OR LOSS (—) IN YEARS OF EXPECTANCY		
	Males	Females	Persons	Males	Females	Persons	Males	Females	Persons
Ages:									
— 5...	39.7	42.8	41.3	50.1	53.8	51.9	+10.4	+11.0	+10.6
5...	44.9	47.7	46.3	49.4	52.9	51.1	+ 4.5	+ 5.2	+ 4.8
10...	42.4	45.3	43.8	45.2	48.7	46.9	+ 2.8	+ 3.4	+ 3.1
15...	38.2	41.2	39.7	40.8	44.2	42.5	+ 2.6	+ 3.0	+ 2.8
20...	34.4	37.3	35.8	36.6	40.0	38.3	+ 2.2	+ 2.7	+ 2.5
25...	31.2	34.0	32.6	32.7	36.0	34.3	+ 1.5	+ 2.0	+ 1.7
30...	28.2	31.0	29.6	28.9	32.1	30.5	+ 0.7	+ 1.1	+ 0.9
35...	25.3	28.1	26.7	25.4	28.4	26.9	+ 0.1	+ 0.3	+ 0.2
40...	22.5	25.2	23.9	22.1	24.7	23.4	— 0.4	— 0.5	— 0.5
45...	19.8	22.4	21.1	18.9	21.1	20.0	— 0.9	— 1.1	— 1.1
50...	17.2	19.4	18.3	15.9	17.7	16.8	— 1.3	— 1.7	— 1.5
55...	14.5	16.4	15.4	13.2	14.6	13.9	— 1.3	— 1.8	— 1.5
60...	12.2	13.8	13.0	10.8	11.8	11.3	— 1.4	— 2.0	— 1.7
65...	9.9	11.2	10.5	8.8	9.4	9.1	— 1.1	— 1.8	— 1.4
70...	8.5	9.3	8.9	6.9	7.5	7.2	— 1.6	— 1.8	— 1.7
75...	7.1	7.5	7.3	5.3	5.7	5.5	— 1.8	— 1.8	— 1.8
80...	6.2	6.5	6.4	4.1	4.5	4.3	— 2.1	— 2.0	— 2.1
+85...	5.4	5.5	5.5	2.0	2.4	2.2	— 3.4	— 3.1	— 3.3
Balance	{ +24.8 —15.3 + 9.5	{ +28.7 —17.6 +11.1	{ +26.6 —16.6 +10.0

The Bureau of the Census issued in June, 1916, life tables prepared under the supervision of Professor James W. Glover. The tables relate chiefly to mortality conditions in the area known as the original registration states comprising Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Indiana, Michigan, and the District of Columbia. The chart on page 1174 is based on the material in these tables.

REFERENCES

- FARR, WILLIAM. *Vital Statistics*, 1885.
- NEWSHOLME, ARTHUR, M.D., F.R.C.P. *The Elements of Vital Statistics*, 1899.
- BAILEY, WILLIAM B., Assistant Professor of Political Economy, Yale University. *Modern Social Conditions—A Statistical Study of Birth, Death, Marriage, Divorce, Disease, Suicide, Immigration, etc., with Special Reference to the United States*, 1906.
- BOWLEY, ARTHUR L., M.A., F.S.S., Lecturer in Statistics at the London School of Economics and Political Science. *Elements of Statistics*, 1907.

YULE, G. UDNY. *An Introduction to the Theory of Statistics*, 1912.

KING, WILLFORD I., M.A., Instructor in Statistics in the University of Wisconsin.
The Elements of Statistical Method, 1915.

Annual Reports and Supplements of the Registrar-General of Births, Deaths, and Marriages in England and Wales.

Annual Reports on Mortality Statistics of the Registration Area for Deaths of the United States by the Bureau of the Census.

Bureau municipal de statistique d'Amsterdam, "Statistique démographique des grandes villes du monde, 1880-1909," 1911.

Statistique générale de la France, "Statistique internationale du mouvement de la population d'après les registres d'état civil-résumé rétrospectif depuis l'origine des statistiques de l'état civil, jusqu'en 1905," 1907.

WHIPPLE, GEORGE C. *Vital Statistics*. John Wiley & Sons, 1919.

SECTION XIII

STATISTICAL METHODS

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Introduction.—The development of theoretical statistics which has taken place during the last thirty to forty years has led to the publication of numerous methods, but their application has followed slowly. Before a theoretical method can be proven useful it must be applied a number of times to data for which it was intended and its applicability appraised. This is frequently difficult in regard to some methods on account of the paucity and unreliability of the data. There is need of more and better data, and, for the novice, less theoretical methods; a frequent application of the more simple methods will solve most of his problems and is necessary to lead him to a point where he may learn safely to apply more complex methods when the data justify their use and when the simple methods are insufficient. In other words, most problems in vital statistics can be solved by arithmetic, for the data seldom justify the application of any method involving complicated mathematics.

There is danger of falling into an attitude of security and confidence which inhibits thought, when applying mathematical formulæ. A careful consideration and study of figures frequently leads to sounder conclusions than does the application of mathematical methods. A formula always gives a mathematically accurate answer, but in a problem that answer may nevertheless be entirely wrong. From the beginning to the end of a statistical problem it is necessary to consider and to reconsider the figures and to maintain throughout a healthy degree of suspicion with regard to any inference obtained. The sources of the figures should first be appraised: how obtained; errors of measurement; bias in selection. The whole field from which they were taken should be studied as carefully as possible. The data should be arranged and rearranged in many ways so as to see them from different standpoints. They should be plotted. With graphic methods an additional sense is acquired somewhat similar to the use of a cardiograph, which adds information that is not perceived by palpation and auscultation. Much real statistical analysis can be done before any calculation from the data is made.

It would pay the beginner to read Westergaard, *J. Am. Stat. Ass.*, 1916-17, 15:255. Textbooks are becoming increasingly plentiful. For the beginner Falk, Whipple, and Newsholme are recommended, and after them, Yule.

TABLE I

DEATH RATES FROM CANCER IN THIRTY-FOUR STATES OF THE UNITED STATES
REGISTRATION AREA IN 1920

State	Death Rate (y)	Deviation from Median (y-M)		Deviation from Mean (y-m)		(y-m) ²
		+	-	+	-	
Massachusetts	97.9	20.7		23.7		562.0
Connecticut	92.8	15.6		18.6		346.0
New York	92.3	15.1		18.1		327.6
Minnesota	92.1	14.9		17.9		320.4
Rhode Island	88.5	11.3		14.3		204.5
Illinois	87.4	10.2		13.2		174.2
California	87.1	9.9		12.9		166.4
New Jersey	86.9	9.7		12.7		161.3
Maryland	86.7	9.5		12.5		156.3
Vermont	85.7	8.5		11.5		132.3
Maine	85.1	7.9		10.9		118.8
Pennsylvania	82.6	5.4		8.4		70.6
Oregon	79.3	2.1		5.1		26.0
New Hampshire	78.9	1.7		4.7		22.1
Michigan	78.1	0.9		3.9		15.2
Wisconsin	78.1	0.9		3.9		15.2
Ohio	77.7	0.5		3.5		12.2
Washington	76.8		0.4	2.6		6.8
Delaware	75.1		2.1	0.9		0.8
Nebraska	74.3		2.9	0.1		0.0
Indiana	72.7		4.5		1.5	2.2
Colorado	71.1		6.1		3.1	9.6
Utah	67.7		9.5		6.5	42.3
Missouri	67.5		9.7		6.7	44.9
Kansas	63.4		13.8		10.8	116.6
Louisiana	62.3		14.9		11.9	141.6
Montana	61.9		15.3		12.3	151.3
Virginia	61.9		15.3		12.3	151.3
Kentucky	57.7		19.5		16.5	272.3
Florida	56.9		20.3		17.3	299.3
Tennessee	51.6		25.6		22.6	511.0
North Carolina	49.7		27.5		24.5	600.0
Mississippi	48.0		29.2		26.2	686.0
South Carolina	45.8		31.4		28.4	807.0
Sum		144.8—248.0		199.4—200.6		
	2521.6	392.8—103.2		— 1.2		6674.1
Sum 34	74.16	11.55—3.04		— .04		196.3

$$M = 77.25 = 77.2$$

$$m = 74.16 = 74.2$$

$$\sigma = \pm \sqrt{196.3} = \pm 14.0$$

$$A. D. = 11.55 = 11.6$$

Statistical Series.—A statistical series is a set of data. It is the enumeration of any thing or things, with respect to any quantitative or qualitative attribute. Table I illustrates a statistical series. It consists of the enumeration of 34 states in the United States registration area with respect to the

quantitative mortality from cancer. The values for mortality are adjusted so that they represent the mortality that would exist in these states if each were populated by the same number of people of the same age and sex distribution, *i.e.*, these values are rates adjusted for age and sex. Figure 126 gives the graphic representation of the data of Table 1. The rates vary from 45.8 to 97.9. They crowd together between 70.0 and 90.0. Over half of them occur in this range. It is desirable to get some measure of the average or centering point of these rates. It is also desirable to obtain some measure of the extent to which they vary from the average or centering point.

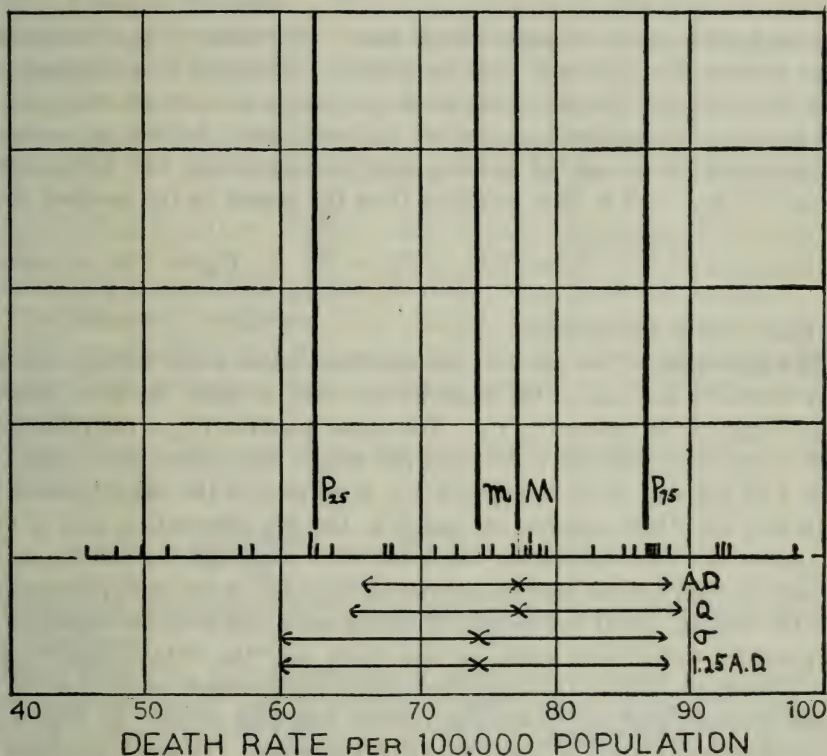


FIG. 126.—DISTRIBUTION OF DEATH RATES FROM CANCER.

Statistical Constants.—These are values computed from the data by which the series is described. The more important of these are the centering constants which measure the central tendency or the average of the series, and the dispersion constants which measure the amount of scattering from the centering constants.

Centering Constants.—The centering constants comprise the mean and the median. The mean, unless otherwise specified, is the arithmetic mean, and is the average of the values. It is the sum of their values divided by the total number of them. It is a purely theoretical value and is not the value of any particular observation. It describes the group with respect to its centering

tendency. The sum of the values of Table I is 2521.6 which when divided by 34 = $\frac{2521.6}{34} = 74.16 = 74.2 = m$, the mean. In Figure 126 the mean is the vertical line marked *m*.

The median is the value below and above which 50 per cent of the observations lie. Similar to the mean it is a theoretical value and is descriptive of the group. In Table I, $\frac{1}{2}$ of the observations is $\frac{34}{2} = 17$. The median is the value

below and above which 17 observations occur. Obviously it is a value somewhere between the 17th and 18th observation. Although it is customary to select the mid-point between them, yet in fact, any point between them can be used and thus the median is somewhat indeterminate. In fact no statistical constant should be thought of as being exactly determined. The 17th observation is 77.7 and 18th is 76.8, counting from the largest to the smallest value.

The median is $\frac{77.7 + 76.8}{2} = 77.25 = 77.2 = M$. In Figure 126 the vertical line marked *M* is the median.

The Quartiles.—The value of the attribute below which 25 per cent and above which 75 per cent of the observations occur is called the lower quartile or the 25 percentile denoted by P_{25} . The upper quartile, P_{75} , is the value below which 75 per cent and above which 25 per cent of the observations occur. In Table I 25 per cent of 34 = $8\frac{1}{2}$ and P_{25} is selected as the value between the 8 $\frac{1}{2}$ th and the 9 $\frac{1}{2}$ th observation which is the 9th observation and is 62.3. P_{75} falls at the 26th observation which is 86.7. Although P_{25} and P_{75} fall at determined observations, they should not be regarded as any better determined than the median, for if the sample of cancer rates had been 36 instead of 34 the quartiles would have fallen at the 9 $\frac{1}{2}$ th and the 29 $\frac{1}{2}$ th observations. The indetermination of the statistical constants decreases as the size of the sample increases and will be discussed under sampling errors. In Figure 127 the vertical lines marked P_{25} and P_{75} are the quartiles. In many problems the subject of chief interest is the study of variation.

Constants of Dispersion.—Centering constants do not fully describe the statistical series. There is always scattering from the average; every observation does not coincide in value with the value of the average. It is very desirable to have some measure of the scattering from the average. Measures of dispersion can be divided into two groups, those that measure the scattering about the median and those that measure the scattering about the mean. The dispersion constants about the median are the quartile deviation denoted by *Q* and the average or mean deviation denoted by A.D. *Q* is independent of the magnitude of the deviations and A.D. is entirely dependent upon their magnitudes. The dispersion constant about the mean is the standard deviation denoted by σ .

Quartile Deviation.—The quartile deviation is one-half the distance between the quartiles. In Table I $P_{25} = 62.3$, $P_{75} = 86.7$. The quartile deviation denoted by $Q = \frac{86.7 - 62.3}{2} = 12.2$; 50 per cent of the observations occur

over the range of $P_{75} - P_{25}$. If the series is symmetrical with respect to the mean or median, 25 per cent of observations fall between the limits of M and $M \pm Q$. In Figure 126 Q is represented as a horizontal line drawn in both directions from the median.

The Average Deviation.—The sum of the deviations from the median, if made without regard for sign (that is, if the plus and minus deviations are regarded as similar or plus deviations), divided by the total number of observations, gives the value of the average deviation denoted by A.D. It is the least when measured from the median. In Table I the deviations from the median designated as $y - M$ are summed, disregarding the sign of the deviation, and divided by 34 and $A.D. = \frac{392.8}{34} = 11.55 = 11.6$. In Figure 126 A.D. is represented by a horizontal line drawn above and below from the median.

The Standard Deviation.—The standard deviation or sigma, σ , is the measure of dispersion about the mean. It is the square root of the sum of the squares of the deviations from the mean divided by the total number of observations.

From Table I the sum of the squares of the deviations from the mean designated as $(y - m)^2$ is

$$\sigma = \pm \sqrt{\frac{6674.1}{34}} = \pm \sqrt{196.3} = \pm 14.01 = \pm 14.0$$

In Figure 127 σ is represented by a horizontal line drawn from the mean in both directions.

Short Cuts.—In the computation of the above constants it is possible to avoid some arithmetic and to eliminate some chance of error by using an arbitrary mean. The arbitrary mean can be any desired number which is subtracted from the values, leaving deviations smaller than the original values. The deviations are then summed and divided by N . The arbitrary mean usually selected is a round number near the mean. If an arbitrary mean is denoted by M_o , then the formula for the computation of the mean becomes $m = M_o + \frac{\Sigma(x - M_o)}{N}$. There will be a series of plus deviations and one of minus deviations. Upon addition these largely cancel one another and the $\frac{\Sigma(x - M_o)}{N}$ denoted by b is usually a small number.

The $\Sigma \frac{x - M_o}{N}$ without regard for sign is the average deviation (approximately).

When computed from an arbitrary origin

$$\sigma = \sqrt{\frac{\sum [(x - M_0) - b]^2}{N}} = \sqrt{\frac{\sum (x - M_0)^2 - b^2}{N}} = \sqrt{\frac{\sum x^2 - m^2}{N}}$$

since $b = m - M_0$.

TABLE II

DEATH RATES FROM CANCER IN THIRTY-FOUR STATES OF THE UNITED STATES
REGISTRATION AREA IN 1920

Death-Rate = (x)	Frequencies	(x-M ₀)	$\frac{(x-M_0)}{10}$ = (x')	(x') Frequencies	(x') ² Frequencies
Under 50	3	- 30	- 3	- 9	27
50-59	3	- 20	- 2	- 6	12
60-69	6	- 10	- 1	- 6	6
70-79	10	0	0	0	0
80-89	8	+ 10	1	8	8
90 and over	4	+ 20	2	8	16
Sum	34 = N			-21 + 16 - 5	69
$\frac{\text{Sum}}{N}$				- .147	2.03

$M_0 = 75$
 $m_x' = -.147$
 $m_x = M_0 + 10m_x' = 75.0 - 1.5 = 73.5$
 $\sigma_x' = \pm \sqrt{2.03 - (-.147)^2} = \pm \sqrt{2.008} = \pm 1.42$
 $\sigma_x = 10\sigma_x' = \pm 14.2$
 $M = 75$

The data of Table I are grouped into classes and shown in Table II. Grouping is desirable when dealing with a large number of observations as it reduces greatly the amount of arithmetic necessary for the determination of the statistical constants and, as a rule, involves no great loss in accuracy. Many published statistical tables are so grouped. The data of Table II are a frequency distribution of 34 states with respect to mortality from cancer.

In Figure 127 is shown the graphic representation of the data of Table II. It is an histogram. By inspection it is evident that the most common or typical cancer rate is somewhere between 70 and 80. The most typical value is called the mode. It can be roughly estimated from the histogram by inspection. The determination of the statistical constants from grouped data differs in slight details from that from the ungrouped data.

When the frequencies or observations are grouped in classes as in Table II the evaluation of the median is made by a slightly different method. To find the point less than which $\frac{N}{2}$ observations fall, the frequencies can be arranged according to the number that lies below a given class. Most frequently the median will fall in a given class. The frequencies of the median class are

divided into two parts, those that lie above the median and those that lie below. The ratio of these parts to the frequencies of the median class gives

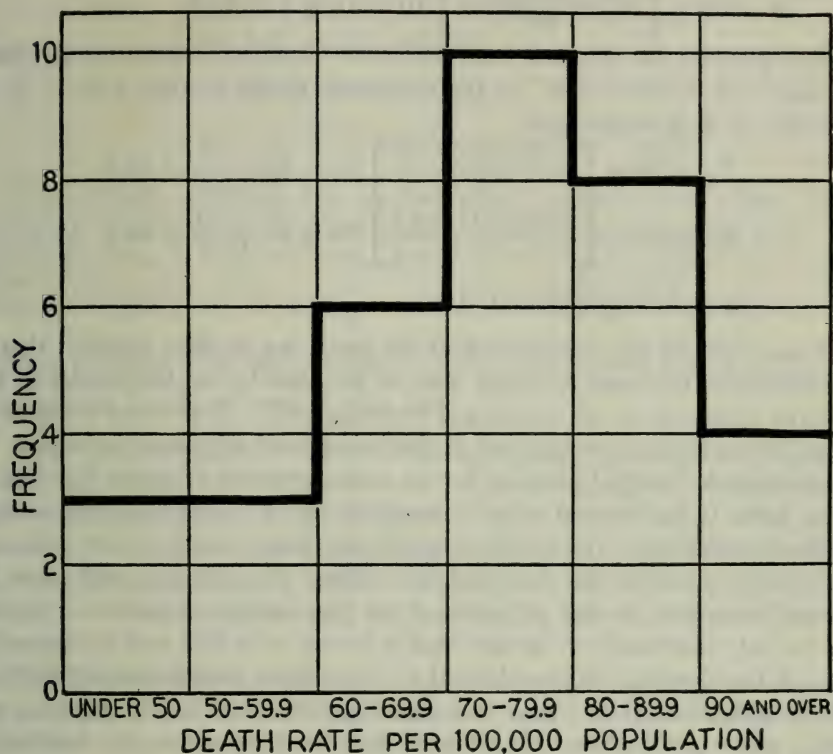


FIG. 127.—FREQUENCY DISTRIBUTION OF DEATH RATES FROM CANCER IN THIRTY-FOUR STATES OF THE UNITED STATES REGISTRATION AREA IN 1920.

the ratio of the interval covered by the median class which lies above or below the median value as the case may be. This can be expressed by a formula:

$$M = v + \frac{.50 N - F}{f} i \text{ when calculated from below.}$$

$$M = v' + \frac{.50 N - F'}{f} i \text{ when calculated from above.}$$

where v = value of the lower boundary of median class.

F = the sum of the frequencies falling below the value of the lower boundary of the median class.

f = the frequencies of the median class.

i = the interval covered by the median class.

N = total frequencies.

v' = value of the upper boundary of the median class.

F' = the sum of the frequencies falling above the value of the upper boundary of the median class.

In table calculating from below

$$v = 70, F = 12, f = 10, i = 10, N = 34,$$

$$M = 70 + \left[\frac{(.50)(34) - 12}{10} \right] 10 = 70 + 5 = 75.0.$$

The quartiles are obtained from the above formula by substituting "quartile class" for "median class" in the definitions of the symbols and .75 N or .25 N for .50 N in the formula.

$$P_{25} = 60 + \left[\frac{(.25)(34) - 6}{6} \right] 10 = 60 + \frac{25}{6} = 64.2$$

$$P_{75} = 80 + \left[\frac{(.75)(34) - 22}{8} \right] 10 = 80 + \frac{35}{8} = 84.4$$

$$Q = \frac{84.4 - 64.2}{2} = 10.1$$

From Table II the computation of the mean can be done directly, that is by multiplying the mean or center value of the class by the frequencies in the class and summing for all classes and dividing by N. It also can be done by the use of an arbitrary origin and if the intervals of all classes are equal, by substituting the integral numbers for the mean or center values of the classes.

In Table II the interval of each class is 10 points in the death rate excepting the first and last. The middle values of the classes are 55.0, 65.0, 75.0, etc. The middle values of the first and last classes are unknown and must be allocated according to one's judgment of the phenomenon in question. In this case the only observation in the last class is known to be 97.9 and the centering value of the class can be placed at 97.5. The three observations of the first class do not center at 45 but do not depart greatly from it. In selecting the middle value of the class it is assumed that the frequencies are distributed more or less uniformly throughout the class. It requires a lot of arithmetic to multiply the center values of the classes by the frequencies in each class, and there is introduced with it the chance of error. If an arbitrary mean be chosen at 75.0 and subtracted from the center values, the values of column designated $x - M_0$ are obtained. This reduces arithmetic. Since $x - M_0$ changes in units of 10 in the death rate, further simplification is obtained by letting $x' = \frac{x - M_0}{10}$. The frequencies are then multiplied by their values of x' , summed and divided by N. The mean of $x' = -.147$. Substituting for x' for its value in terms of x' we have

$$\frac{\sum x'}{N} = \sum \frac{x - M_0}{10} \frac{1}{N} = 1.47$$

$$\frac{\sum (x - M_0)}{N} = 10(-.147)$$

$$\frac{\sum x}{N} - M_0 = -1.47$$

$$\frac{\sum x}{N} = m_x = 75.0 - 1.47 = 73.53$$

Computing σ from Table II

$$\frac{\Sigma(x')^2}{N} = 2.03, \quad b^2 = (-.147)^2 = .022$$

$$\sigma_x'^2 = 2.03 - .022 = 2.008$$

$$\sigma_x' = \pm \sqrt{2.008} = \pm 1.42$$

$$\sigma_x = \pm 10(1.42) = 14.2, \quad \text{since } x' = \frac{x - M_0}{10}$$

The average deviation can be determined roughly from Table II by averaging the deviations from the median class without regard for sign. The sum of the plus deviations is 16 and that of the minus deviations 21. Summing and dividing by 34 gives $\frac{37}{34} = 1.09$. But in this example A.D. = 1.09 is given in terms of x' which equals $\frac{x - M_0}{10}$. To convert it to $x - M_0$, 1.09 is multiplied by 10, which is $10.9 = \text{A.D.}$

The constants computed from the grouped data differ slightly from those of the ungrouped data. This is due to the grouping and is liable to increase with coarser grouping. It is necessary to weigh the advantage of decreased numerical work against the disadvantage of the error introduced by grouping. This discrimination usually suffices in aiding one to decide upon a suitable grouping. Grouping should not be made larger than the sigma of the distribution.

The Normal Curve.—The normal frequency curve is a curve defined by an empirical mathematical formula which is used to describe frequency distributions. It is largely the foundation of statistical method. The normal curve is bell-shaped, unimodal and symmetrical with respect to the mean, median and mode.

The equation of the normal curve is

$$y = \frac{N}{\sigma \sqrt{2\pi}} e^{-\frac{1}{2} \left(\frac{x-m}{\sigma} \right)^2}, \quad \text{where}$$

y = frequency; N = total number of observations; $\pi = 3.1416 \dots$

e = Napierian base of natural logarithms = 2.7183...

x = the variable; m = the mean; σ = the standard deviation or root mean squared deviation. The constants which must be determined from the data are N , the mean and the standard deviation.

The following relations exist in a normal distribution:

$$\text{Mean} = \text{Median} = \text{Mode}$$

$$\sigma = 1.48 \dots \quad Q = 1.25 \dots \quad \text{A.D.}$$

$$\text{P.E. (Probable Error)} = .67448 \dots \quad \sigma = Q$$

The mean is the most reliable centering constant and σ is the most reliable dispersion constant.

Skewness, which is a condition of asymmetry, does not exist in a normal or any other symmetrical distribution. But in any sample there is a great chance of getting some skewness even though the universe from which it was taken be distributed symmetrically. Skewness when it exists can be readily seen in the histogram. It is present when the mean does not equal the median and also when the quartile deviation does not equal the distances between the median and either quartile. However, it is seldom necessary to determine skewness quantitatively. One is generally only interested in knowing whether the observed skewness is so great that it could not have arisen by chance in sampling a normally distributed universe. This question resolves itself into one of determining whether the observed data are distributed normally within limits of their sampling errors, which will be considered later. If the above

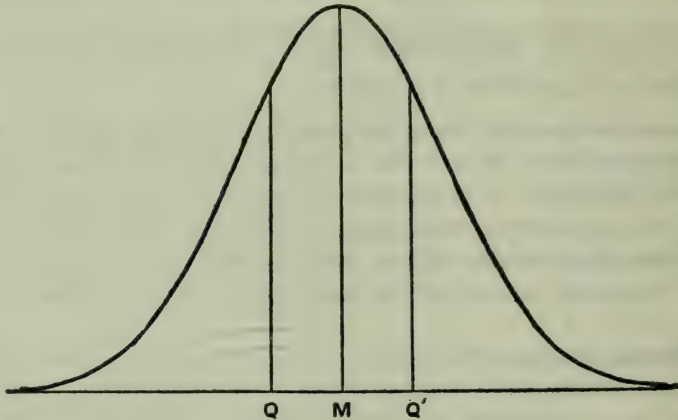


FIG. 128.—NORMAL CURVE.

(Lock.)

relations that exist in a normal curve are found to hold for the observed data within limits of their sampling errors, it can be concluded that the data are distributed normally and that the observed skewness is not so great that it could not have arisen by sampling.

The cancer death rates show slight skewness; the mean and the median do not coincide; Q is not equal to $M - P_{25}$. When, however, the sampling errors are estimated, the above differences are not significant and for all practical purposes this sample of cancer rates can be regarded as being normally distributed.

Choice of Statistical Constants.—When the observed data are known to be distributed normally the mean and the standard deviation should be used. When they are known not to be distributed normally, then for the beginner it is probably safer to use the median and the quartile deviation. When nothing is known about the distribution, any constant can be used—for one of the purposes in computing constants is to gain some knowledge of the frequency distribution.

The median is always safe to use and has always the same significance independent of the type of distribution. It is unaffected by the size of the deviations from it. This is not true of the mean since the sum of the deviations from it equal zero. One large deviation can measurably disturb the mean, whereas upon the median it would have no effect.

In a distribution which is not normal, the use of some type of mean other than the arithmetic mean is found necessary when other statistical constants, such as the correlation coefficient, the trend, standard deviation, etc., are to be computed. These constants, as well as sampling error formulæ, are so closely connected with the theory of least squares and the normal frequency curve that their significance is very questionable when the distribution is not normal.

The average deviation should be computed if the mean and the standard deviation are computed, because it serves to indicate roughly whether the data are distributed according to the normal frequency curve. $\sigma = 1.25$ A.D. (Fig. 126.)

Graphical Methods.—The graphic representation of statistical series leads to a more complete understanding of their characteristics. It supplements the information gained by the computation of the statistical constants. Although generally used to present in the most striking manner statistical facts to the uninitiated, the statistician finds the graphic method invaluable in the analysis of his problem. Hidden characteristics are often brought to light.

TABLE III

DEATHS FROM DIPHTHERIA IN MASSACHUSETTS IN 1924, DISTRIBUTED ACCORDING TO AGE AT DEATH

Age at Death	Frequency	Age at Death (cumulative)	Cumulative Frequency less than Stated Age	Per Cent of Total (cumulative)
Under 1	48			
1	71	1	48	9.0
2	83	2	119	21.2
3	62	3	202	37.8
4	72	4	264	49.4
5-9	141	5	336	63.0
10-14	31	10	477	89.2
15-19	5	15	508	95.1
20-29	8	20	513	96.0
30-39	5	30	521	97.5
40-49	5	40	526	98.5
50-59	2	50	531	99.3
60-69	1	60	533	99.8
Total	534			

The most commonly used plotting papers are the arithmetic or Cartesian paper, the arithmetic-logarithmic paper and the double logarithmic paper. The arithmetic and logarithmic probability papers of Dr. Hazen are useful for

plotting cumulative frequency distributions. Professor Wilson's logistic paper is valuable for plotting epidemics as well as frequency distributions. It is desirable to become familiar with at least one type of paper. The arithmetic is the simplest. All data might well be plotted first upon arithmetic paper and secondly upon the paper most suited to the nature of the data and the information desired from them. It is always necessary to have two associated items of information in order to make a plot, *e.g.*, death rates and years, death rates and months of year, magnitude of death rate and frequency of occurrence, death rate from one disease and death rate from a second. The ordinate or vertical axis measures one item, *e.g.*, death rate and the abscissa or horizontal axis measure the associated item, *e.g.*, year.

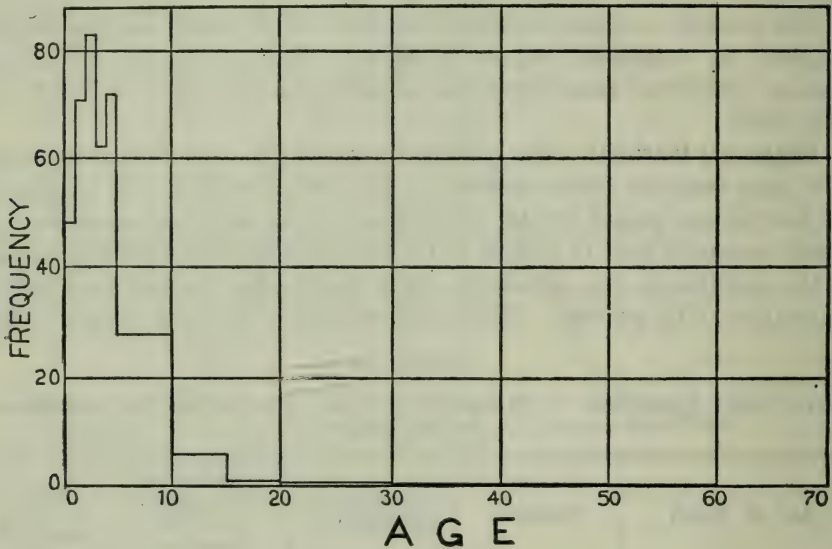


FIG. 129.—AGE DISTRIBUTION OF DIPHTHERIA DEATHS.

In Figure 129 the figures of Table III are plotted on arithmetic paper. The ordinate is the frequency and the abscissa is age. The frequencies are exactly represented by the area enclosed by the plotted lines. The curve is markedly asymmetrical. The peak falls in the vicinity of three to four years, which is the most common age at death from diphtheria.

The observations of Table IV are plotted on arithmetic paper and shown in Figure 130. The ordinate is the diphtheria death rate and the abscissa is time. The trend follows a curve downward and tends to approach a stationary value. The variation about the theoretical trend curve has disappeared to a large extent during the last seven years.

The data of Table VIII are plotted in Figure 131. The plotted points seem to follow a perceptible trend. In those states with high diabetes rates there is a tendency to have high cancer rates. The perceptible trend is practically a straight line.

The data of Table V are plotted in Figure 132. The trends of both cancer and diabetes are practically linear on arithmetic paper. A straight line on this paper pictures the annual amount of increase or decrease. The annual increase of diabetes is much less than that of cancer.

TABLE IV
DEATH RATE PER 100,000 FROM DIPHTHERIA IN MASSACHUSETTS

Year (x)	Death-Rate (y)	Log of DR (log y)	Log y-M ₀ + —	(Log y-M ₀) ²	(x-1912)	(Log y-M ₀) (x-1912) + —
1900...	53.0	1.72	.45	.202	— 12	5.40
01...	40.9	1.61	.34	.116	— 11	3.74
02...	30.2	1.48	.21	.044	— 10	2.10
03...	28.6	1.46	.19	.036	— 9	1.71
04...	23.5	1.37	.10	.010	— 8	.80
05...	21.6	1.33	.06	.004	— 7	.42
06...	24.0	1.38	.11	.012	— 6	.66
07...	23.8	1.38	.11	.012	— 5	.55
08...	23.1	1.36	.09	.008	— 4	.36
09...	18.0	1.26	.01	.000	— 3	.03
10...	20.1	1.30	.03	.001	— 2	.06
11...	16.4	1.21	.06	.004	— 1	.06
12...	13.5	1.13	.14	.020	0	0
13...	17.6	1.25	.02	.000	+ 1	.02
14...	17.9	1.25	.02	.000	+ 2	.04
15...	19.5	1.29	.02	.000	+ 3	.06
16...	16.7	1.22	.05	.002	+ 4	.20
17...	21.8	1.34	.07	.005	+ 5	.35
18...	15.6	1.19	.08	.006	+ 6	.48
19...	15.5	1.19	.08	.006	+ 7	.56
20...	15.3	1.18	.09	.008	+ 8	.72
21...	15.6	1.19	.08	.006	+ 9	.72
22...	15.4	1.19	.08	.006	+ 10	.80
23...	14.6	1.16	.11	.012	+ 11	1.21
24...	13.3	1.12	.15	.022	+ 12	1.80
Sum..			1.78 .97 + .81	.542		.50 22.35 — 21.85
Sum N			+ .032	.0217		— .874

$$M_0 = 1.27$$

$$m = 1.27 + .032 = 1.302$$

$$\sigma_r = \pm \sqrt{.0217 - (.032)^2} = \pm \sqrt{.0207} = \pm .144$$

$$\frac{N^2 - 1}{12} = 52.$$

$$d = \frac{-.874}{52} = -.0168 = -.017$$

$$\sigma_r = \pm [(.0207) - (-.017)^2 52]^{\frac{1}{2}} = \pm \sqrt{.0057} = \pm .0755 = \pm .076$$

The ordinate of the arithmetic-logarithmic paper is ruled on a logarithmic scale and the abscissa on an arithmetic scale. The same picture is produced if the logarithms of the ordinate variable are plotted against the abscissa variable on arithmetic paper. This type of plot is useful in the study of

trends. It pictures the percentage increase or decrease of the variable on the ordinate.

The data of Table IV are plotted on arith.-log. paper and shown in Figure 133. Except for the years 1900 and 1901 the logarithm of the diphtheria death rate practically follows a straight line downward. The percentage decrease of one year over the preceding year is constant. On arith.-log. paper the tendency of the death rate to approach a stationary value is not apparent. The variation about the theoretical straight line trend is distorted on this

TABLE V
DEATH RATES PER 100,000 IN MASSACHUSETTS

Year	Population	Cancer	Diabetes
1900.....	2,805,346	71.2	11.8
1901.....	2,849,047	73.1	11.2
1902.....	2,889,386	74.3	13.1
1903.....	2,929,725	76.8	14.0
1904.....	2,970,064	81.7	14.1
1905.....	3,015,872	83.3	14.2
1906.....	3,089,029	85.5	13.7
1907.....	3,162,186	89.0	15.1
1908.....	3,235,343	89.8	13.9
1909.....	3,308,500	90.6	15.5
1910.....	3,380,151	90.0	17.0
1911.....	3,445,416	92.8	18.3
1912.....	3,510,795	93.5	16.7
1913.....	3,576,174	98.6	17.4
1914.....	3,641,553	98.5	17.3
1915.....	3,701,683	100.2	18.2
1916.....	3,735,166	106.8	21.0
1917.....	3,768,649	107.7	20.6
1918.....	3,802,132	107.8	17.9
1919.....	3,835,615	107.2	16.5
1920.....	3,869,098	116.1	20.6
1921.....	3,902,581	119.0	19.9
1922.....	3,936,064	118.1	24.1
1923.....	3,969,547	120.3	21.4
1924.....	4,003,030	126.8	19.3

paper. Variation above the trend curve is shortened and variation below it is increased. The absence of variation during the last seven years is as well shown on this paper as on the arithmetic.

The death rates of Table V are plotted in Figure 134 on arith.-log. paper. It is very noticeable upon comparing Figure 132 and Figure 134 that the trends are quite different. In Figure 134 the trends of both cancer and diabetes are practically linear and also about equal to one another. Arithmetic paper pictures the amount of increase per year and the arith.-log. paper the per cent of increase of one year over the preceding year. The latter is entirely independent of the size of the death rate.

The double logarithmic paper is more limited in use than the two aforementioned papers. Both the ordinate and the abscissa are ruled on a logarith-

mic scale. It is chiefly applicable in the determination of the type of relation between two variables. Farr's law states that the death rate is proportional to the 10th or 12th root of the density of population. $D.R. = K \sqrt[12]{\text{Density}}$. The 12th root of the density is hardly perceptible on arithmetic paper unless density varies within very wide limits. If logarithms are taken for

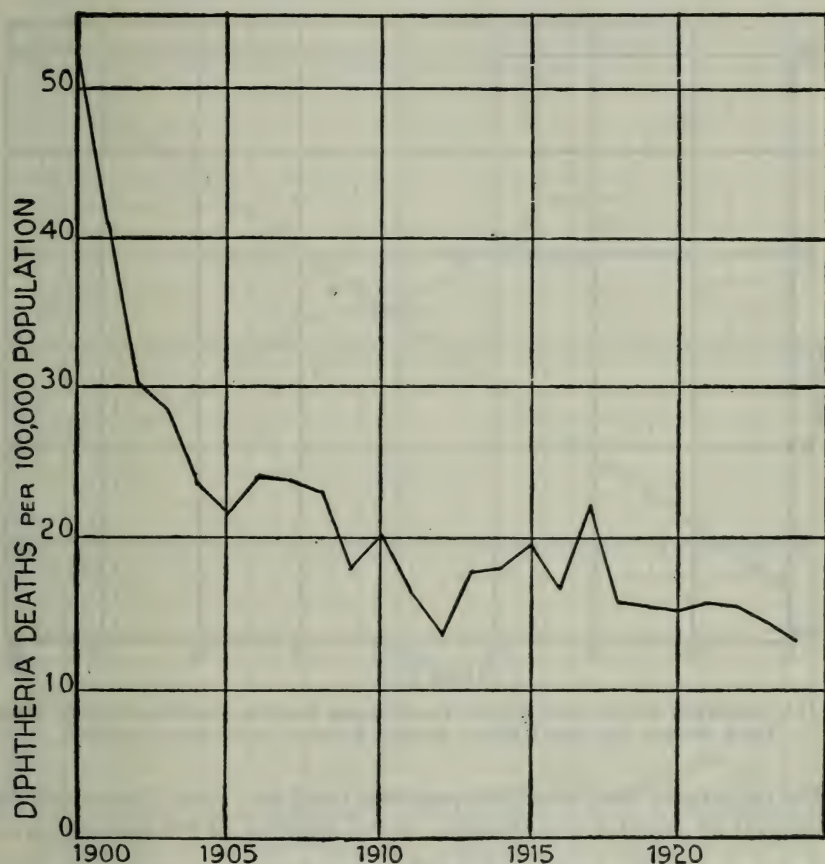


FIG. 130.—TREND OF DEATH RATE FROM DIPHTHERIA IN MASSACHUSETTS.

both death rate and density, then the above equation becomes $\log D.R. = \log K + \frac{1}{12} \log \text{Density}$. This is a straight line if plotted on arithmetic paper and if it has a perceptible upward trend, then the relation is present. The same straight line is produced by plotting D.R. and Density on double logarithmic paper, and on this paper Farr's law can become perceptible.

Assuming as a very rough measure that density is equal to the number of persons per square mile, the cancer death rates of Table V can be plotted

on double logarithmic paper against the density of each state as recorded in the 1920 census and are shown in Figure 135.

There is a perceptible upward trend. The line was drawn in roughly by hand and the trend of the line estimated to be $+.08$. This would indicate

very roughly that $C.D.R. = K \sqrt[12.5]{\text{Density}}$. For an exact determination, the trend should be computed from the data, and its significance evaluated.

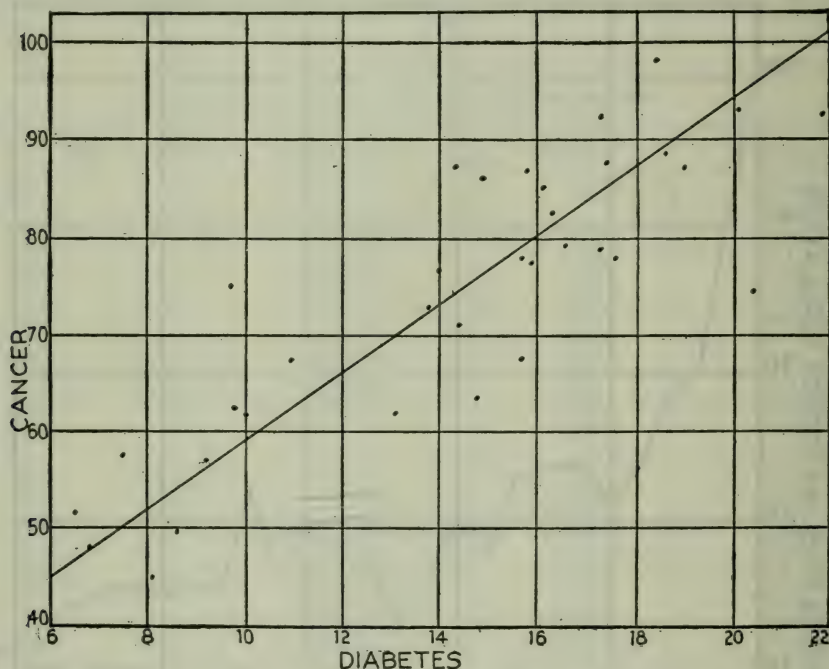


FIG. 131.—SCATTER DIAGRAM OF DEATH RATES FROM CANCER AND DIABETES IN THIRTY-FOUR STATES OF THE UNITED STATES REGISTRATION AREA IN 1920.

The ordinate of the probability papers is ruled on a scale determined from the integral or cumulative summation of the equation of the normal curve of

frequency $y = \frac{N}{\sigma \sqrt{2\pi}} e^{-\frac{1}{2} \left(\frac{x-m}{\sigma} \right)^2}$. The abscissa of the arithmetic proba-

bility paper is ruled on an arithmetic scale and the abscissa of the logarithmic probability paper is ruled on a logarithmic scale. Many frequency distributions are normal in the logarithm of the variable. The paper is of value in determining whether or not the frequency distribution of the data agrees with the normal curve. If it does agree the data will plot as a straight line. The actual frequencies cannot be plotted directly on this paper but are first accumulated and then converted to percentages. The cumulative percentage is plotted. The cumulative percentage is the percentage of all frequencies that are less than the given value of x .

The cumulative percentages of Table VI are plotted against the death rate on arithmetic probability paper in Figure 136. The points follow a straight line very well. The line was drawn in roughly. Being a normal distribution, the point on the abscissa at which the line cuts the 50 per cent line is the value for the mean, median and mode. The distance on the abscissa between

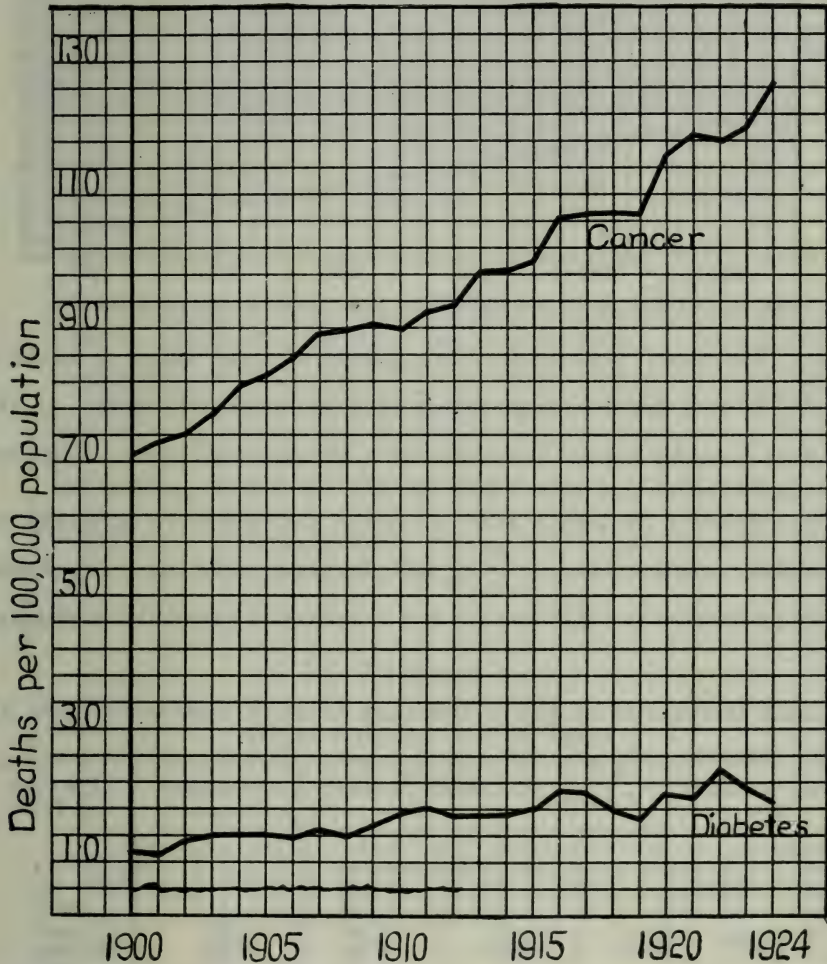


FIG. 132.—TRENDS OF DEATH RATES FROM CANCER AND DIABETES IN MASSACHUSETTS.

the points where the line cuts the 50 per cent and 75 per cent lines is roughly $\frac{2}{3}\sigma$. The use of this paper enables one to estimate quickly and easily and with a fair amount of accuracy the constants of the frequency distribution when the distribution is normal.

The cumulative percentages of Table III are plotted against age on arithmetic probability paper and shown in Figure 137. The points depart greatly

from a straight line. The percentages are plotted on logarithmic probability paper and shown in Figure 139.

Except for the higher ages the points follow a straight line pretty well. The age distribution is more normal in the logarithm of age than it is in age

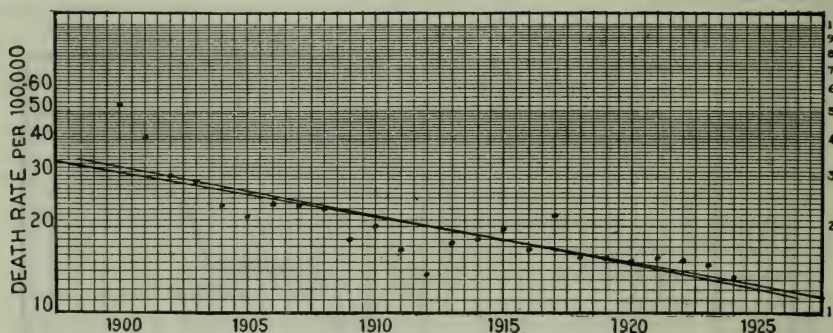


FIG. 133.—TREND OF DEATH RATE FROM DIPHTHERIA IN MASSACHUSETTS.

itself. This would indicate that the geometric mean which is equal to $(10) \frac{\sum \log x}{N}$ is a better average to use than the arithmetic mean. In this case, were it strictly normal in the logarithm, the geometric mean, median and mode would fall at that value where the line cuts the 50 per cent line.

The ordinate of the logistic grid ¹ is ruled so that data which agree with the autocatalytic equation $y = \frac{K}{1 + me^{-ax}}$ will plot as a straight line. The abscissa is ruled arithmetically. All data are first converted to cumulative percentages

TABLE VI

FREQUENCY DISTRIBUTION OF INFANT MORTALITY RATES IN FORTY-SEVEN CITIES AND TOWNS OF MASSACHUSETTS IN 1924

Infant Mortality Rate	Frequency	Infant Mortality Rate	Cumulative Frequency	Per Cent of Total
Under 30.....	1	Less than 30	1	2.1
30-39.....	3	“ “ 40	4	8.5
40-49.....	7	“ “ 50	11	23.4
50-59.....	10	“ “ 60	21	44.7
60-69.....	12	“ “ 70	33	70.2
70-79.....	9	“ “ 80	42	89.5
80-89.....	2	“ “ 90	44	93.5
90-over.....	3			
TOTAL.....	47			

before plotting. This is readily done for epidemics and frequency distributions where the total number of cases and frequencies is known. Ross' theory of epidemics in some cases leads to this equation and the paper offers a short

¹ Edwin B. Wilson, "The Logistic or Autocatalytic Grid." *Proc. Nat. Acad. Sc.*, 1925, 11: 8.

and simple method of testing the agreement between the theory and the observation. Also it is not unlikely that some frequency distributions which are symmetrical but not normal, which have too high a percentage of frequencies at the tails of the distribution, will be found to agree with this equation. This paper is also useful in plotting the growth of populations for the description

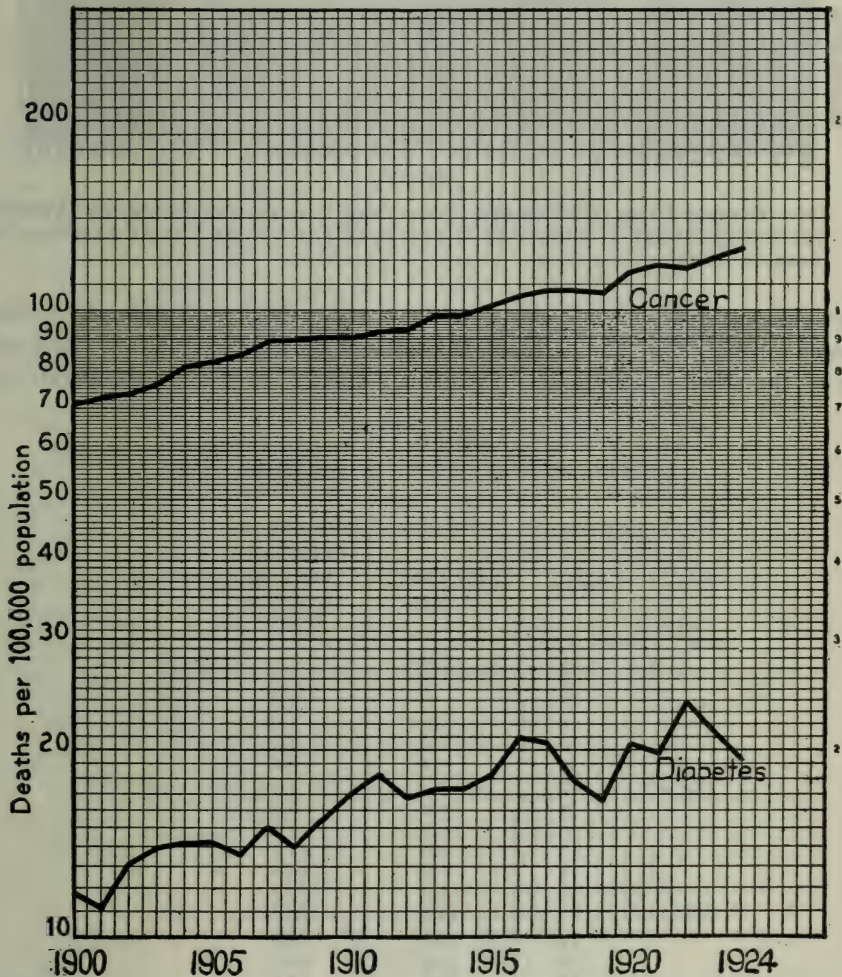


FIG. 134.—TRENDS OF DEATH RATES FROM CANCER AND DIABETES IN MASSACHUSETTS.

of which the above equation is used. The data of Table VII are shown in Figure 139 on the logistic grid.

Trend.—Many phenomena in vital statistics change with time. Annual fluctuations almost invariably occur, but underlying these there is usually a gradual trend upward or downward that is noticeable over longer periods of time. In studying the annual variation of a phenomenon the trend frequently obscures the picture. It is often desirable to remove it. Sometimes it is neces-

sary to form some rough estimate of an unknown datum, as for example the probable death rate in 1918 if the influenza had not struck, or the probable death rate for 1925 not yet published. If the unknown datum lies among the

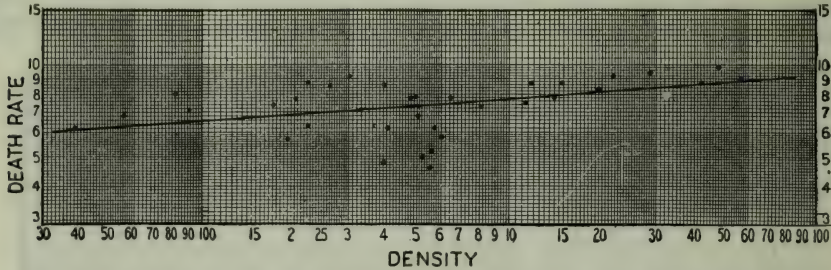


FIG. 135.—SCATTER DIAGRAM OF DEATH RATES FROM CANCER, AND DENSITIES (PERSONS PER SQUARE MILE) IN THIRTY-FOUR STATES OF THE UNITED STATES REGISTRATION AREA IN 1920.

other observations, its interpolated value on the trend line is generally a more reliable estimate than the extrapolated value of an unknown datum lying outside of the other observations. Frequently trends are studied comparatively,

TABLE VII
EPIDEMIC OF POLIOMYELITIS IN BALTIMORE, 1916
(208 CASES)

Week	Per Cent of Cases	Cumulative per Cent Occurring Before
1	.5	
2	.5	.5
3	0	1.0
4	.5	1.0
5	1.0	1.5
6	2.4	2.5
7	1.4	4.9
8	6.2	6.3
9	8.7	12.5
10	5.3	21.2
11	6.2	26.5
12	5.3	32.7
13	14.9	38.0
14	10.1	52.9
15	11.5	63.0
16	7.7	74.5
17	5.8	82.2
18	4.8	88.0
19	2.4	92.8
20	2.4	95.2
21	2.4	97.6

as for example the trend of the death rate of tuberculosis of different age groups. In general, public health workers find many occasions where the study of trends is helpful in obtaining a better understanding of their problems.

The simplest trend is the straight line. The first step in determining a trend is to plot the data on arithmetic paper and to see if there is a trend. It often happens that there is a trend but it is not a straight line. Should the trend follow a curve, then plot on arithmetic-logarithmic paper and very often it becomes linear. Should it, however, fail to straighten out, then it becomes

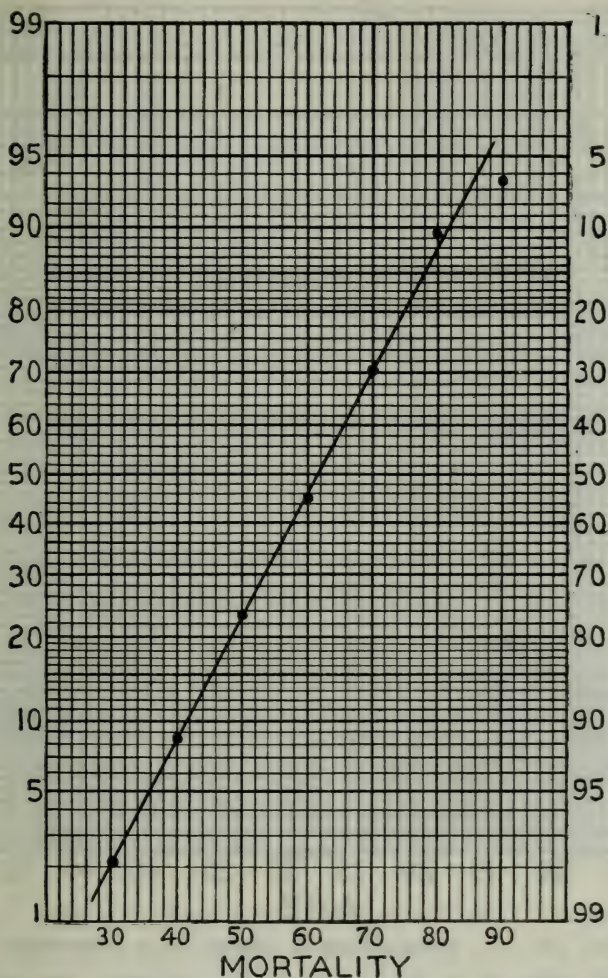


FIG. 136.—FREQUENCY DISTRIBUTION OF INFANT MORTALITY RATES IN FORTY-SEVEN CITIES AND TOWNS IN MASSACHUSETTS IN 1924.

necessary to plot, not the death rate, but some function of the death rate on arithmetic paper. By trying different functions of either the death rate or time it is possible to find one that plots as a straight line. This is not always easy and also not always necessary since by the use of the arithmetic and arith.-log. papers many trends are practically straight, particularly if too long periods of time are not chosen over which to study the trend. Frequently it

is a perfectly adequate procedure to break up a long curvilinear trend into three or four short straight-line trends.

When the plotted points follow approximately a straight line, a line can be drawn through them in such a way that it lies in the middle of them and yet follows the perceptible trend. This graphic method is very often sufficient for

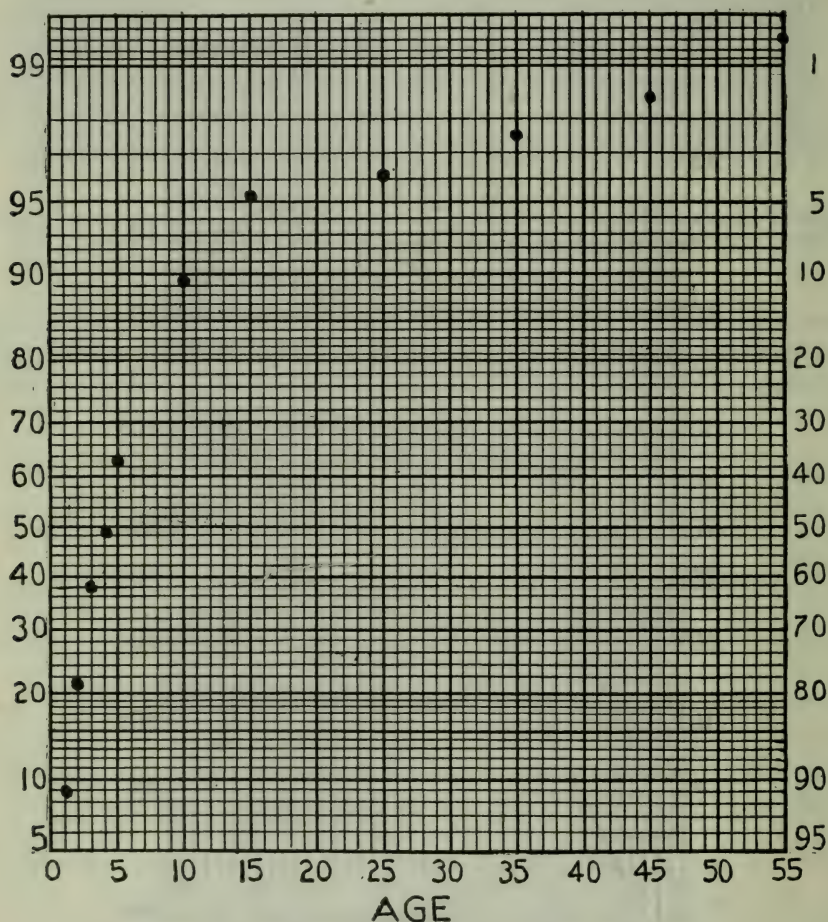


FIG. 137.—AGE DISTRIBUTION OF DEATHS FROM DIPHTHERIA.

graph. The equation of a straight line is $y = a + dx$, where a and d are constants to be determined from the data and where x = time and y = the phenomenon under consideration. When the straight line is derived by the method of least squares, then the constants a and b can be interpreted in terms

of other familiar constants. Then $a = m_y - dm_x$ and $d = \frac{\frac{\sum xy}{N} - m_y m_x}{\sigma_x^2}$

The only unfamiliar term in the above expressions is the mean product $\frac{\sum xy}{N}$.

It is the sum of the products of the associated values of y and x divided by the total number of observations; d is the numerical measure of the trend. It is positive in sign when the trend is upward and negative when downward. It can be evaluated from the line drawn in the graph by reading from the line the y value at the first year and the y value at the last year. The difference between the two y values is the amount of increase or decrease over the number of years from the first to the last. Dividing the amount of increase or de-

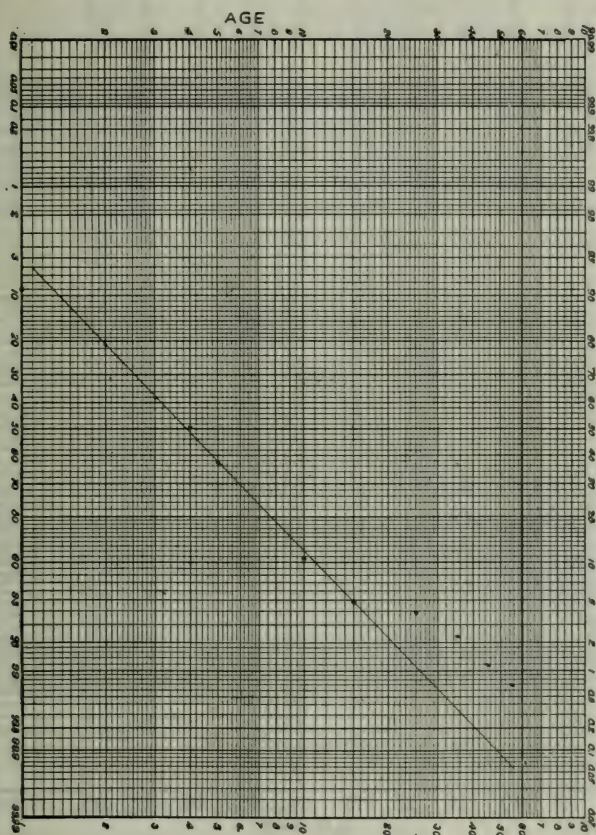


FIG. 138.—AGE DISTRIBUTION OF DEATHS FROM DIPHTHERIA.

crease by the total number of years over which it has taken place gives the amount of increase or decrease per year, which is d , the trend. The evaluation of the constant a is simple. It is the y value read from the line at the point where $x = 0$. Obviously this depends entirely upon the point from which time is reckoned. This point, the origin of x , can be arbitrarily chosen anywhere, but it is most convenient to choose it at the middle year of the series under consideration.

In Figure 130 the death rates from diphtheria in Massachusetts over a period from 1900-1924 are plotted on arithmetic paper. The trend is curvi-

linear. In Figure 133 the same death rates are plotted on arith.-log. paper and the points follow approximately a straight line. The longer line was drawn in to represent the trend. Read from the line the diphtheria death rate at 1900 is roughly 30.9 and at 1924 is 12.9. Since the rates are plotted on arith.-log. paper, which is equivalent to taking the log of the death rate and plotting on arithmetic paper, and since the equation of a line on arith.-log.

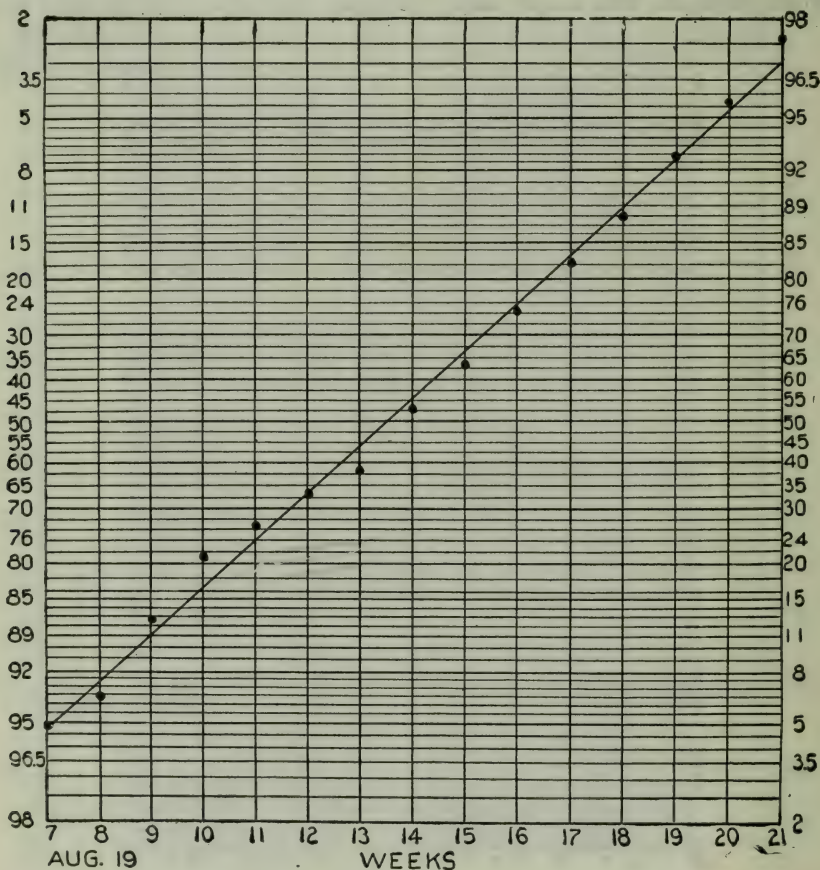


FIG. 139.—EPIDEMIC OF POLIOMYELITIS IN BALTIMORE, 1916.

paper is $\log y = a + dx$, the logarithm of the two values must be taken before finding their differences. The log of 30.9 is 1.490 and of 12.9 is 1.111. The difference is $-.379$ over an interval of twenty-four years. The trend is

therefore $\frac{-.379}{24} = -.0158 = -.016$. Placing the origin of time at 1912,

the middle year of the range, and reading from the line the value of the death rate which is 20 and taking the log which is 1.301 gives the value of a . The equation of the line is then $\log y = 1.301 - .016x$ and converting from log to numbers becomes $y = (10)^{1.301-.016x}$. If this method is not exact enough for the problem in hand then the constants a and d can be computed from the data:

Since the diphtheria rates are straight on arith.-log. paper the rates must be converted to logarithms before computation. The constants a and d of the line $\log y = a + dx$ are practically the same as those given above except that \log

y is substituted for y . Then $a = m_{\log y} - d m_x$ and $d = \frac{\frac{\sum x \log y}{N} - m_{\log y} m_x}{\sigma_x^2}$.

These expressions can be simplified if the origin of time is chosen at the middle year. Then the $m_x = 0$ and $\sigma_x^2 = \frac{N^2 - 1}{12}$, where N is the total num-

ber of observations. The middle year is given by $\frac{N+1}{2}$. When N is odd it

falls on a given year and when N is even it falls between the middle two years.

Substituting the values of m_x and σ_x^2 in the expressions for a and d , $a = m_{\log y}$

and $d = \frac{12}{N^2 - 1} \frac{\sum x \log y}{N}$. This leaves but two terms to compute from the data.

In Table IV are shown the diphtheria death rates with the form of computation. The $m_{\log y} = \frac{\sum \log y}{N} = 1.302$, $\frac{\sum x \log y}{N} = -.874$, $a = 1.302$,

$d = \frac{12}{N^2 - 1} (-.874) = -.0168 = -.017$.

In many problems there arises the need of determining the variation about the trend line. Every measure of dispersion is greater in a phenomenon that has a trend than it is when the trend is removed, and since it is not generally apparent that a long trend has any influence upon annual variations, the actual variation is best studied with the trend removed. The variation about a straight line computed from the data by the above formulæ can be expressed in terms of constants of the line and the original σ of the data.

$$\sigma_\pi = \sigma_y \left(1 - d^2 \frac{\sigma_x^2}{\sigma_y^2} \right)^{\frac{1}{2}} = (\sigma_y^2 - d^2 \sigma_x^2)^{\frac{1}{2}} = \left[\sigma_y^2 - d^2 \frac{N^2 - 1}{12} \right]^{\frac{1}{2}}$$

where σ is the variation about the line.

When computing the line $\log y = a + dx$ then

$$\sigma_\pi = \sigma_{\log y} \left(1 - d^2 \frac{\sigma_x^2}{\sigma_{\log y}^2} \right)^{\frac{1}{2}} = (\sigma_{\log y}^2 - d^2 \sigma_x^2)^{\frac{1}{2}} = \left(\sigma_{\log y}^2 - d^2 \frac{N^2 - 1}{12} \right)^{\frac{1}{2}}$$

It is seen that the σ_π = the original sigma multiplied by a factor less than one if a trend is present. This factor $\left(1 - d^2 \frac{\sigma_x^2}{\sigma_y^2} \right)^{\frac{1}{2}}$ is a pure number varying from 0 to 1 and is zero when all the points fall upon the line.

To determine the variation of the diphtheria rates with the trend removed it is necessary to compute $\sigma_{\log y}^2$ which is $\frac{\sum (\log y)^2}{N} - \left(\frac{\sum \log y}{N} \right)^2 = (.0207)$

$$\sigma_\pi = \left((.0207) - (.017) \frac{N^2 - 1}{12} \right)^{\frac{1}{2}} = (.0057)^{\frac{1}{2}} = \pm .0755 = \pm .076.$$

A trend can be greatly disturbed by large deviations in y from the m_y , particularly if they occur at the extremes of the line. The points at the extremes of the line have greater weight in determining the trend than the points in the middle, and this should be kept in mind when interpreting the trend. Predictions for more than a year or two from the ends of the line are not at all safe and should be made with caution.

Correlation Coefficient.—One of the most widely used methods of measuring relationship is the correlation coefficient. It measures the extent to which the variation of one variable is associated positively or negatively with the variation of a second variable. An appreciation of association of variation between variables can be obtained by a consideration of a two-way frequency graph or scatter diagram such as Figure 131 and a correlation table such as Table IX. In scatter diagrams when correlation is present the points will be seen to cluster more or less closely about a trend and the coefficient measures the amount of clustering. Similarly clustering can be seen in a correlation table.² It is apparent that some trend must be present between the variables, otherwise the coefficient denoted by r will be zero. The trend is always assumed to be a straight line. The true relationship between any two variables in vital statistics and in biological fields in general, very frequently defies description, due to the fact that many other variables are associated to such a degree and probably in such diverse ways that the relationship is entirely obscured. The first approximation to the measurement of a functional relationship between variables is the assumption that they are related linearly or by a straight line. With this assumption r measures the degree of relationship. r varies from -1 perfect negative association, to $+1$ perfect positive association. $r = 0$ means no association. The coefficient does not signify that the clustering is constant throughout all points of the line. Clustering may be complete at one part of the line, that is, all the points may fall upon the line at one part of it and be widely scattered at another part. In such cases r measures only the average clustering. The coefficient r is extremely sensitive to scattering and clustering at both ends of the straight line. There is, however, one exception to the tendency of r to measure average association and that is when both variables form a normal frequency surface, in which case they are distributed according to the normal frequency curve. In this condition r measures the association existing at any point on the line and is not greatly affected one way or the other by the variation at the extremes of the trend line, since when a normal frequency surface is present large deviations at the ends do not exist. However, when distributions are practically normal r is nevertheless sensitive to large deviations even when they are not frequent enough to disturb measurably the normality of the distributions. Even though the distribution of each variable be normal, together they may not form a normal frequency surface.

r is a constant of a straight line when fitted by least squares. It is contained in the trend constant d of the straight line $y = a + dx$.

² Pearl, *Medical Biometry and Statistics*, pp. 293-300.

$$r = \frac{\frac{\Sigma xy}{N} - m_y m_x}{\sigma_y \sigma_x}, d = r \frac{\sigma_y}{\sigma_x} = \frac{\frac{\Sigma xy}{N} - m_y m_x}{\sigma_y \sigma_x} \cdot \frac{\sigma_y}{\sigma_x} = \frac{\frac{\Sigma xy}{N} - m_y m_x}{\sigma_y^2}$$

$a = m_y - d m_x$. As with the trend line the measure of variation about the line is $(\sigma_y^2 - d^2 \sigma_x^2)^{\frac{1}{2}} = \sigma_y \left(1 - d^2 \frac{\sigma_x^2}{\sigma_y^2}\right)^{\frac{1}{2}} = \sigma_y (1 - r^2)^{\frac{1}{2}}$

The computation of r is very similar to that of a trend. It is not very frequently possible to simplify computation by selecting the origin at the mean or middle point of either one of the variables due to the infrequency of

TABLE VIII

DEATH RATE FROM DIABETES AND CANCER IN THIRTY-FOUR STATES OF THE UNITED STATES REGISTRATION AREA IN 1920.

State	Cancer DR (y)	y-M _{oy}	(y-M _o) ²	Diabetes D.R. (x)	x-M _{ox}	(x-M _o) ²	(y-M _{oy}) (x-M _{ox})
		$\begin{smallmatrix} + \\ - \end{smallmatrix}$			$\begin{smallmatrix} + \\ - \end{smallmatrix}$		$\begin{smallmatrix} + \\ - \end{smallmatrix}$
Mass.	97.9	20.9	437.0	18.4	3.4	11.6	71.1
Conn.	92.8	15.8	249.6	20.1	5.1	26.0	80.6
N. Y.	92.3	15.3	234.1	21.9	6.9	47.6	105.6
Minn.	92.1	15.1	228.0	17.3	2.3	5.3	34.8
R. I.	88.5	11.5	132.3	18.6	3.6	13.0	41.4
Ill.	87.4	10.4	108.2	17.4	2.4	5.8	25.0
Cal.	87.1	10.1	102.0	14.3		.5	
N. J.	86.9	9.9	98.0	19.0	4.0	16.0	39.6
Md.	86.7	9.7	94.1	15.8	.8	.6	7.8
Ver.	85.7	8.7	75.7	14.9		.0	
Me.	85.1	8.1	65.6	16.1	1.1	1.2	8.9
Penn.	82.6	5.6	31.4	16.3	1.3	1.7	7.3
Ore.	79.3	2.3	5.3	16.6	1.6	2.6	3.7
N. H.	78.9	1.9	3.6	17.3	2.3	5.3	4.4
Mich.	78.1	1.1	1.2	15.7	.7	.5	.8
Wis.	78.1	1.1	1.2	17.6	2.6	6.8	2.9
Ohio	77.7	.7	.5	15.9	.9	.8	.6
Wash.	76.8	0.2	0	14.0		1.0	.2
Del.	75.1	1.9	3.6	9.7	5.3	28.1	10.1
Neb.	74.3	2.7	7.3	20.4	5.4	29.2	14.6
Ind.	72.7	4.3	18.5	13.8	1.2	1.4	5.2
Col.	71.1	5.9	34.8	14.4	.6	.4	3.5
Utah	67.7	9.3	86.5	15.7	.7	.5	
Mo.	67.5	9.3	90.3	10.9	4.1	16.8	38.9
Kan.	63.4	13.6	185.0	14.8	.2	.0	2.7
La.	62.3	14.7	216.1	9.8	5.2	27.0	76.5
Mont.	61.9	15.1	228.0	13.1	1.9	3.6	28.7
Va.	61.9	15.1	228.0	10.0	5.0	25.0	75.5
Ky.	57.7	19.3	372.0	7.5	7.5	56.3	144.9
Fla.	56.9	20.1	404.0	9.2	5.8	33.6	116.5
Tenn.	51.6	25.4	645.0	6.5	8.5	72.3	216.0
N. C.	49.7	27.3	745.0	8.6	6.4	41.0	174.5
Miss.	48.0	29.0	841.0	6.8	8.2	67.2	238.0
S. C.	45.8	31.2	973.0	8.1	6.9	47.6	215.4
Sum.		148.2 244.6 — 96.4	6945.9		45.1 68.6 — 23.5	596.3	1781.1 29.1 + 1752.0
Sum N		— 2.84	204.29		— 0.69	17.54	51.53

$$m_y = 77.0 - 2.84 = 74.16 = 74.2$$

$$\sigma_y = \sqrt{204.29 - (-2.84)^2} = \pm \sqrt{196.2} = \pm 14.0$$

$$A.D. = 11.55 = 11.6$$

$$1.25 A.D. = 14.44$$

$$m_x = 15.0 - 0.69 = 14.3$$

$$\sigma_x = \pm \sqrt{17.54 - (-.69)^2} = \pm \sqrt{17.06} = \pm 4.13$$

$$A.D. = 3.34$$

$$1.25 A.D. = 4.18$$

variables being evenly spaced. Five constants therefore must be usually computed from the data, the two means, the two sigmas, and the mean product

$\frac{\sum xy}{N}$. Actually it involves the computation of only the product term since both means and standard deviations have usually been previously computed. The use of a median or a round number near the median as an arbitrary origin reduces the arithmetic and the probability of error. In Table VIII are shown the death rates of diabetes and cancer adjusted to age and sex for 34 states in the registration area. Each state has a diabetes and cancer rate and it is desired to determine if they vary together or inversely or independently of each other. Arbitrary origins at 77.0 for cancer and 15.0 for diabetes are chosen and the deviations of the observations from the respective origins computed. The mean product $\frac{\sum xy}{N}$ is found by multiplying each associated deviation and summing them and dividing by the total number of observations. In computing with the deviations from an arbitrary origin the formula for r becomes

$$r = \frac{\frac{\sum x'y'}{N} - b_y b_x}{\sigma_y \sigma_x}$$

where $\frac{\sum x'y'}{N}$ = the product of the deviations and b_y and b_x are the sums of the deviations divided by N or the corrections to the arithmetic means. This gives the same value for r as that obtained by computing with the original observations. r is not changed by a change of origin.

$\frac{\sum x'y'}{N} = (51.53)$, $b_y = (-2.84)$, $b_x = (-.69)$, $\sigma_y = (14.0)$, $\sigma_x = (4.13)$, $r = (.86)$.

Very frequently it is more convenient to group the observations into a correlation table. This is particularly desirable when dealing with a large number of observations. By such grouping the computation involved is greatly reduced. The cancer and diabetes rates are grouped and shown in Table IX. Where the grouping is made in equal intervals as in Table IX an arbitrary origin can be chosen and integral numbers can be substituted for the groups. This makes the computation relatively simple. Care must be taken to substitute the new value of the variable for the mean or middle value of the interval. The arbitrary origin can be chosen at any place but it is often most convenient to choose it at that group in which the median falls. The entire computation is carried through in terms of the substituted value of the variable. In Table IX if y is the cancer death rate and y' the substituted value, they $y' = \frac{y - 77.5}{5}$. Similarly $x' = \frac{x - 15}{2}$. The formula for r then becomes

$$\frac{\frac{\sum x'y'}{N} - m_{x'} m_{y'}}{\sigma_{x'} \sigma_{y'}}$$

TABLE IX

DEATH RATES FROM DIABETES AND CANCER IN THIRTY-FOUR STATES OF THE UNITED STATES REGISTRATION AREA IN 1920.

CORRELATION TABLE

DIABETES DEATH-RATE (x)													
Under 8	8-9	10-11	12-13	14-15	16-17	18-19	20 +	Freq.	y'	y' (Freq.)	(y') ² (Freq.)	x'y' (Freq.)	
40-49	(18) 2	3	6	—	18	108	24, 36, 60
50-54	1	5	—	5	25	20, 20
55-59	(12) 1	2	4	—	8	32	16, 12, 28
60-64	(9) 1	(6) 1	(3) 1	(0) 1	4	3	—	12	36	9, 6, 3, 18
65-69	(4) 1	..	(0) 1	2	2	—	4	8	4, 4
70-74	(1) 1	(0) 1	(-3) 1	3	1	—	3	3	1, -3, -2
75-79	(0) 1	(0) 3	(0) 3	7	0	—	0	0	0, 0
80-84	(1) 1	1	1	—	1	1	1, -1
85-89	(0) 3	(2) 2	(4) 2	..	7	2	—	14	28	4, 8, 12
90-94	(3) 1	..	(9) 2	3	3	—	9	27	3, 18, 21
95 +	(8) 1	..	1	4	—	4	16	8, 8
Freq.	3	5	2	9	7	3	3	34	..	—	50 + 28 — 22	284	172 — 2 — 170 5.0
x'	— 4	— 3	— 2	0	1	2	3	..	Sum — 34	— 0.323	— 0.646	8.35	
(x') (Freq.)	— 12	— 15	— 4	0	7	6	9	33 + 22 — 11	4.38	— 0.323	— 0.646		
(x') ² (Freq.)	48	45	8	0	7	12	27	149
x'y' (Freq.)	24	36	6	..	1	8	— 3
	20	12	4	..	3	8	18
	16	9	3
	60	57	10	4	8	16	15	170	5.0

$$\frac{mx'}{m^2x'} = -0.646$$
$$\frac{my'}{m^2y'} = -0.323$$
$$\sigma_{x'} = \pm\sqrt{8.35 - (-0.646)^2} = \pm\sqrt{7.93} = \pm 2.816$$
$$\sigma_{y'} = \pm\sqrt{4.38 - (-0.323)^2} = \pm\sqrt{4.28} = \pm 2.069$$
$$\sigma_{x'y'} = \pm\sqrt{5.0 - (-0.646)(-0.323)} = \pm\sqrt{4.79} = \pm 2.188$$
$$r = \frac{5.0}{5.83} = 0.858$$
$$(2.82)(2.07)$$

$$m_{x'} = -0.646$$

$$m_{x'} = -0.323$$

$$m_{y'} = \pm \sqrt{8.35} = \pm \sqrt{(-0.646)^2} = \pm \sqrt{7.93} = \pm 2.82$$

$$\sigma_{y'} = \pm \sqrt{4.39} = \pm \sqrt{(-0.323)^2} = \pm \sqrt{4.28} = \pm 2.07$$

$$\sigma_{x'} = \pm \sqrt{4.39} = \pm \sqrt{(-0.646)^2} = \pm \sqrt{4.79} = \pm 2.07$$

$$r = \frac{5.0}{(2.82)(2.07)} = \frac{5.83}{5.83} = 0.821$$

The computation of $\frac{\Sigma x'y'}{N}$ can be done in several ways but probably the best is to write in each cell its value of $x'y'$ being careful of the sign. Then the frequency in each cell is multiplied by the value of $x'y'$ of the cell and the entire table summed and divided by the total number of frequencies. From Table IX

$$\frac{\Sigma x'y'}{N} = 5.0, m_x' = -.323, m_y' = -.646, \sigma_x' = 2.07, \sigma_y' = 2.82 = r = .82$$

r differs slightly due to the grouping.

In most problems it seldom seems necessary to determine with any great refinement the value of r . It is a figure subject to much variation due to slight variations in the frequency distributions of the variables involved. It

TABLE X
CANCER AND DIABETES DEATH RATES

CANCER			Total
	Below Mean	Above Mean	
	Above Mean	Below Mean	
DIABETES	3	17	20
	11	3	14
	14	20	34

is always less well determined than the standard deviations of each distribution. r is only one constant of a frequency surface and by no means adequately defines the surface except when it is a normal frequency surface. Too great an exactness in the determination of r may be arithmetically desired and theoretically impressive but practically of no value. It is therefore desirable to have at hand some formula that is simple in computation and at the same time gives good values of r . Such a formula is $r = \sin 90^\circ \left(\frac{m - n}{m_x + n} \right)$

when the frequency distributions of both variables are normal, where m is the sum of a pair of diagonal cells in a fourfold table and n is the sum of the other pair. In order to apply this formula it is only necessary to count the number of observations that are greater than the means of both variables, those that are greater than the mean of one variable, those that are greater than the mean of the other variable, and the total number of observations. In other words, the observations are grouped to a fourfold table making the divisions at the means. When the distributions are not normal an approxi-

mate value of r might be obtained by making the divisions at the medians since such a procedure does much to normalize a table. In Table X the cancer and diabetes death rates have been grouped to a fourfold table. $m = 28$, $n = 6$
 $r = \sin 90^\circ \left(\frac{22}{34} \right) = \sin 58.2^\circ$. Looking up 58.2° in a table of sines the value of r is found to be .85, which agrees very well with the true value.

In drawing inferences from r it is necessary to avoid the pitfall so often encountered, which is that correlation means causation. It may often mean causation, but the causation must be established upon other evidence than the coefficient itself. It is possible that one variable stands in causal relation to the other or that both are influenced by a common cause or both affected by a proportion of causes influencing each or both affected by entirely independent causes operating at the same time and in such a manner that the correlation is produced. The correlation coefficient is nevertheless a useful constant. The observation and numerical measurement of relationships are among the first steps taken in the search for causes of phenomena, but it is always necessary to keep in mind that in any problem the search is not ended with the computation of a coefficient.

Sampling Errors.—If a sample of N observations be selected from a large universe, say, the heights of 100 men chosen at random from the population of a large city, the mean of that sample will have a certain value. If a second sample of 100 men be taken from the same population the mean of this sample will very probably not have exactly the same value as that of the first sample. If a third, fourth and many more samples be taken and their means computed it would be found that the means themselves form a frequency distribution the mean of which distribution would be the best value for the mean height of all men in that city and the standard deviation of which would represent the amount of scattering of the means of the samples from the best mean height of the city. It has been found by experience, mainly by coin-tossing experiments, that the scattering of the means of different samples from the true mean of the universe is a function of the true standard deviation of the universe and also of the total number of observations of the sample. It is virtually impossible in most cases to measure the whole universe; and indeed one must often be content with one or two samples of the universe. From a few samples it becomes necessary to draw inferences regarding the whole universe. In order to judge whether the mean of one sample is near the true mean of the universe it is necessary to know the true mean and the true standard deviation of the universe. These are never known and therefore it is assumed that the mean and the standard deviation calculated from the sample are the true mean and standard deviation of the universe. With this assumption it is possible to estimate the amount of scattering to which the mean of the sample is subject. Then immediately it is customary to turn about and estimate the chances that the true mean can differ from the mean of the sample by as much as any given amount. Here the assumption is made that the means of the samples are distributed about

the true mean of the universe according to the normal frequency curve, which assumption may or may not be true. The true mean and the true standard deviation of the universe as well as the type of frequency distribution of the means of the samples are all unknown. If one has hard luck enough to pick a biased sample unknowingly, all estimates as to the chances of the true mean differing from the mean of the sample by any given amount would be wrong. Not having the true mean to check up with, one gradually places more and more confidence in the mean of a sample. It can happen that the mean of a sample will differ from the true mean of the universe by a large amount relative to its sampling error, due to the facts that: first, although the sample is a random one, yet by chance alone a random sample can differ by that amount; and secondly, that the standard deviation of the sample which is assumed to be the standard deviation of the universe probably is smaller than the true standard deviation, the result leading to a small sampling error; and thirdly, one can never be sure that the sample is a random one. Probably all errors of sampling are further underestimated by neglecting terms in their derivation which are assumed to be small. Certainly in cases where the derivation of the formula is carefully analyzed, as for instance, the sampling error formula for the correlation coefficient, it is found that the commonly used formula underestimates the sampling error. It is therefore better to avoid a too confident reliance upon the inferences to which the use of sampling errors leads. On the other hand, it is disastrous to ignore them. A conservative use of them is mainly to warn against basing conclusions upon a too small number of observations.

What has been said above with reference to the mean of a sample applies as well to every statistical constant computed from the sample.

The sampling errors are of value in determining the significance of the differences between constants of two different samples as well as in making inferences regarding the universe at large.

An evaluation of the sampling errors of all statistical constants has been made and they are all functions of the total number of observations in the sample. When N is large, sampling errors are small and inversely when N is small, errors are large. Many of the sampling error formulæ include the assumption of a normal frequency distribution of the sample; and when the sample is not so distributed they must be interpreted with the utmost caution. There is plenty of chance to go wrong in making statistical inferences and a small chance of being right. With never-ending caution and suspicion the statistical problem should be approached and naturally with such an attitude it is never solved. It merely approaches solution.

Sampling Error of Median:

$$\sigma_M = \pm \frac{\sqrt{N}}{2} \text{ observations. (Any distribution.)}$$

From Table I $N = 34$ $\frac{\sqrt{N}}{2} = \frac{5.8}{2} = \pm 2.9$ observations. The median is

the $.50 N + \frac{1}{2}$ observation. The median $+ \sigma_M = .50 N + \frac{1}{2} + 2.9 = 20.4$ observation. The 20th observation is 78.1 and the 21st is 78.9. The 20.4 observation lies .4 of the distance between 78.1 and 78.9 above 78.1 which is 78.4. The median $- \sigma_M = .50 N + \frac{1}{2} - 2.9 = 14.6$. The 14.6 observation falls at 73.7. The median can vary between 73.7 and 78.4 to the extent of once its standard deviation. The median is 76.9. It varies upward by 1.5 and downward by 3.2. It is always customary to express σ_M as one figure and 3.2 and 1.5 would be averaged and σ_M would then equal 2.4. The median is then written $M = 76.9 \pm 2.4$.

If it is assumed that the true median lies at 74.2 which is the value of the mean, then the difference between the observed median and the assumed true value is 2.7 which is a little more than once σ_M . Assuming that all differences between theoretical and observed values are distributed according to the normal frequency curve, then the difference is about once sigma distant from the mean difference which is zero, and the chances of getting a deviation as great or greater than once sigma can be read from a table of the integral of the normal frequency curve.³ A deviation as great or greater than one sigma can occur about 16 times in 100 in one direction and about 32 times in 100 in either one or the other direction. This is not a very small chance and therefore one is not apt to regard the difference as so significant that it could not occur by chance or random sampling.

Sampling Error of Mean:

$$\sigma_m = \pm \frac{\sigma \text{ (Any distribution)}}{\sqrt{N}} \text{ when } N > 25.$$

In Table I

$$\sigma = 14.0 \quad \frac{\sigma}{\sqrt{N}} = \frac{14.0}{5.8} = 2.4$$

$$m = 74.2 \pm 2.4$$

In a normal distribution σ_m is always less than σ_M and therefore the mean is the better determined centering constant. When, however, the distribution departs markedly from normal this may not be the rule; the median may be the better determined constant.

Sampling Error of A.D.:

$$\sigma_{A.D.} = \pm .75 \dots \frac{A.D.}{\sqrt{N}} \text{ (Normal distribution)}$$

In the 34 states the average deviation of the cancer death rates is 11.55.

$$\sigma_{A.D.} = \pm .75 \left(\frac{11.55}{5.8} \right) = \pm 1.49$$

$$A.D. = 11.55 \pm 1.5$$

Sampling Error of Q.:

$$\sigma_Q = \pm 1.16 \dots \frac{Q}{\sqrt{N}} \quad Q = 12.2 \pm 2.4$$

³ Pearl, Kelley and Whipple.

Sampling Error of σ :

$$\sigma_{\sigma} = \frac{\sigma}{\sqrt{2N}} \text{ (Normal distribution)}$$

In Table I

$$\sigma_{\sigma} = \pm \frac{14.0}{8.2} = \pm 1.71$$

$$\sigma = 14.0 \pm 1.7$$

In a normal distribution $\sigma = 1.25$ A.D.; 1.25 A.D. = 14.44.

The difference between the sigma calculated and the sigma estimated from A.D. is .49, which is less than $\frac{1}{3}$ of σ_{σ} and is not significant. The distribution of the cancer death rates is practically normal when judged by the difference of σ and 1.25 A.D., as well as the difference of the mean and the median.

Sampling Error of the Trend (d):

$$\sigma_d = \frac{\sigma_r}{\sqrt{N}} \sqrt{\frac{12}{N^2 - 1}}$$

From Table IV the trend of the logarithms of the diphtheria death rates is $-.017$. $\sigma_r = \pm .076$; $\sqrt{N} = 5$; $\sqrt{\frac{12}{N^2 - 1}} = \pm .139$; $\sigma_d = \pm \frac{.076}{5}$ ($.139$) = $\pm .002$.

$$d = -.017 \pm .002.$$

The trend differs from zero by .017, which is over 8 times its sigma and therefore is significant.

Sampling Error of r :

$$\sigma_r = \pm \frac{1 - r^2}{\sqrt{N}}$$

From Table VIII

$$\sigma_r = \pm \frac{1 - (.86)^2}{5.8} = \pm .045$$

$$r = .86 \pm .045$$

r differs from zero by .86, which is nearly 20 times its sigma and is highly significant according to the above formula. R. A. Fisher has shown that the distribution of r is only normal for values near zero and that the above formula underestimates σ_r , thereby exaggerating the significance of r . For higher values of r either plus or minus the distribution of r becomes markedly skew. r can vary toward smaller values with a great deal more freedom than it can vary toward larger values. Fisher has found a function which practically normalizes r and with it is able to get a truer estimate of the sampling error of r . The principal use of the sampling error of r is to judge whether r differs from zero significantly. Since the sampling error of r is a function of N and r and since with σ_r it is possible to estimate the significance in terms of chances or probabilities of the difference between any value of r and zero, it is then possible to construct a table of r and N for various levels of significance. Fisher has done this. Figure 141 shows

the value of r and N necessary so that r differs from zero as much as or less than the chance of 2 times in 100.

A study of the graph will lead one to see that low values of r are only significant when N is large and will also lead one to the conclusion that the two exact computation of r , as for example, to four decimal places, is work for a computing machine but practically of little value; and also that rough estimations of r are accurate enough, saving at the same time a great amount of labor.

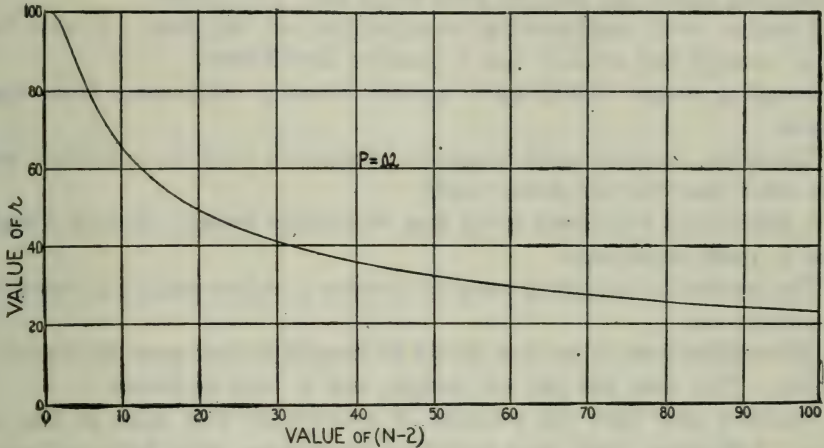


FIG. 140.—SHOWING THE RELATION BETWEEN THE VALUES OF r AND $(N-2)$ WHICH EXISTS WHEN r DIFFERS FROM ZERO BY CHANCE ALONE AS OFTEN AS TWICE IN ONE HUNDRED SAMPLES.

Sampling Error of a Sum or Difference of Two Uncorrelated Variables:

$$\sigma_{diff.} = \pm \sqrt{\sigma_1^2 + \sigma_2^2}$$

The standard deviation of a sum or a difference of two variables is equal to the square root of the sum of the squares of the standard deviation of the two variables. This formula is frequently used in comparing similar constants of two different samples, as, for instance, the means of two samples. They may differ and it is desirable to know if the difference is significant. The difference has a standard deviation which is expressed by the above formula. In the case of the means of two samples it would be

$$\sigma_{diff.} = \sigma_{(m_1 - m_2)} = \pm \sqrt{\sigma_{m_1}^2 + \sigma_{m_2}^2}$$

Computation.—For most statistical problems in the field of vital statistics computation can be satisfactorily carried out with the aid of a slide rule, a copy of E. V. Huntington's *Four Place Tables* and a table of the integral of the normal frequency curve as found in Pearl or Kelly. The use of a printing adding machine is helpful at times. A handy supply of the various plotting papers will be found to be invaluable. With the above aids to computation accuracy to three figures can be obtained. Computation can often

be arranged so that greater precision is unnecessary. There is little use in computing to a precision greater than the sampling error of the data. Furthermore, the sampling error is not the total error and in many phenomena there is without doubt a large error of measurement superimposed upon the sampling error. With due regard to the error of measurement and the sampling error one will not compute to unnecessary precision and also will not place too much confidence in the result of the computation.

Warnings.—Examine the reliability of the data; the source; the manner of obtaining them; the universe from which they were drawn.

Examine every mathematical manipulation of the data. A rate is a simple concept but actually has a complex significance.

Sampling errors should warn against drawing conclusions from small samples.

Correlation does not mean causation. Causation must be established upon facts other than the correlation itself.

A statistically significant result may be entirely wrong. Refined common sense is more dependable.

The practice of combining samples in order to reduce sampling errors is a dangerous one.

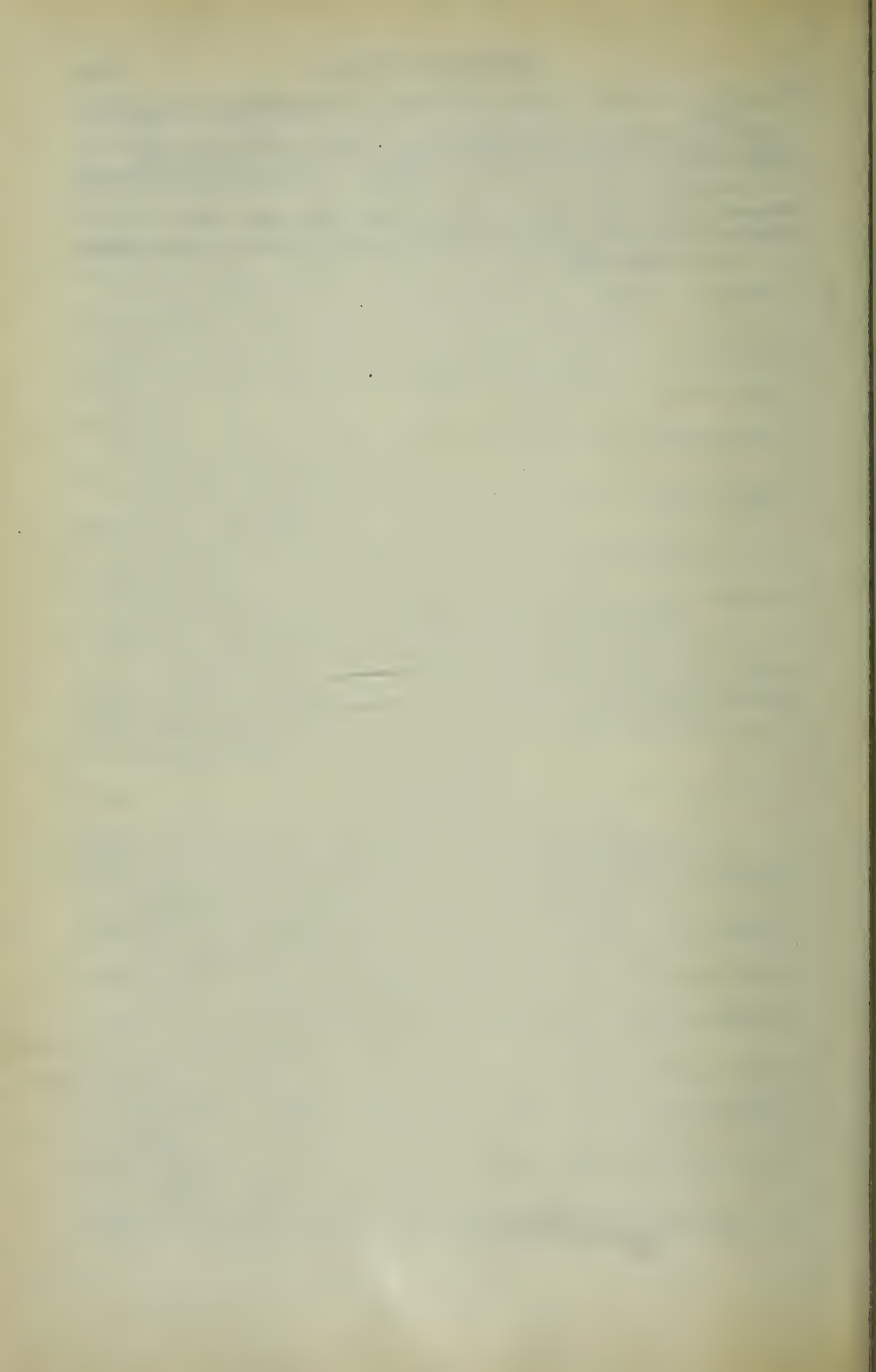
Information from all sources should be brought to bear upon the statistical problem. The data are but one sample and it may be biased.

Statistics only show the presence of occurrences with more or less certainty. Inferences from them should be based upon other facts and occurrences as well. An inference from one sample has little chance of being right. Many samples should be selected. More can be learned from a series of small samples than from one large sample.

REFERENCES

- BAILEY, W. B., and CUMMINGS, J. *Statistics*. McClurg, Boston, 1917.
- BOWLEY, A. L. *Elements of Statistics*. P. S. King, London, 1920.
- DAVENPORT, CHAS. B. *Statistical Methods, with Special Reference to Biological Variations*. Wiley & Sons, New York, 1904, 2nd Ed.
- ELDERTON, W. P., and ELDERTON, E. M. *Primer of Statistics*. The Macmillan Co., New York, 1912.
- FALK, I. *Principles of Vital Statistics*. Saunders, Philadelphia and London, 1923.
- FISHER, ARNE. *The Mathematical Theory of Probabilities*. The Macmillan Co., New York, 1922.
- FISHER, R. A. *Statistical Methods for Research Workers*. Oliver & Boyd, Edinburgh, 1925.
- GOODWIN, H. W. *Precision of Measurements*. Mass. Inst. of Technology Press, 1909.
- HUNTINGTON, E. V. *Four Place Tables*. Harvard Coöperative Society, Cambridge.
- JONES, D. C. *A First Course in Statistics*. Bell & Sons, London, 1921.
- KELLEY, T. L. *Statistical Methods*. The Macmillan Co., New York, 1924.

- KING, W. I. *Elements of Statistical Method*. The Macmillan Co., New York, 1918.
- NEWSHOLME, A. *Elements of Vital Statistics*. Appleton, New York, 1924.
- PEARL, RAYMOND. *Medical Biometry and Statistics*. Saunders Co., Philadelphia, 1923.
- WHIPPLE, G. C. *Vital Statistics*. Wiley & Sons, New York, 1923.
- YULE, G. UDN. *An Introduction to the Theory of Statistics*. Charles Griffin & Co., London, 1922.



SECTION XIV

INDUSTRIAL HYGIENE AND DISEASES OF OCCUPATION

Industrial hygiene is one of the most important topics in preventive medicine and hygiene, as it deals with the health, the welfare, and the human rights of the vast majority of the adult population. Industrial hygiene is a subject in which the medical, economic, and sociologic aspects are closely interwoven, and it requires a broad grasp, as well as an intimate knowledge of the conditions to avoid the dangers and correct the injustices to which people who work are subjected. The questions of industrial hygiene strike at the very root of our social system; they deal with the relation of capital and labor, and the relation of man to his fellow men. The employee must largely accept the conditions as he finds them and is frequently denied many advantages, even necessities. As the power of the employee is limited, he needs the protection of the state to correct the unreasonable demands which capital has ever exacted of labor. Human rights for work people have been wrested from unwilling hands. In recent years, however, there has been a brightening trend towards voluntary profit-sharing and coöperation in industry. The situation has also been improved somewhat through organized labor, which has exerted a healthful influence in limiting the avarice of the employer, in shortening the hours of work, in obtaining a better wage, in improving sanitary conditions, and in exacting a modicum of human consideration. Legislation in the form of workmen's compensation acts also has been a beneficent factor.

Modern conditions have brought entirely new problems into industrial hygiene. These have come about largely through the development of new industries and the invention of new processes, through the ever-increasing size of factories, through improved and changed methods of transportation, and through specialization and crowding in cities and work places, through artificial light, through changing relations between capital and labor, and the intensive and unrelenting pressure of the times. Some of the conditions which oppress the workmen are brought about by the greed of capital and disregard of the human machine; but indifference, carelessness, and ignorance of the workman himself are responsible for many avoidable accidents and preventable maladies. In Eastman's study of work accidents in Pittsburgh it appeared that, out of 410 fatal accidents, the victim or his fellow workers were responsible in 188 cases and the employer in 147 cases. Despite the improvements in labor-saving devices in the production of wealth, the human machine will ever remain the most vital and indispensable as well as the most delicate and sensitive. Both from the standpoint of humanity

and the standpoint of economy it deserves greater care and consideration than any other mechanism engaged in production.

INDUSTRIAL DISEASES

The true diseases of occupation are those that result from or are incident to certain industries, such as liability to lead poisoning in the manufacture of white lead; of phossy jaw in the manufacture of matches made with white phosphorus; of caisson disease in divers and those who work in compressed air; special affections are sometimes caused by exposure to high temperatures; there are extra hazards to life and limb in railroading, mining, and among those who work with explosives; there is a particular danger to those who are compelled to work in a dusty atmosphere, more so if the dust is of an irritating or poisonous nature; and there is danger to those who are compelled to breathe poisonous fumes such as carbon monoxid, hydrogen sulphid or mercury. Poisons are especially hazardous in the form of gases, vapors and dust. Important as they are, industrial poisonings form only a fraction of the whole problem. In addition, there are many forms of neuroses due to certain kinds of work; and finally, infections such as anthrax, glanders, and hookworm disease. These varied instances represent the real diseases of occupation.

The reporting of occupational diseases is a public health measure, and of first importance for their control.¹

Industrial hygiene, on the other hand, includes many conditions not specifically inherent in industry, to which the workman is often subjected which seriously influence health; such as poor ventilation, lack of cleanliness, overcrowding, excessive hours, improper light, fatigue, and a hundred and one conditions which affect the health and the efficiency of the workman. The prevention of the communicable infections among work people is based upon the same principles and practice as for the civilian population. Such diseases are a reflection of those commonly prevalent in the community. A study² of the annual frequency of certain diseases causing disability for eight days or longer among a group of 113,000 wage earners of both sexes in different industries showed that 29.9 per thousand were due to diseases of the respiratory system, 13.0 per thousand to diseases of the digestive sys-

¹ The Massachusetts legislature of 1904 took the first step in this country to obtain definite scientific data on the subject of the occupational diseases. The State Board of Health of Massachusetts made an investigation into the sanitary condition of factories, workshops, and mercantile establishments, and published its first report on the subject in 1907. Since then several states have taken up the subject, also the U. S. Dept. of Labor and the Amer. Assn. for Labor Legislation. The Hospital for Industrial Diseases at Milan, opened March, 1910, is the only institution in the world devoted specifically to the investigation and care of such diseases. The first American Congress on Industrial Diseases met in June, 1910.

The earliest successful attempts in England to enforce sanitation and to secure healthy conditions of work were introduced by the Act of 1833 involving the appointment of paid factory inspectors, who antedated medical officers of health and sanitary inspectors.

² *U. S. Pub. Health Rep.*, 1926, 41: 113.

tem, and 6.4 per thousand to diseases of the nervous system. Emmons,³ in a study of absenteeism among 4,000 employees in a Boston store, found that diseases of the nervous system were more prevalent than gastro-intestinal disturbances. A memorial on industrial injuries prepared by a committee of inspectors, appointed by the President of the Association for Labor Legislation, states that there are probably not less than 13,000,000 cases of sickness each year among those engaged in industrial employments. The money lost each year (for those who find dollars more expressive than health, efficiency and life) is conservatively calculated at nearly three-fourths of a billion dollars. Much of this painful incapacity for work and consequent economic loss can be prevented.

Industrial hygiene runs the whole gamut of hygiene and sanitation. It deals with the problems of dust, cleanliness, light, ventilation, washrooms, emergency clinics, physical examinations, safety devices, temperature and humidity, and the elimination of noise and nuisances. It must take into account the problems of diet, exercise, and health habits at home as well as at work; it must also provide medical, dental and nursing services. Special attention should be paid to defects of teeth, tonsils, adenoids, eyes, ears, shoes, etc.; also to rest and recreation, absenteeism, mental tests to determine fitness for the job, education and morale. One man may make or mar the morale of a group, hence disposition is also an important factor.

The *statistics of morbidity and mortality* in relation to diseases of occupation need careful scrutiny, especially when used for comparison. The factors which enter into such statistics are so numerous and the conditions so variable that misleading conclusions are common. The workmen come and go, they vary very much in physical vigor to start with, are of all ages, both sexes, many nationalities, and are greatly influenced by home conditions and by the character of their recreation. Some industries, while not in themselves particularly hazardous, are rendered so through intemperance or dissipation. On the other hand, there is a relation between low wages and a high morbidity rate in a given industry. Low wages mean poor housing, insufficient clothing, poor food, unhealthful recreation, increased temptation to alcoholism, and an undue proportion of youthful workers, of women, and of men more or less handicapped by poor physique or bad habits. The statistician therefore must be careful to take all factors into account that bear upon the subject. Some industries are blamed for conditions affecting health that may be due to the insanitary home conditions and bad habits of the individual.

In recording the nature of a man's work it is not sufficient simply to state that he is a laborer, mechanic, machinist, mill operator, and the like. Such information is frequently of no more value to the student of the diseases of occupation than the name of the person himself. A "machinist" may set hair springs in watches or repair automobiles. If the person is a blacksmith

³ *Health Control in Mercantile Life*, Harper & Bros., N. Y., 1926.

or works with heavy metals it is plain that he works under a severe physical strain. If he is a sailor upon a sailing ship we know that he is exposed to rough weather and unusually severe conditions, whereas if he is a sailor upon a modern passenger steamship the conditions of his work may be no more severe than those of the janitors and charmen in a large office building. If he is in finance we may be sure that he is subject to severe nervous strain. It is therefore not sufficient simply to give the name of the trade, but detailed inquiry should be made into the precise nature of the individual's work and the particular conditions under which he works.

Most of the investigation of industrial poisons in the United States has been made within very recent years, and the field has not yet been nearly covered. Careful studies have been made of two poisons, white phosphorus⁴ and manganese,⁵ most of the more important lead industries have been studied,⁶ and there are some excellent surveys of the brass industry⁷ and of the various occupations in which mercury⁸ or its compounds are used.⁹ There is need for more study of several poisons which are quite extensively employed in our industries, such as carbon monoxid; white arsenic and arseniuretted hydrogen gas; vanadium; selenium; carbon disulphid; sulphurated hydrogen; benzene; toluene, and its many derivatives, of which anilin is the most important; methyl alcohol; amyl acetate; carbon tetrachlorid; acetylene tetrachlorid, or tetrachlorethane; aldehyds, acetones and cyanogen—the list is constantly growing.

Conditions in American industry vary much more widely in different plants than they do in the older countries, and while our best factories are equal to the best in Europe, or occasionally are even better, the worst fall below the standard required by law in Europe. A great percentage of work is still done in small factories where conditions may be very bad. There is on the whole, in America, more recklessness in the handling of trade poisons and the result is shown in all the statistics of industrial poisoning that have been collected. Thus, in the making of storage batteries, a dangerous lead trade, the rate of poisoning in the five largest factories in the United States in 1914 was 17.9 per cent, while Great Britain's rate for 1912 was 2 per cent, and the largest factory in Germany had only 0.97 per cent.

Good industrial conditions mean money in the pockets of the employer. Thus, Elliot Washburn reports the instance of twenty-eight emery grinders in Massachusetts working in a badly lighted and poorly ventilated basement, who increased their efficiency 30 per cent on being transferred to a well-lighted and ventilated workroom.

An industry may be a nuisance or disturbance to the community as well

⁴J. M. Andrews, *Bull.* 86, U. S. Bureau of Labor.

⁵D. L. Edsall, C. K. Drinker, *J. Ind. Hyg.*, 1919, 1: 183.

⁶*Bull.* 95, U. S. Bureau of Labor, and *Bulls.* 104, 141, 165, 209, U. S. Bureau of Labor Statistics.

⁷E. R. Hayhurst, *Am. J. M. Sc.*, 1913, 145: 723.

⁸Mercury Poisoning in the Industries of New York City and Vicinity. Nat. Civic Federation Women's Welfare Dept., 1913.

⁹L. I. Harris, Dept. of Health, City of New York, Mono. Series No. 12, 1915.

as to those engaged in its various processes. Thus, the noise of a tack factory or boiler shop, the smells from glue or fertilizing factories, or the fumes from smelting or chemical works, or the smoke from chimneys or locomotives, wastes from tanneries, paper mills, and mines do not come directly in the chapter of industrial hygiene, however closely related.

The Physician in Industry.—The physician in industry is one who applies the principles of modern medicine and surgery to the industrial worker, sick or well, supplementing the remedial agencies of medicine by the sound application of hygiene, sanitation and accident prevention; and who, in addition, has an adequate and coöperative appreciation of the social, economic and administrative problems and responsibilities of industry in its relation to society.¹⁰

Industrial medicine and surgery are nothing more or less than good medicine and surgery in industry. Only the best service is justified. A well-organized health service constitutes a health center and is a form of group practice. Medical service in industry presents a special opportunity to advance public health.

SOME FUNDAMENTAL CONSIDERATIONS IN PREVENTION

In order to improve the hygienic conditions under which people work, and in order to prevent the diseases of occupation, there are five fundamental essentials: (1) investigation; (2) laws; (3) inspection; (4) penalties; (5) education. It is self-evident that before anything can be accomplished a careful study must be made of the facts. These investigations must include not only scientific studies, but also economic and sociological factors. Every large industrial center should have a clinic for the study and care of industrial diseases. The subject should be included in the curriculum of medical schools. Suitable laws are necessary, for it has been found in practice that the conditions cannot be corrected by an appeal to voluntary reform. To be effective the laws must provide ample ways and means for their energetic enforcement. A systematic factory inspection is necessary in order not only to protect work people against the preventable diseases of occupation and to correct sanitary defects, but also to enforce the laws concerning hours of occupation, child-labor laws, and related subjects. These laws have little force unless they provide a penalty against both the employer and the employees. Either party to the contract should be held legally responsible in case of violation. Finally, education directed to the employer, the employee, and also to the public at large is necessary to obtain the laws and raise the standards.

Hours of Work.—No general rule can be laid down for the hours of work, which may vary with the character of the employment. Thus, there are rational limitations to the hours of active work of a smith or glass-blower, a worker in a caisson or mine, a locomotive engineer; and of others whose occupations necessitate great muscular effort or intensive concentra-

¹⁰ Definition adopted by the Conference Board of Physicians in Industry.

tion, or exposure to unnatural conditions. Long hours increase exposure to injurious influences and poisons and lessen the chance of recovery and elimination. Formerly, men worked at the quieter occupations all the time not given to sleep; now the day is better divided into eight hours of work, eight hours of "re-creation," and eight hours of sleep. Hygienically, it is important to have one full day's rest each week. It cannot be maintained from the medical side that working longer than eight hours a day is harmful to health, but it is held that no employer has the right to utilize the greater part of a man's day and thus deprive him of the leisure to which he, as a human being, is entitled. Since his whole nature has to be developed, time must be given for the intellectual, moral, and physical welfare of man, which cannot take place if the hours of employment are too long, the work too hard, or of a grinding nature. The hours of work depend somewhat upon the physical exertion required and also upon the nervous tension. The Saturday half-holidays, especially during the heated term, a vacation period, and a tendency to increase the number of holidays are all signs of social improvement which make for health and happiness.

Fatigue.—It pays to give employees a rest at stated intervals and to guard the conditions surrounding workers, so that they are neither molested nor interrupted, that the light and other factors are agreeable, and the sanitary surroundings good. Work and rest must be judiciously alternated. Efficiency ceases when fatigue begins. A tired brain, tired nerves, and tired muscles are a menace to the workman himself, as well as to others. Accidents are especially prone to happen to workmen under these conditions. Thus most accidents in factories happen as the day wears on. The effect of fatigue on the occurrence of accidents is graphically shown by French and Belgian statistics. The number of accidents increases progressively during the morning hours, drops after the noon intermission, and then rises from hour to hour until the end of the working day, affording a practical illustration of Helmholtz's experiment in attention fatigue. Fatigue is not only dangerous to the workman himself, but sometimes to others; thus the overwrought and tired-out train dispatcher may send trains into collision. Further, fatigue of certain nerves and muscles may result in definite symptoms, such as writers' cramp, or more general manifestations, such as neurasthenia or nervous prostration. Typists, telegraph operators, and others suffer from these occupational neuroses.

Spaeth¹¹ draws attention to the difference between normal fatigue which is harmless and cumulative fatigue which is dangerous and may be associated with serious nervous disorders—industrial psychoneuroses. The one merges into the other, and therefore the reduction of normal fatigue is the first logical step in a prophylactic attack.

Fatigue is also believed to predispose to certain infections, such as cerebrospinal fever and perhaps pneumonia and other respiratory diseases. The

¹¹ *J. Ind. Hyg.*, 1920, 1: 435.

experience of the World War clearly showed that all attempts to make a soldier too rapidly, through fatiguing drills, invite disaster.

Next to fatigue, nervous tension and worry are very wearing, and when combined become especially harmful. Diabetes prevails among engine drivers to a considerable extent. Worry, hurry, and a high nervous tension are recognized as a frequent predisposing cause of ill health or breakdown in all walks of life, including the so-called higher professions.

The "Monday Effect."—It has long been known that workers are less efficient on Monday and following holidays than on other days of the week. Efficiency experts well know that the smallest output of the week occurs on Monday.

The usual explanation of this has been misuse of the week-end leisure and of the week's wages—as excessive eating, excessive drinking, late nights and insufficient sleep, or dissipation of other kinds. But it is now apparent that these are not the real reasons for inefficiency following a rest. A. F. Stanley Kent¹² has shown that the week-end rest affects both fatigue-production and recovery from fatigue on the one hand, and output on the other. There is not only a feeling of lassitude and a disinclination to work, but also an actual diminished efficiency—a common phenomenon of habit behavior. In other words, upon return to work after a rest, the workman is out of practice, has forgotten, and finds a certain amount of loss of coördination rather than actual fatigue. He must relearn before the accustomed efficiency can be regained. This same phenomenon is observed in the familiar behavior of a muscle which after a rest shows a progressive improvement as a result of activity—the contractions increasing in a "stair-case" effect.

Workers in nitroglycerin, naphtha and ether always feel the effects of these substances more on Monday or following a holiday. The reason for this is loss of tolerance; in fact, workers in nitroglycerin carry some of this substance in their hatband if they go on a week's vacation in order to avoid the nitroglycerin headache upon reëxposure.

Minors.—The first acts regulating the employment of minors in factories were designed to protect children. The Factory Act of 1802, in England, was the first labor legislation and was promulgated chiefly for the protection of apprenticed children. Similar acts were passed in France and Germany early in the nineteenth century. In 1886, New York passed a factory act prohibiting children under thirteen to work in factories. Since then the minimum was raised to fourteen years in 45 states, and in seven of these the limit is higher. The Federal Child Labor Law passed in 1918 was declared unconstitutional by the United States Supreme Court in 1922. Prior to 1918, the statutes of nineteen states prohibited the employment of children under sixteen in any mine or quarry, while Texas placed it at seventeen years and Arizona and Wisconsin at eighteen years. In 1836 Massachusetts passed an act requiring children between twelve and fifteen years of

¹² *J. Physiol.*, 55: 1915-16.

age employed in factories to attend schools three months in each year. Compulsory school attendance in most states is now placed at sixteen years.

The injustice to the child and the consequence upon its health and development of subjecting it to the monotony and grind of factory life are too evident to need emphasis. Recently it has been claimed that in certain districts, as, for example, the mill district of our southland, the children are better off in a good textile mill of modern construction than they are living under the insanitary conditions of their homes. It would be just as logical to state that they would improve in health if removed to a sanitary prison or almshouse. The child of to-day is the citizen of to-morrow and his health and development are the most important assets of the state.

In Massachusetts minors under eighteen years of age are excluded from the following occupations:

1. Processes involving exposure to poisonous dusts or substances, such as the manufacture of lead, lead pipe, plumbers' supplies; electrotyping, handling white lead or lead monoxid (litharge) in rubber factories; lead-paint grinding; lead working in the manufacture of storage batteries; file cutting by hand; typesetting; cleaning or handling of type in printing offices; glazing in pottery establishments.

2. Processes involving exposure to irritating dusts, as graphite in the manufacture of stove polish; bronzing in lithographing business; cutlery grinding and other grinding; polishing on emery or buffing wheels; cutting, grinding and preparing pearl shell; talc dusting in rubber works; sorting, cutting or grinding rags; filing grooves in steel rolls by hand.

3. Processes involving exposure to poisonous gases and fumes, as exposure to naphtha in the manufacture of rubber goods, in japanned or patent leather; exposure to fumes or gases from lead processes; and the spraying of amyl acetate on pictures.

4. Processes involving exposure to irritating gases and fumes, such as gassing in textile factories; singeing in print works; bleaching and dyeing works, and dipping metal in acid solutions.

5. Processes involving exposure to extremes of heat and other conditions which promote susceptibility to disease, as melting or annealing glass.

Great Britain, Denmark, France, Norway, Sweden and Switzerland have established eighteen as the minimum age for night work; few American states have placed it so high, but the Children's Bureau Conference, called in 1919 to set standards for employment of children in industry, advised the prohibition of night work under twenty-one years, because of the physical and moral dangers which night work for the young entails. American laws also fail to protect children adequately against the unhealthful processes in industry. Pennsylvania alone debarb young persons under twenty-one years from certain lead processes, while a limit of eighteen years is prescribed in Maryland, Massachusetts, New Jersey, Ohio and Wisconsin. This means that in forty-two states boys and girls of fourteen years may be employed in work exposing them to lead, mercury, arsenic, benzene, carbon disulphid,

carbon monoxid, etc. All of the European countries recognize the dangerous nature of such work for immature persons of both sexes, and have placed it under legislative control.

Working papers, required by at least 75 per cent of the cities in the United States at the present time, offer a material protection to children entering industry. The school medical examination may well be used to determine the eligibility of the child to enter industry rather than a special examination at the time the working papers are issued.

The minimal standards for children entering employment were formulated in a report¹³ of the Children's Bureau Conferences in 1919, which emphasized the fact that minimal ages for a variety of employments demand varying degrees of physical endurance and skill. Therefore, the examination for working papers should be based on the physiological and mental equipment of the child concerned, as much as on the number of years that have elapsed since his birth.

Women.—Women are physiologically not capable of doing the same work as men, especially during the period of maternity. Further, several days each month women are more or less incapacitated for most kinds of work on account of menstruation. Pregnant women should not work for several weeks before labor, and after labor not until the uterus has undergone involution, which is a matter of another month. It is in regard to the prohibition of work immediately before and immediately after childbirth, the prohibition of night work, and of employment in poisonous processes, that the laws of many of our states are inadequate. Connecticut, Massachusetts, New York and Vermont have passed legislation to cover the first point. Only nine states prohibit the night work of women, although as long ago as 1906 all the countries of Europe except Norway, Denmark, Roumania and Servia signed the "Berne International Convention" prohibiting it. Only Pennsylvania and New Jersey exclude women from the dangerous lead trades, and New York and Ohio have a curious law prohibiting women from working with emery and polishing powders.

Studies conducted by the United States Public Health Service¹⁴ showed that women had more than twice as many cases per thousand persons as men from ill-defined diseases and unknown causes of disability; neurasthenia, nervousness, and the like; diseases of the pharynx; appendicitis; diseases of the respiratory system other than bronchitis and pneumonia; non-venereal diseases of the genito-urinary system and annexa other than nephritis. They also had higher rates in practically all the other diseases except pneumonia, diseases of the veins, rheumatism and diseases of the bones and organs of locomotion.

Louis Brandeis successfully defended the constitutionality of the ten-hour law for women in Oregon. The brief submitted by this eminent jurist

¹³ "Standards of Child Welfare," Conference Ser. 1, Publication 60, Children's Bureau, U. S. Dept. Labor, 1919.

¹⁴ *U. S. Pub. Health Rep.*, 1926, 41: 113.

in a similar action before the Illinois Supreme Court¹⁵ should be read by those interested in this subject. The primary object of this brief is to show that the demands of public health require legal restrictions in the work of women because of the peculiar importance to the community of the health of mothers. The effect of overwork on the different organs is reviewed, also the effect of night work, of prolonged standing on the feet, of foot power machinery, and of the speeding up required by the "piece-work system." The general literature upon fatigue and overwork is reviewed.

The effect of overwork upon fecundity and upon infant mortality is impressive. Broggi states that of 172,365 Italian women between the ages of fifteen and fifty-four years who were employed in industrial occupations the average child-bearing coefficient was only about one-third of the general fertility of Italian women.

It is now a well-established fact that infant mortality is shockingly high among the babies of women who work in factories and mills. It has been shown in Germany and England that infant mortality increases progressively according to the increase in the proportion of women obliged to work outside of their homes, and this is true even if the mother's work results in higher standards of comfort in the home. The two classical demonstrations of this rule are the great Lancashire cotton famine and the Siege of Paris, during both of which crises there were loss of employment and great privation. In spite of the starvation and the increased general death rate, the infants' death rate fell in Paris actually to 40 per cent simply because the women, being out of work, were obliged themselves to nurse and care for their children. The infant mortality in industrial centers such as Fall River, Lowell, and Lawrence, in Massachusetts, which are mill towns, is twice as high as similar towns without many factories and with better economic standards.

It is plainly the duty of the nation not only to restrict the hours of work of women, but also to prohibit their employment in certain industries known to be particularly hazardous to them. The evidence presented by the British, especially by Oliver,¹⁶ on industrial lead poisoning, shows clearly the greater susceptibility of women to this poison, the severity of the form it takes in women, and the disastrous effect of maternal plumbism on the offspring (see page 1236). Although we have no statistics to prove it, there is every reason to believe that the same thing is true of other poisons used in industry. Protection should also be given to women against less striking evils in industry.

Saleswomen should be provided with seats in shops so as to avoid the ill effects of prolonged standing, they should have one or two days each month for rest during the menstrual period, and should be protected against undue strain and fatigue. While women's work may be regulated in the industries and the hours of employment may be limited by law, there can be no law

¹⁵ Louis D. Brandeis, assisted by Josephine Goldmark, *Brief and Argument for Appellants*, in the Supreme Court of the State of Illinois, December term, 1909.

¹⁶ *Dangerous Trades*. London, 1902, p. 296.

to regulate women's work in the household which is "never done." Men have still to learn the lesson that nervous breakdown and the results of fatigue are as harmful in women who overwork in the home as in those who work in shops and factories. The long hours and confining work of house servants sometimes lead to anemia and other troubles. Cooks are exposed to the effects of excessive heat and to sudden changes of temperature. Domestic "servants" as a class supply a large contingent of patients in hospitals and out-clinics. The long hours and insufficient sleeping accommodations, as well as the nature of the work, lead to ill health which may in part account for the disinclination of women to accept this kind of service.

Factory Inspection.—There is no longer doubt that factory inspection is necessary as a protection to the workman. An efficient system requires a good comprehensive basic law and a capable corps of inspectors. The inspectors should be thoroughly familiar with the law and with the processes of manufacture and also with the problems of preventive medicine. Factory inspectors should be capable of making recommendations outside of the strict regulations under which they operate so as to improve conditions and meet the needs of an ever-changing situation. Factory inspection really falls into two categories, one of which deals mainly with the medical side and the other with the legal and economic side. Both inspectors should take into account the social and humanitarian side. Some of the factors which should engage the attention of a factory inspector are: ventilation, dust, gases, vapors, odors, temperature, moisture, light, cleanliness, overcrowding, excessive heat, dampness, drinking water, children, women, washing facilities, water-closets, cloakrooms, receptacles for expectoration, defective sanitary arrangements, hours of work and rest, the age of the employees, their physical condition, etc. Hanson points out that medical men, through their training and attitude, make the best factory inspectors, for they alone are in a position to make the best use of facts, and learn something of the sanitary conditions of premises where men and women work, to study the possible injurious effects of certain processes, to inspect devices designed to protect the employees against injury or against dangerous fumes and dust, and to judge the effects on the health of operatives of such substances, as well as to detect the symptoms of certain poisons incident to such occupations, to detect and protect the employees and others from infectious diseases, to make physical examinations of minors, and to collect and make proper use of all facts and data, including morbidity and mortality statistics, pertaining to occupational hygiene. The medical inspector is also able to correlate the injurious influences in the factory, in the home, and in the habits of the individual.

INDUSTRIAL ACCIDENTS

The total number of these accidents is appalling. There are approximately 22,000 fatal industrial accidents a year in the United States, and 260,000 serious injuries. Other estimates place the total number killed in

industry at more than 35,000 a year. In 1920, 1,636,837 non-fatal accidents were reported, excluding agricultural labor and domestic service, which are not included in the workmen's compensation laws in most states, and steam-railway accidents which are not taken into account in many states. Up to 1914, trustworthy accident statistics did not exist for a single important industry.¹⁷ Since then the Interstate Commerce Commission, the Bureau of Mines and the Bureau of Labor Statistics, as well as various states, have undertaken to collect these facts, and during this time the whole system of workmen's compensation laws has developed. The figures are incomplete and not always comparable.

The most hazardous callings and their rates are given in the following table:

FATAL INDUSTRIAL ACCIDENTS
*Estimate for the United States for 1920 **

Occupation	Employees, Males	Fatalities	Rate per 1,000
Metal mining	145,262	468	3.22
Coal mining	765,000	2,317	3.03
Fisheries	160,000	480	3.00
Stevedoring, longshoremen	75,000	209	2.79
Electricians (light and power)	105,541	215	2.04
Coke ovens	28,741	53	1.84
Policemen, Firemen, Watchmen	250,000	440	1.76
Quarrying	75,505	125	1.66
Lumber industry	558,000	837	1.50
Navigation	255,900	341	1.33
Explosives	25,000	32	1.28
Ore smelting and dressing plants	61,708	77	1.25
Building and construction	1,575,000	1,969	1.25
Railways, steam	1,960,500	2,454	1.25
Railways, street and electric, interstate	294,829	311	1.05
Teamsters and draymen	720,000	720	1.00
Oil and gas wells	65,300	35	0.54
Telephone and telegraph (including linemen)	296,000	148	0.50
Iron and steel	372,714	183	0.49
Paper and pulp	80,000	34	0.42
Agricultural pursuits	11,600,000	4,060	0.35
Manufacturing (general)	7,900,000	1,975	0.25
All other occupied males	6,500,000	4,017	0.62
TOTAL			
All occupied males	33,870,000	21,500	0.063
All occupied females	8,560,000	642	0.075

* Statistician's Department, The Prudential Insurance Co. of America.

Steam railways have fairly complete records of industrial accidents since 1888. They show a large increase in injuries until about 1916, owing in part to expansion of operations and, in part, to better records than were possible in earlier years. The fatality rate for trainmen is declining. It dropped from 4.48 in 1917 to 1.79 in 1921. Yard brakemen have the highest fatality rate in three of these years, and road freight brakemen for the other two years. In frequency of non-fatal injury, yard brakemen exceed

¹⁷ Hoffman, *Bull.* 157, U. S. Bureau of Labor Statistics.

all other trainmen in each of the five years. The lowest casualty rates are for passenger conductors.

The iron and steel industry, as a whole, appears to show a definite decline in accident rates. The rates for blast furnaces are among the highest found in this industry, and these declined 52 per cent in the second of two five-year periods ending in 1921. Foundries were the only department of the industry to show an increase for both accident frequency and severity. It is possible that the low accident figures for 1921 were partly due to industrial depression, and that rising accident rates will accompany the revival. Accident rates for the erection of structural steel were higher than for any other industrial group found, and were approached only by those for logging and sawmill operations. The accident severity rate for the erection of structural steel in 1920 was 25.9 per thousand hours' exposure, while that for logging (Oregon) was 21.56.

The fatality rate for coal mines has irregularly but steadily declined. From 1907 to 1921 it dropped 34 per cent, and at the same time production per death rose 71 per cent. The serious danger in mines, as shown by the records, is falling material. This accounts for nearly half of all accidents, and there is practically no decline in fatalities from this cause from year to year. Considering the fatality rates from the standpoint of causes of accidents, it seems that the coal mining industry has been standing still in the matter of safety in recent years. Even so, coal mining appears to be less hazardous than operating trains.

Causes of Industrial Accidents.—Some workmen are liable to get hurt. Such men often show psychopathic inferiority and are a problem. This is largely an individual question of fitness for the job. In the United States Navy Yard at Washington, D. C., Bloedorn¹⁸ found that of 4,711 injuries received by 2,178 men, 1,263 were received by 188 men; in other words, a small number, 8.6 per cent, of the total number of men injured received over 26 per cent of the total number of injuries. Here is a field for the application of mental hygiene.

More accidents occur on Monday than on any other day. This may be due in part to the awkwardness of the Monday effect (page 1221). The novice is more apt to get hurt than the seasoned workman. By far the greater number of accidents occur at about 10:00 A.M. and 3:00 P.M. This cannot be due to fatigue, but is explained by the tendency to speed up at these times, and the danger of mischance because of haste. There is much greater liability to accidents on the part of children, even when employed in the less hazardous occupations. Among the obvious causes of accidents are sickness, fatigue, intemperance, poor vision, impaired hearing, ignorance, carelessness and lack of fitness for the job.

Special Injuries.—Some special injuries incident to work are: spinal curvature from faulty posture; flat feet or varicose veins from prolonged

¹⁸ *U. S. Nav. M. Bull.*, 1906, 10: 585.

standing, as in nurses and footmen; injuries to the eyes from metal splinters or stone fragments; impairment of vision from improper lighting, or eye strain, as in garment workers and gun pointers, or miners' nystagmus; injuries to the ears, as rupture of the tympanum from air pressure in caissons; labyrinthine disease leading to deafness in boiler-makers and gunners. Injuries to the skin are commonly caused by violence, but may result from excessive moisture, as hydrocystoma of laundresses; chilblains of cold storage workers; and ulcers caused by x-ray or radium—most of which are readily preventable.

Typhoid Fever an "Accident."—In the case of *Vennen v. New Dells Lumber Company*, 154 N. W. Rep. 640 (Oct. 26, 1915), the Supreme Court of Wisconsin decided that the death of an employee caused by typhoid fever, which was contracted by drinking impure water furnished by the employer, was the result of an "accident" under the terms of the Workmen's Compensation Law, and that the employer was liable. Similar decisions were rendered by the Supreme Court of Michigan, *Frankamp v. Fordney Hotel et al.*, 193 N. W. R. 204; by the Supreme Court of Illinois, *Christ v. Pacific Mutual Life Insurance Company*, 144 N. E. R. 161; by the Supreme Court of New York, Appellate Division, Third Department, *Carr v. Donner Steel Company et al.*, 201 N. Y. Supp. 604; by the Supreme Court of Illinois, *Barmore v. Robertson et al.*, 134 N. E. R. 815; the Appellate Court of Indiana, *Wasmuth-Endicott Co. v. Karst*, 133 N. E. 600. The laws and their interpretation vary in the different states. The Supreme Court of Ohio ruled that the term "injury" in the Workmen's Compensation Law does not include disease contracted in the course of employment, and accordingly holds that death from typhoid fever is not compensable.

Prevention.—Much can be done to cut down the toll of industrial accidents. The chief measures are responsibility of employer and employee, education, laws—especially workmen's compensation acts—safety devices, mental hygiene applied to industrial conditions, and investigation based upon careful collection and study of the facts.

Workmen's compensation acts have done wonders. A system of workmen's compensation, by which the victim of industrial accidents, except when caused by his own neglect, is entitled by right, and without legal proceedings, to a proper money equivalent for the injury received, is simple justice which has been long delayed in this country. Workmen's compensation laws have been in successful operation in all the principal European countries. Many of our larger corporations voluntarily and automatically compensate employees in case of accidents.

Workmen's compensation laws, the first of which was enacted in 1911, are now enforced in forty-two states and the Federal Government provides such protection for its own million civilian employees. The compensation provided runs from 50 per cent of the wages to 66 $\frac{2}{3}$ per cent, and in case of death, the plan generally adopted is to give the widow 35 per cent of

her deceased husband's wages with 10 per cent in addition for each child, the total never to exceed 66 $\frac{2}{3}$ per cent.¹⁹

A compensation law should cover all industries and not alone those that are hazardous; it should include disability from occupational poisons as well as from accidents; the payment should be adequate and certain. A good compensation law is one of the best preventive measures, for it has been found that employers soon discover it pays to safeguard the workmen.

Many accidents are preventable, in fact prevented in other countries. Fatalities are four times as common among our railroad employees as among those of England, and other accidents seven times as frequent. Coal mining was nearly as fatal in Belgium between 1830 and 1840 as it is in the United States to-day, but the Belgians have cut their death rate down to less than one-third of what it was.

The bulletins, posters and activities of the National Safety Council, 168 North Michigan Avenue, Chicago, are admirable and serve a useful purpose in educating and warning. The importance of mechanical guards to avoid accidents does not need emphasis. Mental hygiene, especially psychological examinations to fit the man for his job, to promote morale, and to keep the unfit out of positions that may endanger others, is a useful preventive measure, but little realized.

Accidents in General.—America has a black record of deaths by accident. It is estimated that there occurred 84,000 deaths from fatal accidents in the United States during 1923, and this number was 7,500 in excess of the year before. In England and Wales during 1922 the fatal accident rate was 321 per million of population, and in the United States, 698 per million! Our excess over the British rates amounts to 35,000 or 40,000 of our citizens each year—a city full.

The figures in the following table are significant:

TABLE SHOWING ESTIMATED NUMBER OF FATAL ACCIDENTS IN CONTINENTAL UNITED STATES FOR THE MORE IMPORTANT CAUSES *

CAUSE OF ACCIDENTAL DEATH	ESTIMATED DEATHS				
	1923	1922	1921	1920	1911
All fatal accidents	83,772	76,255	73,542	75,983	79,255
Accidental burns	7,304	6,992	6,362	8,088	7,214
Accidental drowning	6,529	6,992	7,872	6,066	8,806
Accidental falls	15,382	13,219	12,293	12,557	14,052
Traumatism by machines	2,324	2,185	1,941	2,660	1,967
Railroad accidents	8,078	6,664	6,362	7,769	12,179
Automobile accidents	15,714	13,656	12,293	11,067	2,061
Street-car accidents	1,771	1,748	1,725	2,128	2,998

* L. I. Duplin, *Statistical Bull.*, Metropolitan Life Insurance Co., New York.

¹⁹ American Association for Labor Legislation, 131 E. 23rd Street, New York City.

The number of death rates from automobile accidents alone in the United States for the five-year period 1920-24 were as follows:

Year	Number in the Registration Area	Rate
1920	9,103	10.4
1921	10,168	11.5
1922	11,666	12.5
1923	14,411	14.9
1924	15,528	15.7

In 1923, the death toll from accidents amounted to 1,462 per week or 209 per day! Automobile accidents and injuries head the list with 262 deaths per week and 37 deaths per day in Continental United States. Then come falls, with 254 deaths per week or 36 per day, and drownings, with 134 deaths each week or 19 for each day. There were 128 killed each week in railroad accidents, or 18 per day. These are lamentable facts of destruction of valuable lives and a frightful price for carelessness, apathy, speed and deficient knowledge. Accidents have become one of the major causes of deaths, being outnumbered only by such causes as cancer, pneumonia, tuberculosis and organic diseases of the heart. The rate of non-industrial accidents increased from 7.8 cases per thousand males in 1922, to 9.0 in 1923, and 9.6 in 1924.²⁰

Sedentary Occupations.—Sedentary occupations in themselves may lead to harm, especially in the cases of those who bend forward while at work, causing contraction of the chest and pressure upon vital organs which interferes with important physiological functions. The circulation is impeded, respirations are shallow, the utilization of food is diminished and the appetite fails, constipation and hemorrhoids are common, and there is a predisposition to common colds and diseases of the lungs.

Health insurance, or "sickness insurance," is a method by which the economic loss caused by sickness is distributed among a group of persons. The distribution is effected by the payment of periodic premiums on the part of members of the group. In this way the cost of sickness arising from the stoppage of income, from fees of doctors, nurses and hospitals, from expenditures for medicines and the like, does not come as a sudden financial burden to the insured individual. This kind of insurance is now provided in the United States by many commercial companies and by thousands of fraternal orders and benefit associations of a wide variety of types, and is taken advantage of by a large proportion of those who are thrifty enough and financially able to pay the premiums. In England there is compulsory insurance for all manual workers and all other employed persons with an income below 250 pounds per annum. In the principal European countries sickness insurance of wage earners has been made a governmental function, but with certain fundamental differences from that form of sickness insurance which exists in

²⁰ U. S. Pub. Health Rep., 1926, 41: 113.

this country. Among these differences are its extension to all wage earners upon a compulsory basis, the addition of medical and hospital service and certain other benefits of the cash payments to the sick, and the distribution of the cost of insurance not only among the insured, but also among the two other groups—employers and the public—who are considered responsible to some degree for the conditions which affect the health of the insured. The proposals for governmental health insurance in the United States not only adopt the principles just mentioned, but include additional features. Among these are adequate medical service for the insured, and definite provisions for rendering the health insurance system an aid to disease prevention. It has been proposed that the preventive force of governmental health insurance should not be limited to the financial relief during sickness, to the medical services afforded, and to the possible economic incentive to reduce sickness, but that it should be greatly increased by linking the health insurance system to the existing public health agencies. In this sense, "sickness" insurance, it is believed, would become a real health measure. It would not be merely a variety of commercial or mutual insurance, or another type of public relief, but a practical method of improving and extending the present facilities for the prevention of disease.

Classification.—Oliver divides the occupational diseases into five general classes:

1. Diseases due to gases, vapors, and high temperatures
2. Diseases due to conditions of atmospheric pressure
3. Diseases due to metallic poisons, dusts, and fumes
4. Diseases due to organic or inorganic dust and heated atmospheres
5. Diseases due to fatigue

The principal health risks in industry, as far as we know them, may be conveniently classified according to their nature as follows:

1. Dusts, fumes, gases, vapors, and acids (poisonous and non-poisonous)
2. Harmful bacteria and microorganisms
3. Compressed or rarefied atmospheres
4. Improper lighting
5. Extremes of temperature
6. Excessive strain

Many other classifications have been attempted, but it is evident that no general system can be entirely satisfactory. Each occupation requires individual study and separate consideration. In many occupations a combination of varying factors, such as dust, fumes, poisons, fatigue, etc., operate coincidentally. In some cases mixed poisons are the cause of industrial intoxication. In the following pages only the well-known and better studied diseases of occupation and the conditions which render them hazardous, as well as methods of prevention, are considered.

INDUSTRIAL POISONS

Industrial poisons are typically chronic; acute forms are comparatively rare. The number of industrial poisons is rapidly growing as a result of new processes and because the subject is receiving more careful attention. The protection of workers in the dangerous trades of America is still fragmentary and incomplete. Thus recently it has been shown that workers with heated tallow and other animal grease are subject to gastro-intestinal disturbances, apparently due to the volatile fatty acids that are given off and that the workers ingest and inhale. A large number of cases of poisoning by fumes of arseniuretted hydrogen have been reported in recent years, the gas being accidentally evolved when a metal such as zinc or iron containing arsenic as an impurity is brought in contact with a heavy acid. Skin diseases are caused by many of the coal tar derivatives, by some of the petroleum distillates, and are also frequently found among workers in the following trades: galvanizing, cutters of glass and pearl shell, workers with tar, paraffin, cement, dyes, printer's ink, and polishers.

A comprehensive list of substances that injure working people would be long and impressive. It includes acids and alkalies; metals, such as lead, arsenic, mercury, manganese and antimony; gases, such as carbon monoxid, hydrogen cyanid, sulphurated hydrogen; petroleum, benzin, naphtha and paraffin oil; wood alcohol, banana oil, formaldehyd, carbon tetrachlorid, tetrachlorethane; carbon disulphid; coal-tar benzene, anilin, nitrobenzene, phenol; turpentine; chromates. Serious as they are, the industrial poisons cause only a fraction of the total disabilities in industry.

The subject is illuminatingly considered in Dr. Alice Hamilton's book *Industrial Poisons in the United States*,²¹ which contains in detail our knowledge of each poison, with a comprehensive and well-selected bibliography. Dr. Hamilton has generously helped in revising this chapter and the reader is referred to her book for further information.

LEAD

Lead poisoning is the most frequent, most serious, and most insidious of all the occupational intoxicants. If a pound of lead drops on a workman's head the catastrophe is more obvious than if minute quantities of lead are taken into the system day by day, but the poisoning may be as fatal as the accident.

Lead is a typical cumulative poison. A large amount may be taken at one time without noticeable effect, but small quantities inhaled and ingested daily are absorbed, stored in the body, resulting in chronic poisoning and even death.

Practically all forms of lead are poisonous, even the metal itself. The basic carbonate and the suboxid are the most dangerous; the higher oxids,

²¹ The Macmillan Co., New York, 1925.

litharge and red lead, come next. The sulphate and chromate are less soluble in human gastric juice and the sulphid is the least soluble. The water-soluble compounds, the acetate, chlorid and nitrate, are unimportant in industry and do not often give rise to trouble.

Plumbism is not confined to industry. It is common in the population at large. "Epidemics" of lead poisoning sometimes occur from the drinking water, from beer and from other unusual sources. There are many curious sources of lead poisoning. Babies poison themselves by eating the lead paint from their cribs. A serious epidemic occurred in Philadelphia from lead chromate in a baking powder. In England, lead-lined vats for brewing beer caused a widespread outbreak. Face powders and cosmetics, a bullet in the body, or even bullets carried in the pocket have caused plumbism. A peculiar source is the lead-foil wrapping of packages of snuff which caused fatal poisoning in a young housewife.

Absorption.—Lead is absorbed from the respiratory tract and this is the most serious channel of entrance. It also comes in through the gastrointestinal tract. Absorption may take place through the skin, but the amount thus taken up from the usual sources seldom causes symptoms. Tetra-ethyl lead is rapidly absorbed through the skin. In the majority of cases of lead poisoning in the industries, the lead comes through the air to the victim as dust, sometimes as fumes. Preventive measures must, therefore, be directed toward keeping the air about the workmen free from lead. A lead trade is dangerous in proportion to its dustiness. The whole field of controversy over the mode of entrance of lead into the body, the way it is transmitted, its absorption, storing and excretion, has been illuminated by the "Lead Studies" which were carried on at Harvard, between 1922 and 1924, by Aub and his colleagues.²²

The dust or fumes are drawn into the nostrils, pass down through the nasopharynx, mingle with the mucus and saliva in part, and in part pass on to the lower respiratory tract. Absorption takes place all along the way, first from the nasal mucosa,²³ finally from the alveoli of the lungs, while the part that is mixed with the saliva is swallowed and reaches the stomach. The most acute and serious forms of lead poisoning follow exposure to large amounts of dust and fumes, for that which is taken up by the respiratory tract is directly absorbed by the blood stream, while lead conveyed to the mouth by dirty fingers or food must pass from the intestinal tract through the liver, a very efficient defense.

Absorption through the skin is possible, especially when lead is mixed with oil, as in paint; also from cosmetics and hair dyes containing lead. Whatever be true of the skin absorption of lead, its practical importance in industry is slight compared with the importance of absorption through the inspired air

²² J. C. Aub, L. T. Fairhall, A. S. Minot and P. Reznikoff, *Lead Poisoning*, Med. Mono., Vol. VII, Williams and Wilkins, 1926.

²³ H. L. Blumgart, "The Absorption of Lead from the Naso-pharynx," *J. Indust. Hyg.*, 1923, 5: 153.

or by the mouth. Süssman²⁴ concludes that the maximum cutaneous absorption under favorable conditions is from 0.1 to 0.2 milligrams of the metal per square decimeter ($\frac{3}{8}$ in.) of surface. Quantities of this magnitude are not large enough to produce lead poisoning.

The most satisfactory calculations are those of Legge,²⁵ based on analyses made by Duckering of the lead content of the air in certain workshops. Duckering concluded that 2 milligrams per day is the lowest dose, which, when inhaled as dust or fumes, may, in the course of years, set up lead poisoning. If the air breathed during the working hours does not contain more than 5 milligrams, encephalopathies and palsies will not occur, and colics rarely.

Nuns in a convent near Lyons were seriously poisoned and three died from using drinking water containing 2.7 milligrams of lead per liter. Only two escaped, one of whom had been there only five months, and the other one year. At the Château de Claremont, 13 of 24 persons were poisoned in a short time by drinking water with about 16.5 milligrams of lead per liter.

Excretion.—Lead is excreted principally in the feces, to a much less extent in the urine, and probably little if any at all by the skin. The elimination is slow and uncertain. It passes out directly through the large intestines, and also in the bile from the liver. Lead probably can be excreted by normal kidneys, but they are damaged, for a high incidence of chronic intestinal nephritis is found in chronic lead poisoning. The only constant finding in the urine is an increase of hematoporphyrin.

Studies of the United States Public Health Service²⁶ determined the fact that the analysis of normal feces of persons who gave no history of lead exposure showed an excretion of lead in varying amounts. It seems that our city dwellers daily take in some lead.

Transportation and Storage in the Body.—Lead is carried in the plasma of the blood largely as an insoluble triple phosphate. It is also picked up by the red corpuscles uniting with the phosphate of the cell membrane to form insoluble lead phosphate on the surface of the red cells. This makes the corpuscles hard, brittle and inelastic.

Lead is stored in the bones, probably as lead phosphate, which is insoluble in the normal hydrogen-ion concentration of the blood. As long as it remains in the bones it is harmless, and as long as the bones release the lead gradually and in small amounts it is excreted in the feces and urine without causing symptoms. It is during the transportation of lead that damage to the tissues occurs, and when transported in toxic amounts, poisoning results. The damage during transportation occurs both before and after storage in the bones, that is, after absorption and after release from the bones.

Lead is safely stored in the bones as long as there is a positive calcium balance and a normal hydrogen-ion concentration of the blood. A condition of acidosis or alkalosis will dissolve lead out of the bones, especially if calcium

²⁴ *Arch. f. Hyg.*, 1921, 90: 175.

²⁵ Legge and Goadby, *Lead Poisoning and Lead Absorption*, p. 207.

²⁶ *J. Indust. & Engin. Chem.*, 1926, 18: 193.

is deficient in the diet. This explains why an attack of lead poisoning may be precipitated in a leaded individual by an alcoholic spree, by an acute infection, or by an unbalanced acid diet. The treatment under these circumstances is again to fix the lead in the bones by an alkaline regimen rich in calcium. Hence the prophylactic value of milk.

Symptoms.—The classic symptoms of lead poisoning are colic, constipation, palsy, anemia, a blue line on the gums, rheumatic pains and various nervous manifestations. Plumbism may be either acute or chronic.

Chronic lead poisoning is more common than the acute form. It is characterized by anemia, dyspepsia, loss of appetite, headache, depression, constipation, colic; various forms of paralysis, especially paralysis of the extensor muscles of the forearm leading to wrist-drop; and a blue line along the edges of the gums, due to the formation of sulphid of lead deposited in the tissues. Optic neuritis may come on. There is an increase in the blood-pressure. Chronic lead poisoning leads to arteriosclerosis, fibrosis of the kidneys, and the remote consequences of these changes. Muscular paresis, pain and swelling of the joints, often occur and may be mistaken for "rheumatism." In some cases gout is closely simulated. The pain is usually worse at night. Headache is common. Lead insanity or apoplexy may supervene. There is a lowered resistance to certain infections, especially tuberculosis. From time to time there may be in the course of chronic plumbism, a typical attack of lead colic and acute symptoms. The picture of chronic lead poisoning is endlessly varied.

The early symptoms may be vague and readily overlooked. Early diagnosis is important. Mild cases of lead poisoning may show only symptoms of anemia, vague or fugitive pains, or a mild type of peripheral neuritis. This stage of lead poisoning, which does not vary essentially from other intoxications of mild degree, is readily overlooked clinically. Among the earliest manifestations of lead poisoning is the pallor of the skin, out of proportion to the anemia as determined by the redness of hemoglobin and the number of red blood-cells. Other early symptoms are the grayish (saturnine) pallor; the pinched appearance of the face; loss of appetite; disagreeable sweetish taste; sense of weariness disproportionate to the exertion; loss of strength; loss of weight; gastric distress; constipation; disturbed sleep. Aids to diagnosis are the detection of lead in the feces or urine and the discovery of stippled red blood-cells, which are not significant unless the number is more than 250 out of a million.

The anemia is secondary, seldom pronounced, the blood counts usually running between four and five million and the hemoglobin 80 per cent, rarely under 50 per cent. Nucleated cells are present. The most characteristic feature of the blood picture is a stippling of the red corpuscles with a remarkable granular basophilic degeneration, which shows with polychrome methylene-blue. Borax toluidin blue is most satisfactory. This granular degeneration is found in other conditions, as pernicious anemia, and even in normal blood, but is most constant and most numerous in lead poisoning.

Lead palsy is fairly common in chronic but rare in acute plumbism. It is a motor paralysis affecting those muscles which have been subjected to greatest use; in painters, the extensors of the wrist; in file cutters, the interosseous muscles of the hands; in lead smelters, the muscles of the upper arm and shoulder; and in children, the muscles of the foot and ankle.

Acute lead poisoning may be severe and is often fatal. It occurs especially when the lead is absorbed from the respiratory tract in persons recently exposed. There may be intense gastro-intestinal symptoms, constipation, colic or diarrhea with enteritis, and a rapidly developing anemia. Acute poisoning is apt to manifest itself in the cerebral form of plumbism known as lead encephalopathy, characterized by epileptiform convulsions, acute delirium, maniacal excitement, delusions, attacks of unconsciousness, and partial or total blindness. It may end in death, or in recovery, or pass into lasting insanity.

Maternal and Congenital Effects.—Statistics as to the sterility of lead mothers with regard to premature deliveries, stillbirths, and the mortality of babies during the first year of life, come chiefly from England. They show that lead is a race poison in the sense that the effects are passed on to the offspring.

Fetal death and abortion are common in maternal lead poisoning. Legge abstracted from reports of the British Factory Inspection Department for 1897 the following statistics concerning women lead workers: Of 77 married women, 15 never became pregnant; 15 of the 62 who became pregnant never bore a living child; among the 62 there were in all 212 pregnancies, but these resulted in only 61 living children, 21 stillbirths, 90 miscarriages, and of the 101 children born alive, 40 died soon after birth. Tardieu reported to the French Government in 1905 that 608 out of 1,000 pregnancies in lead workers had resulted in premature birth. The offspring may even be affected when the mother is normal but the father "leaded." These results are fully confirmed by animal experimentation.

Cole and Bachhuber²⁷ fed lead acetate to rabbits and fowls. In the rabbits the mortality of the young during the first four days after birth was 47.7 per cent for offspring of "leaded" males, as contrasted with 29.2 per cent for offspring of normal males. The average weight at birth was 48.9 grams for the former and 59.0 grams for the latter. With Leghorn hens the results were similar. Weller²⁸ used commercial white lead (basic carbonate), which he fed in capsules to guinea-pigs. The experiments showed that paternal lead poisoning in guinea-pigs does not result in sterility or in stillbirth, but in reduction of weight at birth, and this underweight persists through life. Next to this, the most striking change is the high rate of mortality during the first few days after birth.

Lead during the period of growth has a dwarfing and crippling effect. It stunts the growth of children and has a like effect on animals and plants.²⁹

²⁷ *Proc. Soc. Exper. Biol. & Med.*, 1914.

²⁸ *J. Med. Research*, 1915, 33: 271, also *J. Am. M. Ass.*, 1915, 45: 26.

²⁹ W. B. Bell, *Lancet*, 1924, 1: 267.

Lead, like alcohol, syphilis and other poisonous influences, may so injure the sperm, the egg, or the fetus as to cause mental and physical defect in the offspring.

Susceptibility.—The individual susceptibility to lead poisoning varies remarkably. Of a number of individuals equally exposed some will suffer and others escape. Of those who suffer, the degree of intoxication varies considerably. It is quite common to find that among the members of a family using a water containing lead only one is stricken, while the others seem to be “immune”; that is, they either do not absorb the lead or are able to store or eliminate it. Young persons are much more susceptible than old. Women are more susceptible than men. Recognizing this fact, in 1898 England abolished female labor in the dangerous process of white lead manufacture.

Plausible reasons were formerly given to explain the differences in susceptibility, such as acidity of the gastric juice favoring solution and absorption, but the true explanation seems to be the varying degrees of success by which lead is stored and released from the bones. Diet that maintains a positive calcium balance plays a rôle. Alcoholism and other influences that cause acidosis precipitate an attack.

Personal cleanliness is another important factor, and workers in lead who do not give scrupulous attention to cleanliness of person and clothing suffer most. Persons who are not particularly careful about cleaning their hands before eating, or who frequently carry their fingers to their mouth and nose, run especial risks.

It has long been stated that the body can successfully handle 0.1 milligram of lead a day because this amount does not cause symptoms. The careful work of Bell and his workers³⁰ determined that the average amount of lead producing stipples is 0.14 milligram, and the average time, 12 days; the average amount producing polychromacy, 0.157 milligram, average time 13.4 days; the average amount producing nucleated cells, 0.136 milligram.

Effect of Occupation.—The character of certain occupations has an influence on the type of lead poisoning which develops. Thus Teleky finds that, while compositors in Vienna seldom suffer from colic or from the severer types of lead poisoning, they are subject to an unusual extent to diseases of the lungs and kidneys. The relation between tuberculosis and chronic plumbism is shown in Hahn's diagrams based on the records of typographical trades in Vienna and Berlin, the curves of the two diseases showing a remarkable parallelism. Colic is said by Legge to be most frequent among workers in white lead, red lead, enameling, storage batteries, coach painting (which involves sandpapering), while the slower form with paralysis is found in brass workers, plumbers, printers, file cutters, and tinsmiths. The former are very dusty trades; therefore, poisoning occurs rapidly and encephalopathy is more frequent than paralysis.

The manner in which lead is handled makes a vast difference so far as the

³⁰*Lancet*, 1925, 209: 795.

liability to plumbism is concerned. Thus Stüler found in Vienna that carriage painters are ten to twenty times more subject to lead poisoning than house painters. This has been confirmed by Edsall in this country. The reason for this is that carriage painters apply a large number of coats of paint and varnish, polishing between coats, thereby enveloping themselves in dust which contains much lead; furthermore, carriage painters are required to work indoors. For lead poisoning due to water, see page 1050.

Lead Oxids, Litharge and Red Lead.—In the manufacture of litharge and red lead, the metal is roasted in reverberatory furnaces and raked from time to time. The raking or rabbling and the discharge from the furnace may be mechanical, in which case the danger from fumes and dust is very much lessened. The oxids are light and fluffy and it is hard to control the dust produced in dumping, grinding, sifting and packing them, and although great improvements have taken place in many oxid works of late years, the rate of plumbism in this industry is still fairly high.

The oxids are used in making storage batteries, the plates of which are leaden grids with a paste of litharge or red lead rubbed into the interstices. This industry is recognized in all countries as one of the most dangerous of the lead trades. Litharge is also used in rubber compounding, and men employed in weighing out the compounds and on the mixing mills may suffer from lead poisoning. Red lead is used as paint for bridges, structural iron work, certain parts of railway cars, and ships—especially battleships. Stitt³¹ reported 3 cases of lead poisoning and encephalopathy in men who were chipping old red lead paint from the bottoms of battleships. Brown³² cited 83 cases of plumbism among men engaged in metal-cutting in the destruction of battleships, due to the volatilization of lead in the paint. Red lead is also used in making glazes for tiles and terra cotta, and enamel for porcelain enameled sanitary ware, and in these occupations there is a great deal of industrial plumbism.

White Lead.—Most of the lead carbonate is still made by the old Dutch method or by the Carter process. The old Dutch method consists in the transformation of metallic lead into the white carbonate by a slow and double process of conversion. Numerous earthenware pots containing 3 per cent of acetic acid are placed on tan bark in a large three-walled chamber, and upon these pots are laid thin strips of metallic lead and subsequently planks of wood. Tier after tier of pots resting on bark and covered with metallic lead and wood are thus superimposed until the chamber, 25 or 30 feet in height, is filled to within 6 feet from the top. This chamber, known as the "blue" bed, is kept closed for fourteen weeks or longer. Fermentation causes a rise in temperature and a production of carbon dioxid. The acetic acid acts upon the lead and converts it into acetate of lead, while the carbon dioxid evolved from the bark changes the acetate into carbonate or the well-known white lead of commerce. The danger of plumbism occurs during the emptying or stripping

³¹ *U. S. Nav. Med. Bull.*, 1912, 6: 161

³² *Ibid.*, 1925, 23: 187.

of what is now called the "white" bed. If sufficient time has not been given for the very soluble acetate to have become changed into the carbonate the danger is thereby greater. During the stripping of the "white" bed there is a considerable quantity of dust raised, a large part of which is white lead.

Legge found that of 1,463 persons employed off and on in white-lead works the incidence of lead poisoning was 6 per cent of the average number regularly employed, and in those casually employed 39 per cent. This shows the great risk of exposing unskilled labor in a dangerous occupation.

In making so-called pulp lead, the white lead suspended in water is not dried but is ground in oil which gradually displaces the water, a method involving far less risk of poisoning.

The Carter process, much used at present in the United States, is a rapid corrosion of atomized lead, the great advantage of which is that it is largely mechanical and requires very little labor in proportion to output.

The Manufacture of Pottery and Earthenware.³³—Lead poisoning in potteries and tile works has been notorious for years in Europe. In England and Germany, especially, great efforts have been made to protect the workers against this danger. Aside from the precautions taken to avoid dust and other dangers in potteries, the English use a great deal of leadless glaze, and when this is not possible they frit the lead, that is, they fuse it with the other constituents of the glaze, thus changing soluble white lead or red lead in part or wholly to the insoluble disilicate. In American potteries this is not usually done and the glazes often contain large quantities of lead soluble in gastric juice. It is also true that American potteries are rarely as clean as are German and British potteries, and personal protection for the potters against lead poisoning is lacking.

The chief centers for the manufacture of table and toilet ware, sanitary earthenware, so-called art pottery, and tiles, are Trenton, New Jersey, and the East Liverpool and Zanesville districts of Ohio. The dangerous processes are mixing the glaze, dipping the ware in the glaze, cleaning the dipped ware to get rid of the excess of glaze, and stacking it on boards or trays to be fired, firing it in the glaze kilns, and decorating it by the processes known as color blowing, or tinting and ground laying.

An investigation of American potteries, made by the United States Public Health Service and published in 1921 as Bulletin 116, showed very dangerous conditions. Seventy-eight of 107 samples of glaze contained over 10 per cent soluble lead and 11 had from 20 to 50 per cent. Much lead dust was found in the air of the mixing and dipping rooms, and little or no provision for personal cleanliness existed in most potteries, while in none was medical supervision found. Of 1,809 potters examined, a high rate, 22.8 per cent, of plumbism was found. The British rate in 1913 was only 0.9 per cent.

Lead Mining, Lead Smelting and Refining.—In lead mining there is less danger of poison, for the ore is chiefly sulphid which is only sparingly

³³ B. J. Newman, W. J. McConnell, O. M. Spencer, and F. M. Phillips, "Lead Poisoning in the Pottery Trades," *U. S. Pub. Health Bull.* No. 116, 1921.

soluble in human gastric juice. Further, this is a moist process. Smelting and refining of the ore are industries attended with a great deal of plumbism from both dust and fumes. In smelting, the greatest danger is found in the work at the open hearths, the blast furnaces, and the cleaning out of flues and bag houses; in refineries the danger from fumes should not be so great, but there is more dust because the refineries work up not only lead bullion but all varieties of lead scrap and refuse, some of which is very powdery and fluffy. The industry in the United States employed in 1913 some 7,500 men, and the rate of lead poisoning was over 22 per cent, whereas in England during the previous year the rate was 1.8 per cent among about 2,000 men.³⁴

Tetra-ethyl lead, $\text{Pb}(\text{C}_2\text{H}_5)_4$, has been developed to increase the efficiency of gasoline. Accidental exposure to tetra-ethyl lead in the plant where it is made has caused deaths and serious poisoning. These accidents occurred in connection with the manufacture and blending of concentrated tetra-ethyl lead and throw no light upon the hazard that may be connected with the general use of tetra-ethyl-lead gasoline. The amount used to treat gasoline is about 1 part per 1,300.³⁵

The United States Bureau of Mines showed that tetra-ethyl lead in gasoline gives an average of less than half the amount of carbon monoxid found in the average automobile exhaust gas, and "indicates seeming remoteness of any danger of undue lead accumulation in the streets through the discharging of scale from automobile motors." Some public health authorities have warned against, and even forbidden, the use of gasoline treated with tetra ethyl lead.

The question has recently been studied by Leake of the United States Public Health Service, whose admirable work is one of the outstanding achievements in industrial hygiene. The following conclusions were endorsed by a special committee:³⁶

1. Drivers of cars using ethyl gasoline as a fuel and in which the concentration of tetra-ethyl lead was not greater than 1 part to 1,300 parts by volume of gasoline, showed no definite signs of lead absorption after exposures approximating two years.

2. Employees of garages engaged in the handling and repairing of automobiles and employees of automobile service stations may show evidence of lead absorption and storage, as indicated by the lead content of the feces and the appearance of stippled cells in the blood. In garages and stations in which ethyl gasoline was used the amount of apparent absorption and storage was somewhat increased, but the effect was slight in comparison with that shown by workers in other industries when there was a severe lead hazard (Group E); and for the periods of exposures, studies were not sufficient to produce detectable symptoms of lead poisoning.

3. In the regions in which ethyl gasoline has been used to the greatest extent

³⁴ *Bull. No. 141*, U. S. Bureau of Labor Statistics.

³⁵ An organic halogen (first chlorin, then bromin) compound is added to convert the organic lead compound to lead chlorid (or bromid) in the exhaust; a colored dye is added to denote the treated gasoline.

³⁶ *J. Indust. Engin. Chem.*, 1926, 18: 193.

as a motor fuel for a period of between two and three years, no definite cases have been discovered of recognizable lead poisoning or other disease resulting from the use of ethyl gasoline.

In view of these conclusions your committee begs to report that in their opinion there are at present no good grounds for prohibiting the use of ethyl gasoline of the composition specified, as a motor fuel, provided that its distribution and use are controlled by proper regulations.

Gasoline containing tetra-ethyl lead was first put on the market February 1, 1923, and its sale was voluntarily discontinued May 5, 1925, during which time about 300 million gallons were distributed.

Miscellaneous Industries.—Layet computed that in France 111 industrial processes involved the use of lead. Hamilton found 70 such processes in Illinois in which lead or its compounds are handled and which have caused lead poisoning in recent times.³⁷

Some of the industries in addition to those mentioned above in which lead poisoning may occur are: making and selling wall paper; retouching photographs with white lead paint for catalogues and advertisements; polishing brass (the alloy containing lead); polishing cut glass with lead putty powder; mixing compounds for rubber; the making and handling of storage batteries. Leake³⁸ found lead in the dust of garages varying in quantities from 0.82 milligram to 22.31 milligrams per gram of dust. The trades that use metallic lead are very numerous, including the making of wire, sheet, piping, plumbers' goods, machine parts, tinfoil, car seals and can seals, picture frames, trimmings for coffins, and the use of solder and Babbitt. The most important one, however, is the printer's trade, where the amount of lead dust and fume is slight, but long continued exposure brings about a serious form of chronic plumbism marked especially by a lowered resistance to tuberculosis.

Certain occupations which are dwelt on in the European literature as sources of serious lead poisoning are fairly free from this danger in the United States because our methods are different. File-cutting is one of these, the file being strapped to a strip of lead; another is diamond polishing, the jewel being imbedded in lead. Very little jewel polishing of this sort is done in the United States and file-cutting is mostly done by machinery. Europeans also use lead colors in printing textiles and dyeing them, as we do not, and while we plate kitchen ware with a lead-free enamel or with a pure tin, they plate it with a mixture of tin and lead, often a large proportion of the latter.

PREVENTION

The prevention of lead poisoning rests, in the main, upon the fact that the lead comes to the workman usually as dust, sometimes as fumes through the air, but it must be remembered that lead is also carried to the mouth by deposits on the hands and other objects. Practical efforts for the protection of lead workers must be directed first to the prevention of dust and the removal

³⁷ *J. Am. M. Ass.*, 1911, 56: 1240.

³⁸ *J. Indust. & Engin. Chem.*, 1926, 18: 193.

of fumes; second to provisions for cleanliness of body, clothing and surroundings.

The first essential then is to keep the air which the workman breathes and which surrounds him free of lead. Most cases of lead poisoning could be averted by a proper system of ventilation. Certain processes should be carried on under hoods with a strong draft, or in cabinets, or in special rooms with an air current so arranged that the lead is kept away from the mouth, nose, hands, and clothes of those who are exposed. It takes money, engineering skill and intelligence to keep lead out of the air.

On the part of the workman the prevention of lead poisoning consists in cleanliness of the hands and of the finger nails, frequent bathing, and the use of special clothing while at work. Care must be taken not to carry the fingers, which may be contaminated with lead, to the mouth and nose, and to wash the hands thoroughly before eating. Workmen should never take their lunch in the rooms where there is a suspicion of lead in the air. The hazard of chewing tobacco under such circumstances is obvious. Respirators may afford protection in emergency and short exposure, but cannot be depended on as a routine precaution, because the workmen will not wear them continuously.

Cleanliness is one of the all-essential requirements. A special room for the clothes of the workmen and special overalls should be provided for those who are exposed to lead. It is ignorance of the danger and the want of personal cleanliness that make casual labor in lead works especially dangerous. Even the women who wash the clothes of the workmen employed in lead factories may sometimes suffer from lead poisoning. Lavatories should be provided at the factory with warm water, soap, nail brushes, and towels.

Workmen should alternate employment and not remain too long in the dangerous departments. Supplanting hand labor by machinery diminishes the number exposed to the risk. A medical inspection is an important preventive guard in educating the workmen and in detecting mild and beginning cases.

A radical measure would be the substitution of zinc-white for lead paints. Zinc may be used as a substitute for lead, especially in indoor work; in fact this has been required by law in France. White lead appears to be superior to zinc for outdoor work.

Milk taken frequently and in generous amounts has long been known to be preventive. It was formerly believed that milk acted by keeping down the acidity of the gastric juice, but it now appears that milk is preventive because it is rich in calcium which keeps the lead safely stored in the bones.

The Massachusetts State Board of Health issues the following protective measures against lead poisoning: ³⁹

The poison gains entrance into the system:

1. By swallowing minute particles of lead.

³⁹ See also *U. S. Public Health Rep.*, Dec. 19, 1919, p. 2905.

2. By inhaling lead dust or the fumes of lead in a molten state, or the vapor of lead in a fused state.
3. By absorption from the skin in handling lead.

Advice to Employees

1. General personal cleanliness is of the first importance.
2. Thoroughly clean your hands before touching food and before leaving the workroom.
3. Thoroughly rinse your mouth before eating.
4. Take good, nutritious food and plenty of milk.
5. Take a substantial breakfast; an empty stomach is more susceptible to the poisonous effects of lead.
6. Never eat at your work. Eat your luncheon outside of the workroom if possible; if not, in a part of the room away from the lead. Never smoke or use tobacco in any form while at work.
7. Avoid all excesses; alcoholic beverages are especially injurious.
8. Wear overalls or a long coat at your work; also a cap or some head covering. Whenever practicable wear gloves when lead is to be handled.
9. Persons working in white lead or other powdered compounds of lead should always wear a respirator while at work. Cause as little dust as possible.
10. Consult a physician at the first sign of ill health.

Advice to Employers

1. Provide washing facilities, lockers, and a place for the employees to eat luncheons away from lead.
2. Provide respirators for all the workers who have to handle white lead or other powdered compounds of lead.
3. The floors of the workrooms and benches at which men work should be cleaned daily after thoroughly moistening them.
4. These regulations should be posted in a conspicuous place in the workroom.

PHOSPHORUS

There are two kinds of phosphorus: (1) the white or yellow, discovered by Brandt of Hamburg in 1669, (2) the red or amorphous, discovered by Schröter of Vienna in 1845. The amorphous phosphorus is obtained from the white phosphorus by exposing it in a closed vessel for some time to a temperature of 250° C. The white or yellow phosphorus is poisonous and was formerly the cause of much suffering in the match industry. One to 3 grains will cause death. The red or amorphous phosphorus is practically not poisonous.⁴⁰

Three kinds of matches are made: (1) The safety match, which contains

⁴⁰ J. B. Andrews, "Phosphorus Poisoning in the Match Industry," *Bull* 86, U. S. Bureau of Labor.

no phosphorus and is not poisonous. The match heads contain potassium chlorate or chromate and other compounds rich in oxygen from which the oxygen required to induce conflagration is evolved. The paste applied to the side of the match box contains antimony sulphid and red phosphorus. (2) The strike-anywhere match contains the poisonous white phosphorus in the head, and in addition glue, chlorate of potassium, powdered glass, and magenta or some other coloring agent. The paste, or composition, contains on an average 5 per cent of phosphorus. It is in mixing this paste, especially when done by hand in open vessels, and also in dipping the wooden splints, that the work-people are exposed to fumes that become a menace to health. (3) The strike-anywhere match made with the non-poisonous sesquisulphid of phosphorus.

Phosphorus poisoning in industry is now controlled. This chapter in preventive medicine reads like the story of one of Dickens' novels with a purpose. Several years ago the Belgian Government offered a prize of 50,000 francs to any person who would invent a safety strike-anywhere match free from white phosphorus. The problem was solved by Sevène and Cahan of France, who demonstrated that the sesquisulphid of phosphorus would accomplish all that white phosphorus does without causing poisoning. It has been found that the sesquisulphid of phosphorus acts, in some instances, as an irritant, causing conjunctivitis and edema of the eyelids, also eczema of the skin. This may be obviated by bathing the eyes and douching the nostrils twice a day before leaving the factory with an alkaline solution of bicarbonate of soda.

White phosphorus lucifer matches were first made in 1833 and the disease known as "phossy jaw" was first reported by Lorinser of Vienna in 1845. Soon after that, similar cases were reported from practically all European countries. In the United States, lucifer matches were first made in Springfield, Massachusetts, in 1836, and in 1851 there was the first description of a case of phossy jaw in America, a man who was treated in the Massachusetts General Hospital. The cases of phosphorus poisoning in match workers attracted widespread attention because of the painful and disfiguring effects, and it was not long before the public in all European countries began to agitate against the conditions giving rise to it and to demand their abolition. After all forms of governmental regulation had been tried without success, the principal countries of Europe entered into an agreement, the Berne Convention of 1906, by which the manufacture, importation, and sale of white phosphorus matches were prohibited. The United States was not a party to this agreement, and, at this time and for several years after, there was a general belief in this country that our superior methods and our factory sanitation had driven phossy jaw out of American match factories, but the thorough investigation carried on by John B. Andrews in 1910 showed that this was a fallacy. He discovered a total of 150 cases of phosphorus poisoning in the match industry, some of them very severe, attended with shocking disfigurement, and four of them fatal. The publication of his report resulted in the passage of the Esch Law in 1912, which placed a prohibitive tax on white

phosphorus matches. In 1913 their importation became illegal and the following year their exportation.

Many other industrial poisons are much more prevalent and serious, but they seem to lack the dramatic interest of phossy jaw. The phosphorus fumes enter through defective teeth, reaching the periostium of the jaw-bones where they set up necrosis. Sometimes the periostium of other bones is attacked, rendering them brittle and liable to fracture.

ARSENIC

The common causes of poisoning are from the uses of arsenical compounds in syphilitic remedies, from its use for criminal or suicidal purposes, and from accidental poisoning resulting from arsenical colors and from accidental contamination of food or drink. Such poisoning may be acute, subacute, chronic.

Arsenic acts as an irritant to the skin and mucous membranes, setting up conjunctivitis, coryza, eczema and ulcerations; it also produces general poisoning, causing anemia and neuritis and degenerative changes in liver and kidneys. Arsenical neuritis is particularly severe. In industry the skin lesions are the most common and troublesome features and these usually come from dust. Next to the skin lesion the outstanding feature of chronic arsenic poisoning is multiple peripheral neuritis with sensory, motor and trophic disturbances. Sometimes the optic nerve is involved, causing blindness. Workers in compounds of arsenic often present general symptoms resembling those of lead poisoning; namely, anemia, loss of strength, loss of appetite, gastric disturbances, but instead of palsy of certain muscles there is a painful neuritis and the arsenical worker suffers from lesions of the skin which serve to distinguish this form of poisoning very sharply from plumbism. Workers in Paris green suffer from painful redness of the eyes and from eczema of the eyelids, they have painful ulcers around the lips and nostrils and in the folds of the skin where the perspiration collects. Ulcers of the scrotum are particularly common. Arsenic also appears in the urine.

Arsenic is present in almost all the sulphid ores of the metals and is therefore encountered in iron, lead, zinc and copper smelting. The flue dust of the western lead smelters contains large quantities which are recovered by sublimation as white arsenic—the trioxid, used in various industrial processes. The other compounds of arsenic which are industrially important are lead arsenate and the aceto-arsenite of copper, or Paris green, both of which are used to kill insects and parasites. Lead arsenate causes lead poisoning rather than arsenic poisoning. White arsenic is used as a sheep dip, and a preservative for hides, skins and feathers. It is also used in curing furs. The Massachusetts law forbids more than one grain of arsenic per square yard, but analyses reveal that it may reach 170 grains. Out of 42 samples of fur recently examined in America, 11 were found heavily loaded with arsenic. The presence of such large quantities of arsenic in furs that are worn or in rugs for rooms must be a source of danger.

The danger from arsenical colors in wall paper is due to the curious property of a certain mold, *Penicillium brevicarule*, which is likely to form in the paste in damp houses, decomposing the arsenic, liberating a poisonous gas, diethyl arsin. Arsenic is no longer used in the manufacture of the fuchsin group of dyes, which were poisonous. Scheele's green and rarely Schweinfurt green are used to color fabrics for artificial flowers and wall paper. This is no longer a danger in the United States for, inspired by the book of J. J. Putnam, Massachusetts passed a law in 1900 limiting the amount of arsenic in wall paper and textiles not used for clothing to 0.1 grain per square yard, and to 0.01 grain in textiles for clothing. In 1904 the United States Department of Agriculture found that only 4 out of 537 samples of wall paper had more than the legal amount. Wall papers examined in England were found to contain as high as 50 or 60 grains per square foot. There is also danger in the use of arsenical sprays for clothing to protect them against moths.

An Epidemic of Arsenic Poisoning.—During the latter part of 1900, there occurred in England and Wales an epidemic of arsenic poisoning, affecting at least 6,000 persons. The districts principally affected were in Lancashire and Staffordshire. The cause was arsenic which contaminated beer from breweries that used glucose and invert sugar from a single firm. From 0.008 to 0.131 per cent of arsenic was found in the glucose, and from 0.02 to 0.062 per cent in the invert sugar, estimated as arsenous oxid. The arsenic came from the sulphuric acid, used to hydrolyze the starch to make the glucose and invert sugar, and contained from 1.4 to 2.6 per cent of arsenous oxid.

Arsenic, as well as other irritants, favor growths of a cancerous nature. That the long-continued irritation of small quantities of arsenic may be followed by the formation of epithelial new growths was asserted by Jonathan Hutchinson in 1887, and many subsequent observations have confirmed his view.

MANGANESE

Up to 1913, this rare form of industrial poisoning was not known to exist in the United States, but the researches of Casamajor⁴¹ and of Edsall and Drinker⁴² during recent years have brought to light some 39 cases, while the number reported from European countries is only 15. There have been reported recently six cases of manganese poisoning.⁴³ The poisonings follow exposure, usually for some months, to dust containing manganese dioxid. The symptoms were likened by von Jaksch and Seelert to those of multiple sclerosis, by Edsall and Drinker to progressive lenticular degeneration. The last two authors summarized the symptoms in such poisoning as follows: A history of work in manganese dust for at least three months; languor and sleepiness; muscular twitchings, from tremors to gross rhythmical movements of arms and legs, trunk and head; cramps and stiffness of the

⁴¹ *J. Am. M. Ass.*, 1913, 40: 646.

⁴² *J. Indust. Hyg.*, 1919, 1: 183.

⁴³ *J. Am. M. Ass.*, 1925, 86: 2008.

calves, usually at night; slight increase in tendon reflexes; ankle and patellar clonus; retropulsion and propulsion; peculiar slapping gait; occasionally uncontrollable laughter, less often weeping; absence of sensory, gastrointestinal, eye, genito-urinary disturbances; negative blood, urine and spinal-fluid findings.

ARSENIURETTED HYDROGEN (AsH_3)

More serious than arsenical dust is the gas, hydrogen arsenid, commonly called arseniuretted hydrogen, or arsin, which is accidentally evoked in the course of many industrial processes. The effects of this gas often fail to be recognized because the presence of the arsenic is not suspected. Almost all of the metals used in industry are derived from arsenic-bearing ores, and are therefore frequently contaminated with arsenic. The larger part of the sulphuric acid used in industry is made from iron sulphid or pyrites, and may also have traces of arsenic. Whenever, therefore, such an acid and metal come in contact, hydrogen arsenid may be given off and severe, often fatal poisoning results from the inhalation of the gas. Workmen cleaning out or repairing iron tanks which have held arsenic, have been poisoned in this way. Other occupations which involve this danger are the production of hydrogen from zinc dust and hydrochloric acid, and the use of such hydrogen for the flame of the lead burner and for filling balloons. Similar accidents have occurred in making acids from arsenic-bearing compounds; treating waste zinc from a galvanizing plant with hydrochloric acid to make zinc chlorid; pickling metals, *i.e.*, dipping in acid in preparation for plating or enameling; and recovering copper by electrolysis. Wholesale poisoning from this gas occurred on a British submarine, the source being the storage batteries in which acid came in contact with plates made from an antimony-lead alloy which contained arsenic.

The symptoms of this form of poisoning are quite different from those caused by arsenical dusts. There is a rapid onset, with nausea, vomiting, pain in the epigastrium, headache, dizziness, dark or even bloody urine due to hemolysis. The red blood-corpuscles undergo a rapid destruction, falling sometimes to less than a million, and the liver and kidneys in fatal cases are found in a condition of hemorrhagic inflammation. The mortality in industrial cases is about 36 per cent.⁴⁴

AsH_3 is exceedingly poisonous in small amounts. Five milligrams per kilo of weight is fatal for cats. For man, estimates of the fatal dose vary from 0.1 to 0.15 gram. Serious symptoms may follow the inhalation of 0.01 milligram.

MERCURY

Mercurial poisoning may be contracted by workmen employed in extracting mercury from cinnabar (sulphid of mercury), in which it is usually found in

⁴⁴ T. M. Legge in Kober and Hanson's *Diseases of Occupation*, Philadelphia, 1916, p. 3.

nature. The ore is simply roasted and the mercury volatilizes and readily condenses in metallic form. Mercury volatilizes at a low temperature and it is this circumstance which creates much of the danger to those who work with this element, especially to men who work in a closed and heated atmosphere containing the vapor given off by the metal. Mercury is absorbed by the digestive system, by the respiratory tract, and also through the skin. As an instance of the absorption of mercury through the skin Edsall cites two cases in dentists who were poisoned as a result of the custom of working up the amalgam in the palm of the hands.

The occupations in which mercury is used and in which mercurial poisoning occurs are: the separation of gold and silver from their respective ores, which is done by means of an amalgam; the manufacture of incandescent lamps, in which mercury pumps are used to create a vacuum; in barometer and thermometer making; in felt-hat and fur dressing, in which mercuric nitrate is used; in fire-gilding, where an amalgam of gold or silver, after having been applied to an object, is heated and the mercury driven off; and other industries.

The New York and New Jersey section of the National Civic Federation in three months' time found 60 cases of mercurial poisoning, a nervous disease called in the trade "the shakes," among the hat makers of Brooklyn, Newark, and Orange as a result of the mercury salts used in preparing felt.

The symptoms of mercurial poisoning are: anemia, headache, dizziness, tremor of the muscles, especially the tongue and limbs, fetid breath, soft, swollen, and ulcerated gums, and loosening of the teeth. The submaxillary and other glands of the neck become painful and the secretion of saliva excessive. Erethism and apprehensiveness are common; in severe cases depression and melancholia. A persistent and apparently causeless diarrhea is frequently a symptom of mercurial poisoning. Salivation and stomatitis often result from the medical use of mercury compounds.

Daily exposure for two or three months to an atmosphere containing as small a quantity as 0.02 milligram of mercury per cubic foot of air results in poisoning. If the exposure is from three to five months, daily, it is estimated that there would be a total absorption of mercury varying from 0.771 to 1.285 milligrams.⁴⁵

Prevention.—The prevention of mercury poisoning is almost a direct counterpart of the prevention of lead poisoning. The air must be kept free of mercury, and this can be accomplished by enclosing all apparatus in which mercury is used, by proper systems of ventilation, or the use of hoods with forced draft and other devices to keep the mercury fumes away from the workmen. Rubber gloves may be worn to prevent absorption through the skin and also to prevent the carrying of the mercury to the mouth. Here again scrupulous cleanliness in and after leaving the workroom, a change of clothing, and washing the hands before eating are essential.

⁴⁵ J. A. Turner, *U. S. Pub. Health Rep.*, 1924, 39: 329.

BRASS

Brass is an alloy of copper and zinc, the cheaper varieties containing a large proportion of zinc and from 1 to 13 per cent of lead. Brass founders' ague has been known for fully a century. It is an acute ague-like attack, coming on usually after exposure to the fumes of molten brass. The worker suffers from a severe chill, weakness, nausea, and then fever, sweating and prostration, but usually he is able to return to work the next day and hardly ever considers the attack serious enough to send for medical assistance. It is the zinc in the alloy, not the copper, that causes sickness, and it is only when volatilized in the form of fumes and inhaled that the effect is produced. Brass polishers do not suffer from "the shakes," and the sickness described as brass poisoning in brass polishers is often, if not always, lead poisoning from the dust of cheap brass containing lead. True brass founders' ague occurs also in braziers who weld together metallic surfaces with the aid of a solder containing brass; also in zinc smelters, and rarely in galvanizers working over a hot zinc bath, in autogenous welders working with zinc. The brass industry is generally considered more unhealthful than the average, for in addition to brass fumes and lead dust, workers are exposed to fumes of acids, of volatile solvents for shellac and lacquer, and to great heat.

Other metallic poisons found in the industries are antimony, chromium, manganese, vanadium, and selenium.

PETROLEUM DISTILLATES

Petroleum, or aliphatic or fatty series, contains a large number of compounds which are important in industry. The petroleum distillates are mixtures of the hydrocarbons from CH_4 to $\text{C}_{35}\text{H}_{72}$. They are much used in industry for fuel and as solvents for fats, gums, resins, rubber, gutta-percha, and as illuminants and also as lubricating oils.

Alice Hamilton states ⁴⁶ that it is important to distinguish clearly between the petroleum solvents and the more highly toxic coal-tar solvents, benzene and toluene. It is very unfortunate that there should be two words so similar as benzin and benzene covering such different compounds, and that the term "solvent naphtha" should mean not petroleum naphtha but a mixture of coal-tar distillates. The confusion is increased by the fact that both classes of solvents are used in the same industries, and in the foreign literature it is sometimes impossible to tell just which body is meant by the writer, whether coal-tar benzene or petroleum benzin.

Naphtha, benzin and petroleum ether are volatile petroleum distillates which have a toxic action on the central nervous system. Acute poisoning is characterized by symptoms of mild intoxication very like that of alcohol. The condition is known as "naphtha jag" and is familiar to workers in rubber

⁴⁶ *Industrial Poisons in the United States*, The Macmillan Co., New York, 1925, p. 400.

factories. The more serious forms of poisoning occur usually when men are obliged to go into a tank or tank car which has held petroleum. A chronic form of benzin, naphtha or gasoline poisoning is probably common, but is not easy to diagnose because it does not produce characteristic symptoms.

Oil Furunculosis.—The lesions caused by the heavier distillates for the most part are confined to the skin. Kerosene which is used to clean oil from machinery may cause acne or eczema. Lubricating oils are more troublesome, especially the fractions between the paraffins and vaselins. These lubricating or cutting oils are a prolific source of skin lesions in machine shops. They irritate the ducts of the sebaceous follicles, which swell, close and clog, and the resulting inflammation may eventuate in a general furunculosis.

All cutting oils have the power of penetrating the skin and carrying bacteria with them. Bacteria grow in some oils, and these are especially harmful. This explains why oil folliculitis and furunculosis is more common in some machine shops than others. Prevention consists in cleanliness, boiling the oil from time to time, or adding a germicide as phenol.

The literature concerning skin lesions in workers with coal tar, paraffin, vaselin, soot, pitch, shale, etc., is very confusing. Alice Hamilton states that it is impossible in most instances to gain any clear idea as to just what substances are playing a part in the acne, furunculosis, warty growths, keratomatous plaques or cancers which are described. It is commonly believed that tar cancer comes from coal tar rather than petroleum products.

Tar Cancer.—Evidence has gradually accumulated pointing to the presence of some cancer-provoking substance, not only in certain kinds of coal tar, but in petroleum oils, coal tar oils, pitch, and paraffin. This form of cancer was met abroad in chimney sweeps and in briquette making. The cancer of mule-spinners is usually on the left anterior aspect of the scrotum, and this is explained by the custom of the mule-spinner to lean much of his time with his left groin pressed against a steel bar which is covered with oil, and his clothes at this point are soaked with oil so that the skin is constantly irritated with a paraffin oil.

Leitch⁴⁷ states that paraffin cancers follow long exposure to action on the skin of crude mineral oils, and although they are called paraffin cancers, it is not at all certain that the harmful agent is a member of the paraffin series.

Experimental skin cancer by the external application of coal tar was first successfully produced by Yamagiwa and Ichikawa on the ears of rabbits, and since then a large number of experimenters have produced such growths not only with coal tar, but with the watery, alcoholic and ether extracts.⁴⁸

Naphtha Cleaning.—Dry cleaning establishments in this country are of two kinds—wholesale or retail. The retail establishments handle women's lighter clothing, curtains, gloves, etc.; the wholesale, chiefly men's clothing, women's heavy clothing, and bedding. Naphtha is almost universally used

⁴⁷ *Brit. M. J.*, 1922, 2: 1101.

⁴⁸ Murray, *Brit. M. J.*, 1922, 2: 1103.

by both. Benzene was introduced into many plants just after the war, but the risk from explosion was too great and it was given up. Several owners also decided that it was dangerous for the employees. It costs a little more a gallon than naphtha and only a very few plants now use it.

COAL TAR DISTILLATES

Benzol or benzene, C_6H_6 , is one of the coal-tar distillates, and is used either pure or as commercial benzene, which contains such impurities as xylene and toluene. Benzene is an excellent solvent for gums, fats and resins, and is used most largely in the manufacture of rubber goods, sanitary food cans, fabrikoid and artificial leather, and in varnish, lacquer, quick-drying paint, and paint and varnish removers. (Note distinction between benzene and benzin, page 1249.)

Benzene enters the body as a vapor, irritates the respiratory tract, and produces acute and chronic poisoning. In acute poisoning the symptoms are of respiratory and nervous origin and range from cough, vertigo, tinnitus, vomiting, perspiration and pruritus, to cyanosis, irregular pulse, anesthetics, delirium, convulsions, coma and death. In the chronic form there are purpuric hemorrhages under the skin and from the mucous membranes, and an aplastic anemia, with marked loss of white cells as well as red cells and absence of new forms. Death is caused by the anemia, often preceded by an uncontrollable hemorrhage.

Anilin, nitrobenzene, etc.—These benzene derivatives differ in their effect from benzene. They cause the production of methemoglobin and the cyanosis caused by this is a prominent and early symptom. There is a destruction of red blood-cells and a consequent hematogenous jaundice, which is usually not marked except in poisoning from the nitro compounds, such as trinitrotoluene. An acute attack comes on with flushing of the face, a throbbing headache, dizziness, confusion, then lividity of face and tongue, the voiding of dark brown or smoky red urine, loss of consciousness, with shallow breathing and a small pulse. At this point if blood is drawn it is thick and chocolate-colored. Death may occur from paralysis of respiration, but this is rare. The nitro compounds are more toxic than the hydro (phenol), the amid (anilin) or the chlor compounds.

Poisoning may occur through the breathing of fumes, but skin absorption is far more important, and protection against this group of poisons depends on strict cleanliness of the premises and ample washing facilities for the workers. The compounds are manufactured for use as explosives, dyes, drugs, and accelerators of rubber vulcanization. Anilin was formerly much used in the rubber industry, but its place has been taken by less toxic compounds.

OTHER TOXIC GASES AND VAPORS

Carbon Disulphid.—This is a powerful solvent for fats and gums and has long been used in the rubber industry for so-called cold or acid vulcanization.

Sulphur monochlorid is dissolved in carbon disulphid and the rubber is dipped in the fluid or painted over with the fluid or hung in its vapors. This mode of vulcanization is not so generally used in the United States as in European countries.⁴⁹ The symptoms of carbon disulphid poison consist in excitement followed by depression and apathy. There is an increasing weariness and loss of strength, most marked in the legs, climbing stairs becomes difficult. There is usually drowsiness, even mental confusion, and sometimes severe headache and insomnia. In serious cases which come on rapidly, there is acute mania; in those that come on more slowly, paralysis. There may also be impairment of sight from atrophy of the optic nerve. Because of its inflammable character, carbon disulphid is gradually being replaced in rubber works by the non-inflammable and far less toxic carbon tetrachlorid. It is, however, coming into use in a new industry, the making of artificial silk. (See also pages 272 and 866.)

Wood Alcohol.—Industrial wood alcohol poisoning has assumed great importance of late years in connection with the use of shellac, varnish, paint and varnish removers, the stiffening of felt hats, the making of celluloid and the making of dye intermediates and explosives. In 1914, Tyson and Schoenberg⁵⁰ estimated that two and one-half million workers in the United States are exposed to wood alcohol by inhalation or contact in the course of their work, and they have discovered about 100 industrial cases. The effect of wood alcohol differs in many respects from that of grain alcohol. It acts more slowly, and is far more slowly eliminated. In a typical case, the workman suffers for some time from attacks of dizziness and headache and weakness, which gradually increase in severity, then he notices a dimness or foginess before the eyes which may suddenly pass into complete blindness. This blindness is due to an optic neuritis, followed by atrophy and the result is partial or total permanent blindness. Severe poisoning causes death, preceded by blindness, convulsions and coma. See also page 469.

A revenue bill passed in 1906 permits the use in industry of grain alcohol denatured by the addition of 2 per cent, 4 per cent, 10 per cent, or 20 per cent of wood alcohol, and 0.5 per cent of pyridin bases. This is of course far safer than pure wood alcohol, but the use of the formulæ containing the larger proportions of wood alcohol is not unattended with risk, for some people are decidedly susceptible and react to a small quantity of the poison.

Carbon monoxid is colorless and inodorous and therefore gives no warning of its presence. It is responsible for more deaths than all other gases put together. Carbon monoxid is not a true poison, that is, it seems to be entirely inert except in one respect—its power of combining with the hemoglobin of the blood-corpuscles, thereby disabling their chemical function of carrying oxygen to the tissues. The effect produced by carbon monoxid is due, therefore, to lack of oxygen and the symptoms are those of anoxemia. The former view that carbon monoxid is a direct poison to the nervous tissue and may act

⁴⁹ U. S. Dept. of Labor Statistics, *Bull. No. 179*.

⁵⁰ *J. Am. M. Ass.*, 1914, 63: 915.

as a chronic poison is now denied. It is one of the products of the incomplete combustion when coal, wood, or gas is burned in an atmosphere without enough oxygen; carbon monoxid being formed instead of carbon dioxid and water.

It is met with in coal mines and other subterranean galleries where blasting has been effected by dynamite and gunpowder. It forms 7 to 10 per cent of ordinary illuminating gas (coal gas) and 30 per cent of water gas. It is the source of blue flame seen on the surface of an ordinary coal fire. The gas is given off in quantities from coke ovens; it is evolved from blasting furnaces in the smelting of iron, especially during the charging of furnaces and their tapping. Carbon monoxid frequently remains in the furnace, and workmen who enter such a furnace in order to clean it may be overcome. In England the law requires two workmen to clean furnaces; one stands by in case of accident. Carbon monoxid is also evolved from hot-water heaters; in the Leblanc process of soda manufacture; in cement and brick works; from the use of producer gas and a fuel oil. Carbon monoxid is given off in the exhaust gases from motor cars, and is responsible for the serious poisoning, often fatal, which has occurred when the engine of a motor has been allowed to run in a closed garage.

The poisonous properties of carbon monoxid are, according to Haldane, due to the great affinity it has for the hemoglobin of the red corpuscles. It has from 140 to 250 times greater chemical affinity for hemoglobin than oxygen. It forms carbon monoxid hemoglobin, a more stable compound than oxyhemoglobin, and therefore prevents the oxygen being given to the tissues. When the percentage of carbon monoxid rises to 0.4 the atmosphere becomes dangerous to animal life. Henderson states that an exposure to 4 parts in 10,000 parts of air for forty-five minutes causes no ill effects.

There is no known poison, the effects of which are as varied and widespread (Lewin). Mental changes and paralysis following gassing are common. The inhalation of carbon monoxid causes headache and a sense of loss of power in the lower extremities. It is this circumstance which explains many of the cases of fatal poisoning in confined spaces. There are also dizziness, throbbing of the temples, ringing in the ears, a sense of lassitude, and, in severe cases, convulsions and loss of consciousness. The inhalation of small quantities also lead to delusions and other mental symptoms. If the gas enters a bedroom and is inhaled by persons who are asleep the sleep only becomes deeper and profound narcosis is developed from which there may be no awakening.⁵¹ The damage done to the cells of the brain and cord may be permanent, paralysis and mental symptoms persisting after recovery from the immediate effects of the gassing.

Oliver gives the following illustration of the subtle poisoning by carbon monoxid at Pelton Fell, a mining village in Durham County. Some shale which had been tipped at the edge of a ravine caught fire. The carbon monoxid gas given off during the combustion traveled through the soil and en-

⁵¹ See also Bureau of Mines Technical Papers 156 and 106.

tered two houses in different streets, full 30 feet away, causing the death of two elderly people. It is to the breathing of this gas during sleep that the death of tramps, drawn to the coke ovens by their inviting warmth on a winter's night, is attributed. I have already instanced the case of death from carbon monoxid resulting from the imperfect operation of a gas water-heater. The subject is discussed in detail on pages 856 *et seq.* The literature on carbon monoxid poisoning is voluminous.⁵²

Hydrogen sulphid is an extremely poisonous gas causing death instantaneously if inhaled in large quantities. In smaller amounts the symptoms caused are nausea, vertigo, headache, general malaise, all of which soon disappear if the workman goes into the open air.

The industrial processes in which hydrogen sulphid gas may be encountered are: the decomposition of organic matter, in tanning, fat rendering and soap making, glue manufacture, sewage disposal, in certain chemical processes, as the decomposition of sulphids by acids, the preparation of barium trisulphid and sulphur monochlorid and sulphur dyes, especially brown and khaki; and in making illuminating gas and coke by-products.

In nature, hydrogen sulphid may occur in dangerous amounts in coal mines, from the decomposition of pyrites, and in smaller quantity about privies, the mud of marshes, and collections of filth and manure, but in amounts too small seriously to influence health. See page 863.

Other toxic gaseous vapors and fumes occurring more or less commonly in industrial processes are: Acetaldehyd, acrolein, ammonia, amyl acetate, amyl alcohol, carbon tetrachlorid, chlorin, diazomethane, dimethyl sulphate, dichlorethylene, dinitrobenzol, ether, formaldehyd, hexamethylenetetramin, hydrocyanic acid and cyanids, naphtha and gasoline, nitrobenzol, nitroglycerin, nitrous gases, phosgene, phosphoretted hydrogen, sulphur dioxid, tetrachloramethane, turpentine and many others. Only a few of these have been discussed as examples. See also poisonous gases in the atmosphere, page 856.

OCCUPATIONAL POISONS⁵³

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
1. Acetadehyd.	Irritation of the mucous membranes of the nose, larynx, bronchi, and eyes; acceleration of the heart's action; profuse night sweats.	Aldehyd pump men; celluloid makers, dye makers; explosives workers; mirror silverers; varnish makers; vinegar workers.
2. Acridin.	Irritation and inflammation of skin and mucous membranes, severe burning and itching of the skin, violent sneezing.	Dye makers.

⁵² Alice Hamilton, *Industrial Poisons in the United States*, The Macmillan Co., New York, 1925, p. 371.

⁵³ L. I. Dublin, and P. Leiboff, "Occupation Hazards and Diagnostic Signs," Bur. of Labor Statistics, 1922, *Bull. No. 306*.

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
3. Acrolein.	Itching in the throat, irritation of the eyes, exciting lachrymation, conjunctivitis, irritation of the air passages, bronchial catarrh.	Bone renderers; fat renderers; galvanizers; lard makers; linoleum makers; linseed oil boilers; soap makers; stearic-acid makers; tallow refiners; tanners; varnish boilers.
4. Ammonia.	Acute inflammation of the respiratory organs, cough, edema of the lungs, chronic bronchial catarrh, redness of the eyes, increased secretion of saliva, retention of the urine.	Acetylene makers; ammonium-salts makers; artificial-ice makers; artificial-silk makers; boneblack makers; bronzers; coke-oven workers; dye makers; dyers; galvanizers; gas (illuminating) workers; gas purifiers; glue workers; mercerizers; refrigerating-plant workers; salt extractors (coke-oven by-products); sewer workers; shellac makers; shoe finishers; soda makers; sugar refiners; tanners; varnish makers.
5. Amyl acetate.	Nervous symptoms, headache, fullness of the head, giddiness, numbness, nausea, disturbances of digestion, palpitation of the heart, inflammation of the respiratory organs, fatty degeneration of the liver.	Alcohol-distillery workers; art-glass workers; artificial-silk makers; battery (dry) makers; bronzers; buffers (rubber); celluloid makers; cutlery makers; enamellers; explosives workers; furniture polishers; gilders; incandescent-lamp makers; jewelers; lacquer makers; linoleum makers; mottlers (leather); patent-leather makers; polishers; shellac makers; shoe factory workers; shoe finishers; smokeless-powder makers; toy makers; wirers (incandescent lamps).
6. Amyl alcohol.	Congestion of the head, oppression of the chest, irritation of the air passages, lowering of the blood pressure, faintness, nausea.	Alcohol distillery workers; dye makers; fruit-essence makers; mordanters; shoe finishers.
7. Anilin and other amino compounds of benzol and its homologues.	Pallor of the skin, vertigo, unsteady gait, loss of appetite, increased frequency of respiration, anemia, slowing of the pulse, eczematous eruptions, bloody urine, spasmodic muscular pains, cyanosis.	Anilin makers; artificial-leather makers; calico printers; coal-tar workers; compositors; compounders (rubber); dye makers; explosive workers; feather workers; lithographers; millinery workers; mixers (rubber); painters; paint-makers; pencil (colored) makers; photographic workers; pressroom workers (rubber); printers; reclaimers (rubber); rubber workers; tannery workers; vulcanizers.
8. Antimony and its compounds.	Itching eruptions of the skin; inflammation of the mouth, throat, and stomach; albumin in the urine, weakness of the heart, vertigo, faintness, coryza, dyspepsia, intestinal colic, nephritis.	Antimony extractors (refiners); brass foundries; burnishers (iron and steel); burnishers (rifle barrels); calico printers; color makers; compositors; compounders (rubber); dye makers; enamel makers; filers; fireworks makers; glass mixers; glaze dippers (pottery); glaze mixers (pottery); grinders (metals); grinders (rubber); lead smelters; linotypers; mixers (rubber); monotypers; mordanters; pressroom workers (rubber); printers; rubber workers; shot makers; stereotypers; vulcanizers.
9. Arsenic and its compounds.	Headache, melancholia, sleeplessness, gastric disturbances, emaciation, catarrh of the mucous membranes, skin diseases of various forms, falling out of the hair and nails, melanosis, perforations of the nasal septum, bleeding gums, peripheral multiple neuritis, paralysis.	Arsenic roasters; artificial-flower makers; artificial-leather makers; bookbinders; brass foundries; bronzers; calico printers; candle (colored) makers; carpet makers; carroters (felt hats); chargers (zinc smelters); color makers; colored-paper workers; compounders (rubber); copper foundries; copper smelters; curriers (tannery); cut-glass workers; decorators (pottery); dye makers; electroplaters; enamellers; feather curers; feather workers; felt-hat makers; ferrosilicon workers; fur handlers; fur preparers; galvanizers; gardeners; glass mixers; glaze dippers (pottery); glaze mixers (pottery); gold refiners; insecticide makers; japan makers; jewelers; lead smelters; linoleum colorers; lithographers; mixers (rubber); mordanters; paper glazers; paperhangers; pencil (colored) makers; pitch workers; pottery workers; pressroom workers (rubber); printers; pyrites burners; refiners (metals); rubber-tire builders; rubber washers; rubber workers; sealing-wax makers; sheep-dip makers; shot makers; sprayers (trees); sulphur burners; sulphuric-acid workers; tannery workers; taxidermists; tanners; toy makers; velvet makers; wallpaper printers; wax-ornament makers; wire drawers; wood preservers; zinc miners.

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
10. Arseni- uretted hydrogen.	General malaise, difficulty of breathing, fainting fits, gastric disturbance, jaundice, bluish discoloration of the mucous membrane, pain in the region of the spleen and kidney, darkened urine, fetor of the mouth resembling garlic.	Acid dippers; anilin workers; balloon (toy) fillers; battery (dry) makers; bronzers; carbonizers (shoddy); dimethyl-sulphate makers; dye makers; enamellers; ferrosilicon workers; fertilizer makers; galvanizers; lead burners; lime burners; nitroglycerin makers; picklers; refiners (metals); shoddy workers; submarine (storage battery workers); sulphuric acid workers; tanners; towermen (sulphuric acid); zinc chlorid makers.
11. Benzene.	Headache, vertigo, nausea, cough, irregular respiration, weakness of the heart, drowsiness, cyanosis, twitching of the muscles, psychosis, skin lesions.	Art-glass workers; bronzers; buffers (rubber); cast scrubbers (electroplaters); celluloid makers; cementers (rubber shoes); cement mixers (rubber); compositors; compounders (rubber); curriers (tannery); decorators (pottery); degreasers (fertilizer, leather); dippers (rubber); dry cleaners; electroplaters; enamellers; feather workers; furniture polishers; gilders; glue workers; japan makers; lacquer makers; linoleum makers; lithographers; millinery workers; mixers (rubber); mordanters; painters; paint makers; pressroom workers (rubber); printers; putty makers; rubber-glove makers; rubber-tire builders; rubber washers; rubber workers; shellac makers; shoe finishers; tannery workers; type cleaners; varnish makers; vulcanizers.
12. Benzol.	Headache, vertigo, anemia, muscular tremor, scarlet lips, spots of extravasated blood in the skin, irritant cough, fatty degeneration of liver, kidneys, and heart.	Anilin makers; artificial-leather makers; battery (dry) makers; benzol stillmen; bronzers; carbolic-acid makers; cast scrubbers; cementers (rubber shoes); cement mixers (rubber); coal-tar workers; coke-oven workers; color makers; compounders (rubber); decorators (pottery); degreasers (fertilizer, leather); dry cleaners; driers (rubber); dye makers; electroplaters; explosives workers; feather workers; fertilizer makers; gas (illuminating) workers; gilders; glue workers; lacquer makers; lithographers; millinery workers; mixers (rubber); mordanters; painters; paint makers; photo-engravers; photographic workers; pressroom workers (rubber); reclaimers (rubber); rubber-tire builders; rubber washers; rubber workers; shellac makers; shoe-factory workers; shoe finishers; smokeless-powder makers; still (coal-tar) cleaners; treaders (rubber); varnish makers; vulcanizers.
13. Brass (zinc).	Headache, general malaise, throat irritation, cough, nausea, vomiting, constipation, trembling, muscular pains, accelerated respiration, profuse sweating, deposit of green tartar on the teeth, metallic taste in the mouth, anemia, premature old age, respiratory and degenerative diseases.	Bench molders (foundry); blowers-out (zinc smelting); brass foundries; braziers; bronzers; chargers (zinc smelting); core makers; floor molders (foundry); galvanizers; junk-metal refiners; luters (zinc smelting); pourers (brass foundry); welders; zinc smelters.
14. Carbon diox- id.	Anemia, cyanosis, headache, drowsiness, vertigo, tinnitus, and general nervousness.	Alkali-salt makers; blacksmiths; boiler-room workers; brass foundries; brewers; brick burners; caisson workers; carbon-dioxid makers; charcoal burners; drying-room workers (miscellaneous); fertilizer makers; furnace workers; lime burners; limekiln chargers; miners; pottery workers; sewer workers; silo workers; soda makers; starch makers; sugar refiners; vinters; white-lead makers; yeast makers.
15. Carbon di- sulphid.	Headache, pain in the extremities, trembling, deafness, reduction of the reflexes, acceleration of the heart's action, nausea, digestive trouble, emaciation, disturbance of sense of vision, excitement and violent temper followed by depression, hyperstimulation of sexual instinct, later its abnormal decline, chronic dementia.	Ammonium-salts makers; artificial-silk makers; asphalt testers; carbon-disulphid makers; celluloid makers; cementers (rubber shoes); cement mixers (rubber); dry cleaners; driers (rubber); enamellers; glue workers; insecticide makers; match-factory workers; oil extractors; paint makers; paraffin workers; putty makers; reclaimers (rubber); smokeless-powder makers; sulphur extractors; tallow refiners; vulcanizers.

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
16. Carbon mon- oxid.	Headache (usually frontal), dizziness, sense of fullness of the head, fatigue, nausea, general weakness, polycythemia.	Acetylene makers; bakers; bisque-kiln workers; black-smiths; blockers (felt hats); boiler-room workers; brass founders; cable splicers; calico printers; carbide makers; celluloid makers; charcoal burners; charges (zinc smelting); chimney sweepers; coal-tar workers; coke-oven workers; copper smelters; core makers; drying-room workers (miscellaneous); enamellers; felt-hat makers; filament makers (incandescent lamps); finishers (incandescent lamps); flangers (felt hats); flue cleaners; foundry workers; furnace workers; garage workers; gas (illuminating) workers; glost-kiln workers; incandescent-lamp makers; kiln tenders; laundry workers; lead smelters, lime burners; limekiln chargers; mercury smelters; miners; patent-leather makers; phosgene makers; pottery workers; pressers; refiners (metals); sealers (incandescent lamps); silver melters; singers (cloth); soda makers; stockers; teasers (glass); temperers; tabulators (incandescent lamps); zinc smelters.
17. Chlorid of lime.	Irritating cough, inflammation of upper air passages, difficulty of breathing, bronchitis, asthma, sometimes hemoptysis, conjunctivitis, lachrymation, hyperhidrosis, burning eruption on the skin.	Bleachers; chlorid of lime makers; chloroform makers; disinfectant makers; dye makers; laundry workers; tannery workers.
18. Chlorin.	Pallid countenance, emaciation, decayed teeth, bronchial irritation and asthma, gastric disturbances, irritation of the skin, chloracne.	Alkali-salt makers; beatermen (paper and pulp); bleachers; broom makers; calico printers; chlorid of lime makers; chlorin makers; disinfectant makers; dye makers; laundry workers; phosgene makers; photographic workers; sulphur-chlorin makers; zinc-chlorid makers.
19. Chlorodinitrobenzol.	See Nitrobenzol.	
20. Chloronitrobenzol.	See Nitrobenzol.	
21. Chromium compounds.	Pitlike, phagedenic ulcers, very difficult to heal and very painful, perforation of the nasal septum at the cartilaginous portion, irritation of the conjunctiva, small areas of inflammation in the lungs, inflammation of the kidneys, chronic gastritis, anemia.	Artificial-flower makers; battery (dry) makers; bleachers; calico printers; candle (colored) makers; chromium workers; color makers; compounders (rubber); dye makers; dyers; enamellers; glass mixers; glaze dippers (pottery); glaze mixers (pottery); ink makers; linoleum colorers; lithographers; match-factory workers; mixers (rubber); mordanters; paperhangers; pencil (colored) makers; photo-engravers; photographic workers; press-room workers (rubber); rubber-tire builders; rubber washers; rubber workers; tannery workers; vulcanizers; wall-paper printers; wax-ornament workers; wood stainers.
22. Cyanogen compounds.	Headache, vertigo, unsteadiness of gait, nausea, loss of appetite, disturbance of gastric and intestinal functions, slowing of the pulse, albuminuria.	Acid dippers; ammonium-salts makers; black-smiths; blast-furnace workers; browners (gun barrels); calico printers; case hardeners; celluloid makers; dye makers; electroplaters; fulminate mixers; fumigators; gas (illuminating) workers; gas purifiers; gold refiners; photographic workers, picklers; silver refiners; tannery workers, temperers.
23. Dimethyl sulphate.	Strongly corrosive effect on the skin and mucous membranes, hoarseness, lachrymation, conjunctivitis, edema, photophobia.	Dimethyl-sulphate makers; dye makers; perfume makers.
24. Dinitrobenzol.	See Nitrobenzol.	
25. Gasoline.	See Naphtha.	
26. Hydrochloric acid.	Irritation of mucous membranes; conjunctivitis; coryza; pharyngeal, laryngeal, and bronchial catarrh; dental caries.	Acid dippers; acid finishers (glass); acid mixers; acid recoverers; acid transporters; alkali-salt makers; ammonium salts makers; anilin makers; battery (dry) makers; calico printers; camphor makers; carbolic-acid makers; carbonizers (shoddy); cartridge dippers; dye makers; dyers; enamel makers; fertilizer makers; galvanizers; glass finishers; glass mixers; glue workers; hydrochloric; acid makers; jewelers; petroleum refiners; picklers; pottery workers; reclaimers (rubber); rubber workers; shoddy workers; solderers; sulphur-chlorid makers; tanners; vignettters; zinc-chlorid makers.

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
27. Hydrofluoric acid.	Intense irritation of the eyelids and conjunctiva, coryza, bronchial catarrh with spasmodic cough; ulceration of the nostrils, gums, and oral mucous membrane; painful ulcers of the cuticle, erosion and formation of vesicles, suppuration under the finger nails.	Antimony fluorid extractors; art-glass workers; bleachers; dyers; etchers; fertilizer makers; glass finishers; silicate extractors.
28. Lead and its compounds.	Sallow, pale, yellowish hue of the skin; metallic taste, nausea, anorexia, constipation, lead line, asthenia, lassitude, headache, arthralgias and neuritis, weakness of grip, tremors of fingers and tongue; lead paralyses, especially of muscles used most; atrophy of optic nerve.	Acid finishers (glass); amber workers; art-glass workers; artificial-flower makers; babbitters; battery (dry) makers; bench molders (foundry); blacksmiths; blooders (tannery); bookbinders; bottle-cap makers; brass foundlers; brass polishers; braziers; brick burners; brickmakers; bronzers; browners (gun barrels); brush makers; buffers (rubber); burners (enameling); cable makers; cable splicers; calico printers; canners; cartridge makers; celluloid makers; chargers (zinc smelting); color makers; colorers (white) of shoes; compositors; concentrating-mill workers (lead and zinc); cut-glass workers; cutlery makers; decorators (pottery); diamond polishers; dye makers; dyers; electroplaters; electrotypers; embroidery workers; emery-wheel makers; enamel makers; file cutters; filers; floor molders (foundry); galvanizers; glass finishers; glass mixers; glass polishers; glaze dippers (pottery); glaze mixers (pottery); glost-kiln workers; gold refiners; grinders (metals); grinders (rubber); heater boys (riveters); imitation-pearl makers; incandescent-lamp makers; insecticide makers; japan makers; jewelers; junk-metal refiners; labelers (paint cans); lacquer makers; lead burners; lead-foilmakers; lead miners; lead-pipe makers; lead-salts makers; lead smelters; linoleum makers; linotypers; linseed-oil boilers; lithographers; lithotransfer workers; match-factory workers; mirror silverers; mixers (rubber); monotypers; musical-instrument makers; nitric-acid workers; nitroglycerin makers; painters; paint makers; paint removers; paper-hangers; patent-leather makers, petroleum refiners; photograph retouches; pipe fitters; plumbers; polishers; pottery workers; printers; putty makers; putty polishers (glass); reclaimers (rubber); red-lead workers; refiners (metals); riveters; roofers; rubber workers; sagger makers; sandpaperers (enameling and painting auto bodies, etc.); screen workers (lead and zinc smelting); sheet-metal workers; shellac makers; shot makers; slip makers (pottery); slushers (porcelain enameling); solderers; stainers (shoes); steel engravers; stereotypers, storage-battery makers; sulphuric-acid workers; table turners (enameling); tannery workers; temperers; tile makers; tin-foil makers; tinnerns; toy makers; transfer workers (pottery); tree sprayers; type foundlers; typesetters; wallpaper printers; welders; white-lead workers; wood stainers; zinc smelters.
29. Mercury and its compounds.	Ptyalism; swelling, inflammation, and bleeding of the gums; blue line on the gums, rodent ulcers, pallor, mercurial tremor, digestive disturbances, localized white spots in the mucosa surrounded by pale blue or reddened area, general weakness of the hand and digital extensors, foul breath, corrosion of the teeth, furunculosis, sleeplessness and depression or drowsiness and apathy, loss of energy and initiative.	Artificial-flower makers; battery (dry) makers; blowers (felt hats); bronzers; browners (gun barrels); brushers (felt hats); cap loaders; carroters (felt hats); cartridge makers; chlorin makers (electrolytic); color makers; coners (felt hats); dentists; detonator cleaners; detonator fillers; detonator packers; devil operators (felt hats); dye makers; Edison storage battery workers; explosive workers; felt-hat makers; fireworks makers; fulminate mixers; fur handlers; fur preparers; gold refiners; hardeners (felt hats); incandescent-lamp makers; jewelers; mercurial-vapor-lamp makers; mercury bronzers; mercury miners; mercury-salts workers; mercury smelters; mercury-solder makers; mercury-still cleaners; mirror silverers; mixers (felt hats); paint makers; photographic workers; primers (explosives); refiners (metals); sizers (felt hats); sole-stitchers (Blake machine); starters (felt hats); steel engravers; stiffeners (felt hats); storage-battery makers; taxidermists; thermometer makers; water gilders; zinc-electrode makers.

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
30. Methyl alcohol.	Headache, nausea, abdominal cramps, ringing in the ears, muscular prostration, insomnia, delirium, difficulty of breathing, inflammation of the throat and mucous membrane of the air passages, conjunctivitis, serious affections of the retina and optic nerve resulting in blindness, fatty degeneration of the liver.	Aldehyde pampmen; art-glass workers; artificial-flower makers; artificial-silk makers; bookbinders; bronzers; brush makers; calico printers; celluloid makers; cementers (rubber shoes); dimethylsulphate makers; dry cleaners; dryers (felt hats); dye makers; explosives workers; feather workers; felt-hat makers; filament makers (incandescent lamps); fitters (shoes); furniture polishers; gilders; hardeners (felt hats); incandescent-lamp makers; ink makers; japan makers; lacquer makers; lasters (shoes); linoleum makers; millinery workers; mottlers (leather); painters; paint makers; patent-leather makers; perfume makers; photo-engravers; photographers; polishers; shellac makers; shoe-factory operatives; shoe finishers; soap makers; stiffeners (felt hats); stitchers (shoes); type cleaners; upholsterers; varnish makers; vulcanizers; wood alcohol distillers; woodworkers.
31. Methyl bromid.	Vertigo, headache, staring look, pallor of the skin, retarded pulse, constipation, excitability, trembling.	Antipyrin makers; dye makers.
32. Naphtha.	Headache, vertigo, nausea, vomiting, dyspnea, palpitation, insomnia, hysteria.	Bronzers; chauffeurs; degreasers (fertilizer, leather); dyers; furniture polishers; garage workers; gilders; metal-polish makers; painters; petroleum refiners; polishers; rubber workers; shoe finishers; waterproof-cloth makers; woodworkers.
33. Nitranilin.	<i>See Anilin.</i>	
34. Nitrobenzol and other nitro compounds of benzol and its homologues.	Icteric skin which gradually becomes cyanotic, methemoglobin formation, general debility, anemia, presence of hemaphorpyrin, albumin, and sometimes free poison in the urine; skin eruptions, visual disturbances, dyspnea, odor of bitter almonds in breath.	Anilin makers; dye makers; explosives workers; perfume makers; smokeless-powder makers; soap makers.
35. Nitroglycerin.	Severe headache, vertigo, nausea, paralysis of the muscles of the head and eyes as well as of the lower extremities, cyanosis, reddening of the countenance, burning in the throat and stomach, disturbances of digestion, trembling, neuralgia, colic, retarded respiration and heart action, obstinate ulcers under nails and on the finger tips, eruptions on the plantar aspect of the feet and interdigital spaces, with extreme dryness and formation of fissures.	Explosives workers; nitroglycerin workers; shell fillers.
36. Nitronaphthalene.	<i>See Nitrobenzol.</i>	
37. Nitrous gases and nitric acid.	Irritation of air passages, cough, labored respiration, inflammation of the eyes, corrosion of the teeth, erosion and perforation of nasal septum.	Acid dippers; acid mixers; acid recoverers; acid transporters; anilin makers; artificial-leather makers; bleachers; carroters (felt hats); cartridge dippers; celluloid makers; dimethyl-sulphate makers; dippers (gun-cotton); enamellers; etchers; explosives workers; felt-hat makers; fertilizer makers; fur preparers; galvanizers; glue workers; gun-cotton dippers; guncotton wringers; imitation pearl makers; incandescent-lamp makers; jewelers; lithographers; miners; mordanters; nitrators; nitric-acid workers; nitroglycerin workers; photo-engravers; picklers; picric-acid makers; refiners (metals); soda makers; steel engravers; sulphuric-acid workers; towermen (sulphuric acid); wringers (gun-cotton).

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom. Condition. or Disease to Look For	Occupations Which Offer Such Exposure
38. Petroleum.	Inflammation of the skin, acne, suppurating ulcers; papilloma; numbness and irritation of the Schneiderian membrane; headache and sensory disturbances; affections of the respiratory organs.	Brownners (gun barrels); feather workers; furniture polishers; lampblack makers; millinery workers; oil-flotation-plant workers; oil-well workers; paraffin workers; petroleum refiners; temperers.
39. Phenol.	Erosion of the skin, eczema, irritation of respiratory organs, digestive disturbances, symptoms of degeneration of the blood, emaciation, nephritis, gangrene, icterus.	Bakelite makers; calico printers; coal-tar workers; dye makers; dyers; etchers; gas (illuminating) workers; gas purifiers; lampblack makers; picric acid makers; rubber workers; smokeless powder makers; stillmen (carbolic acid); surgical-dressing makers; wood preservers.
40. Phenyl hydrazin.	Vesicular eruptions on the skin with itching and burning, diarrhea, loss of appetite, granular degeneration of the blood corpuscles, formation of methemoglobin, a sense of general malaise.	Antipyrin makers; dye makers.
41. Phosgene.	Destruction of lung tissue, emphysema and edema, myocardial insufficiency due to the emphysema, pleural thickening and adhesions, chronic bronchitis, mild diffuse bronchiectasis, nocturnal dyspnea, polycythemia.	Dye makers; phosgene makers.
42. Phosphorus.	Inflammation and sclerosis of the bones and of the periosteum, necrosis of the bones of the jaw, swelling and ulceration of the gums and buccal membrane, loosening and falling out of the teeth, suppuration and destruction of jawbone with fistulous channels burrowing through the cheek, meningeal inflammation, brittleness of bones, digestive disturbances, emaciation.	Boneblack makers; brass foundries; fertilizer makers; fireworks makers; insecticide makers; match-factory workers; phosphate-mill workers; phosphor-bronze workers; phosphorus-compounds makers; phosphorus extractors.
43. Phosphuretted hydrogen.	Oppressed feeling in the chest, headache, vertigo, tinnitus aurium, general debility, loss of appetite, great thirst.	Acetylene makers; ferrosilicon workers; phosphorus extractors; phosphorus (red) makers.
44. Picric acid.	Itching, inflammation of the skin, vesicular eruptions, yellow pigmentation of epidermis and conjunctiva, inflammation of buccal mucous membrane, digestive disturbances, vertigo, jaundice, nasal catarrh, nephritis.	Dye makers; dyers; explosives workers; photographers; picric acid makers; shell fillers; smokeless-powder makers.
45. Sulphur chlorid.	Symptoms are due to the combined effects of chlorin, hydrochloric acid and sulphur dioxide. Sulphur chlorid when in contact with moisture reacts with water to form these products.	Rubber-substitute makers; vulcanizers.

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
46. Sulphur dioxide.	Irritation of the mucous membrane of respiratory organs and eyes, spasmodic cough, bronchial catarrh, digestive disturbances, bloodtinged mucous.	Alkali-salt makers; blast-furnace workers; bleachers; brass foundries; brick makers; broom makers; carbolic acid makers; chambermen (sulphuric acid); chargers (zinc smelting); copper smelters; dye makers; fertilizer makers; flue cleaners; fruit preservers; fumigators; galvanizers; glue workers; lead smelters; mercury smelters; oil-flotation-plant workers; petroleum refiners; pottery workers; pyrites burners; refiners (metals); rubber workers; storage battery makers; sugar refiners; sulphite cooks; sulphur burners; sulphurizers (hops and malt); sulphuric-acid workers; tannery workers; towermen (sulphuric acid); zinc smelters.
47. Sulphuretted hydrogen.	Headache, debility, vertigo, nausea, disturbances of digestion, sallow complexion and emaciation, slowing of the pulse, conjunctival catarrh, tendency to the formation of boils.	Alkali-salt makers; artificial-silk makers; blast-furnace workers; bronzers; cable splicers; celluloid makers; dye makers; fertilizer makers; flax-rettery workers; gas (illuminating) workers; gas purifiers; glue workers; match-factory workers; miners; oil-flotation-plant workers; petroleum refiners; pyrites burners; sewer workers; soda makers; sodium sulphid makers; starch makers; sugar refiners; tannery workers.
48. Sulphuric acid.	Inflammation of respiratory organs, injury to teeth through softening of the dentine, chronic catarrh.	Acid dippers; acid finishers (glass); acid mixers; acid recoverers; acid transporters; ammonium-salts makers; ammonium-sulphate makers; artificial-leather makers; beta-still operators (beta naphthol); burnishers (iron and steel); calico printers; carbolic acid makers; carbonizers (shoddy); cart-ridge dippers; celluloid makers; chambermen (sulphuric acid); dimethyl-sulphate makers; dye makers; explosives workers; felt-hat makers; fertilizer makers; galvanizers; glass finishers; guncotton dippers; hydrochloric acid makers; jewelers; linoleum makers; mercerizers; nitrators; nitric-acid makers; nitroglycerin makers; oil-flotation-plant workers; patent-leather makers; petroleum refiners; phosphorus-evaporating machine operators; picklers; picric acid makers; reclaimers (rubber); salt extractors (coke-oven byproducts); shoddy workers; storage-battery makers; sulphuric-acid workers; tallow refiners; tannery workers; temperers; towermen (sulphuric acid); wire drawers.
49. Tar.	Tar itch, diffuse acne, eczema or psoriasis, loss of appetite, nausea, diarrhea, headache, numbness, vertigo, albuminuria, edema, ischuria, conjunctivitis, bronchitis.	Battery (dry) makers; briquet makers; brush makers; chimney sweepers; coke-oven workers; cord makers; flue cleaners; gas (illuminating) workers; insulators; paint makers; paraffin workers; pavers; petroleum refiners; roofers; roofing-paper workers; still (coal-tar) cleaners; tar workers; wood preservers.
50. Tetrachlorethane (acetylene tetrachlorid).	Abnormal sense of fatigue, profuse perspiration, general discontent and grouching, inability to concentrate, nocturia, slight polyuria, dreaming, headache, vertigo, nervousness, insomnia, loss of appetite, constipation, diarrhea, gas in stomach, general abdominal pain, nausea, eructations of gas, vomiting, loss of weight, jaundice, enlarged liver, bile in the urine, abdominal tenderness, increase of mononuclear cells, appearance of many immature large mononuclears, elevation in the white count, slight anemia, slight increase in number of platelets.	Airplane-wing varnishers; artificial-silk makers; tapers (airplanes).

POISONS, THEIR SYMPTOMS AND OCCUPATIONS OFFERING EXPOSURE—*Continued*

Health Hazard	Symptom, Condition, or Disease to Look For	Occupations Which Offer Such Exposure
51. Trinitro-toluol.	Nose and throat irritation, obstinate cough, bluish color of the lips and lobes of the ears, yellowing of the whites of the eyes, expectoration of yellow mucous, discoloration—a mixture of lividity and jaundice, rash on the skin, shortness of breath, anemia, palpitation of the heart, bile-stained urine, rapid weak pulse.	Explosives workers; shell fillers.
52. Turpentine.	Irritation of the mucous membrane of the eyes, nose, and upper air passages; cough, bronchial inflammation; salivation; giddiness, headache, irritation of the kidneys, odor of violets in urine, severe irritation of the skin, eczema and hardening of the epidermis.	Art-glass workers; cable splicers; calico printers; camphor makers; cementers (rubber shoes); decorators (pottery); dry cleaners; dye makers; enamelers; enamel makers; feather workers; furniture polishers; japan makers; lacquer makers; linoleum makers; lithographers; millinery workers; painters; paint makers; patent-leather makers; printers; rubber workers; sealing-wax makers; shellac makers; transfer workers (pottery); turpentine extractors; varnish makers.

*DUSTY TRADES*⁵⁴

Dust is a great enemy of the workman. Much ill health is caused by the inhalation of dust, some of which is also injurious when ingested and some of which is irritating to the skin, eyes, or exposed mucous membranes. Dust of all kinds, both organic and inorganic, is met with in various industries. Organic dust is usually less irritating and dangerous than inorganic dust, which becomes harmful particularly when the particles are sharp and therefore irritating, although the chemical nature of the dust may be even more important than its physical character. The principal trades and occupations in which excessive amounts of dust are found are: the abrasive industry; all forms of grinding and many processes of polishing and cleaning; the textile industries; in the lead, copper, and iron trades irritating and poisonous dusts are raised; also in pottery works and masonry, and in the handling of leather, skins, feathers, wool, cotton, wood, paper, tobacco, cement, cutting diamonds and other precious stones; emery, glass, horn, bone and shell, grain and flour, etc. The amount and nature of the dust vary greatly. The amount may be very great;⁵⁵ thus Hesse found in one cubic meter of air the following amounts of dust in the occupations named:

⁵⁴ See also page 845.⁵⁵ C.-E. A. Winslow, L. Greenburg, and D. Greenburg, "The Dust Hazard in the Abrasive Industry," Rep. No. 530, *Pub. Health Rep.*, May 30, 1919, p. 1171; also F. G. Miller, and H. F. Smith, "The Dust Hazard in Certain Industries," *J. Am. M. Ass.*, 1918, 70: 599.

	Milligrams
Felt hat factory.....	175
An old flour mill.....	48
A new flour mill.....	4
Mechanical knitting.....	3
Sculpturing	9
A paper factory.....	4-25
Iron works	72-100
A coal mine.....	14
A living room.....	0

The kinds of dust vary greatly in their hygienic significance. Some are poisonous, some act as mechanical irritants. The principal poisonous dusts found in the industries are lead, mercury, arsenic, manganese, and zinc; less often substances from tobacco, wood, dyes, and chemical works. The dust particles which act by mechanical irritation are especially the hard, irregular particles with sharp edges from iron, steel, and other metals; from granite or basalt; while those from coal, chalk, cement, marble, and plaster of Paris are less irritating. Dust containing silica is especially harmful because it produces lesions resembling tuberculosis.

Some dust is especially irritating to the conjunctiva, as wood dust or arsenic. Certain kinds of dust are prone to cause chronic catarrhal inflammation of the upper respiratory passages, while dust containing specific microorganisms such as anthrax may lead to acute pneumonia (wool-sorter's pneumonia).

Effects of Different Kinds of Dust.—The effect depends primarily upon the character of the dust, and secondarily on the duration and amount of exposure. The most injurious types of dust were formerly supposed to be those with hard, sharp corners and insoluble particles, but we now know that the injurious effect is due to the chemical nature rather than the physical structure of dust. The effects are also related to the size and number of the particles. So far as silica is concerned, Collis⁵⁶ sums up our present knowledge in a sentence when he states that “the noxious influence of silica is now accepted to be a chemical rather than a mechanical one.” In arsenical and cobalt dusts, which are believed to cause serious inflammatory changes and even lead to cancer, the chemical nature is the more important. On the other end of the scale come most organic dusts, as animal and vegetable fibers from the textile trades, which seem not to affect the lungs at all, although they may cause trouble in the upper respiratory passages. The comparative harmlessness of some dust is well testified by the continuance of workers exposed to the tremendously dusty atmosphere that occurs in and about cement plants. While this is true for tuberculosis, recent studies have shown that such exposure increases the incidence of other respiratory diseases. Coal dust also appears to be relatively bland; in fact, coal miners are rela-

⁵⁶ *J. State Med., Roy. Inst. Pub. Health*, 1925, 33: 183.

tively free from pulmonary tuberculosis. Indeed coal dust has been administered therapeutically by inhalation in the treatment of tuberculosis. Statistics have been brought forward to show that there is a higher tuberculosis mortality rate among miners' families than among the miners themselves. Even with marked exposure, coal dust usually takes from twenty to thirty years to produce changes sufficient to cause evident symptoms. We have no explanation of this paradox.

It is believed that calcareous dusts, instead of being harmful, have a protective influence on tuberculosis. Fisac⁵⁷ reports that among 40,824 deaths from tuberculosis, only seventeen, or 0.41 per cent, were in lime and gypsum burners. He also reports that among 400 employees in a gypsum factory no death from tuberculosis occurred in seventeen years. There is so much similar evidence that inhalation of lime dust or residence in and about lime kilns has been recommended as a therapeutic measure in tuberculosis. Gardner and Dworski⁵⁸ studied the inhalation of marble dust in guinea-pigs, and found that the calcium carbonate particles are readily absorbed, presumably as the bicarbonate, and hence produce no injurious effects; but if siliceous particles are present, they cause harm. Willis⁵⁹ emphasizes the importance of the time factor, for he found that guinea-pigs that were made to inhale coal dust in large amounts at frequent intervals for a year did not accumulate as much coal dust or fibrosis in their lungs as other animals that had lived two years under laboratory conditions without special exposure to dust. This author confirms the contention of Beattie, Haythorn and others that the fibrosis which develops in response to the presence of certain dusts tends to limit the spread of the infectious process, and should in this sense be regarded as protective.

Dust and its effects is considered in detail in the section on Air.

Silicosis.—Dust containing silica appears to be the most irritating and destructive of the "non-poisonous" dusts. This was formerly explained by the fact that the particles of silica are sharp and hard, but it now appears that silica causes a special, even specific tissue reaction. There are three stages in the development of simple silicosis: (1) the provocation of "dust-cells"; (2) the accumulation of these cells in the lymphatics and their collection into "pseudotubercles," and (3) the fibrosis of obstructed lymphatics and of pseudotubercles. The early lesions of silicosis and pulmonary tuberculosis are much alike.

Silica does not seem to harm the dust cells, which pick it up, for it prevents their autolysis or digestion. Hence, they accumulate in the lymph channels, which they eventually fill up and block. Fibrosis then takes place. Although all dusts tend to "drift" towards the pleura, cells laden with silica have a special tendency to agglutinate there in masses. This aggregation of

⁵⁷ Quoted by Maendl, *Ztschr. f. Tuberk.*, 1921, 35: 184.

⁵⁸ *Am. Rev. Tuberc.*, 1922, 6: 782.

⁵⁹ *Ibid.*, 1922, 6: 798.

dust-filled cells is called pseudotubercles.⁶⁰ Landis⁶¹ states that silica dust may bring about a complete crippling of the worker in as brief a period as from two to eight years if the exposure is sufficiently intense.

Nomenclature.—Persons exposed to excessive amounts of dust for long periods of time suffer from a general condition known as *pneumokoniosis*; when due to coal dust the condition is known as *anthracosis*; when due to stone dust, *siderosis* or *chalicosis*; when due to vegetable fibers such as cotton, *byssinosis*. We also hear of stone cutters' phthisis and potters' rot, etc.

WOOD DUST

It is well known that workers in wood are subject to the mechanical effects of ordinary sawdust, which is moderately irritating. Workers with boxwood, teak, and sequoia (redwood) are subject also to the general poisonous effects of alkaloids and other substances contained in these woods which may have more marked general effects, especially on the circulation and, still more frequently, marked local effect on the mucous membranes and the skin. In 1902 Young observed that men working with Maracaibo boxwood complained of dryness of the throat and inflammation of the eyes which lasted two or three days. This wood is used in the making of rulers. Oliver notes that joiners that saw and chip sequoia wood suffer with symptoms resembling a bad cold in the head and chest; a tolerance seems to be established except by men who are liable to bronchitis and asthma. Wounds caused by splinters of the wood invariably suppurate and do not heal readily. Oliver found that rats were also susceptible to sequoia sawdust. They suffer from a running at the nostrils.

Certain kinds of wood have a bad reputation among joiners. Some sawdusts are more irritating than others, probably from the large amount of inorganic matter they contain. A West African boxwood from which shuttles are made causes headache, coryza, excessive secretion of tears, and attacks of asthma. These woods contain alkaloids, glucosids, and other extractives. Workers in teakwood occasionally suffer from dermatitis.

General Principles of Prevention.—Much of the dust raised in industrial processes may be limited by improvements in machinery or preventive devices. Sometimes the dust may be kept down by moisture, sprays, or even conducting the work under water when practicable. Certain dusty operations should be conducted in inclosed hoods or special cabinets so as to confine the dust and thus protect the work people, or the dust may be removed by suction fan devices. Good ventilation diminishes the danger very much. When workmen are compelled to stay in dusty atmospheres they should wear respiratory masks, and the number of persons thus exposed should be reduced to a minimum. Some exceedingly dusty processes, such as cleaning castings with a sand blast, demand the wearing of a protective headgear. Many workmen

⁶⁰ A. Mavrogordato, *Pub. So. African Inst. Med. Res.*, March, 1922.

⁶¹ *Am. Rev. Tuberc.*, 1922, 6: 766.

prefer taking chances to wearing uncomfortable respirators. ¹ For a further discussion of dust, smoke, etc., see Section VII.

THE TEXTILE INDUSTRIES

The manufacture and handling of cotton, linens, silk, and jute have received an unenviable reputation as dangerous occupations, despite the fact that these industries need not in themselves be particularly unhealthy occupations. The textile industries illustrate several points in the diseases of

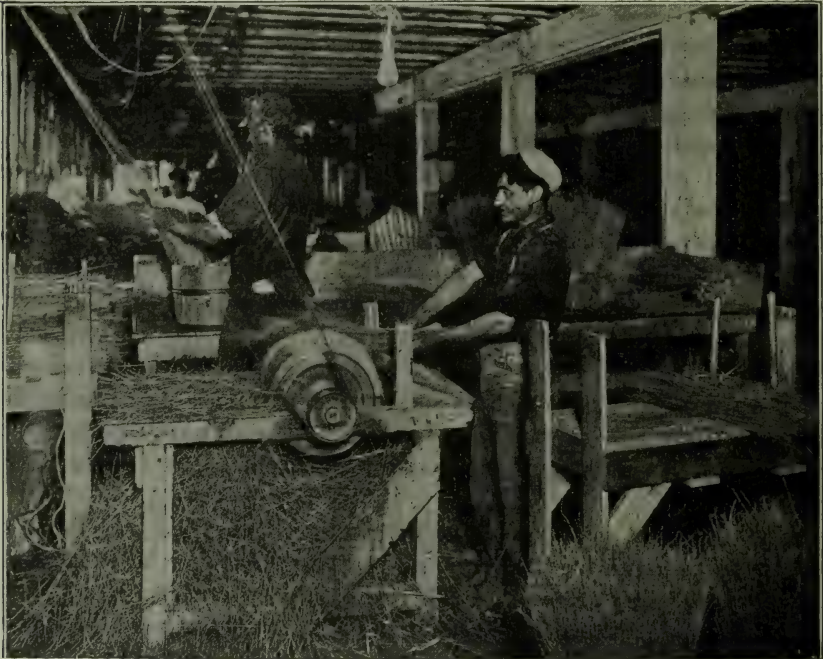


FIG. 141.—A VERY DUSTY TRADE.

Drum with nails which combs out the small pieces of broom corn.
(Mass. State Board of Health.)

occupation. One is that an entire industry should not be condemned because one of its processes is attended with a certain amount of danger. The other is that the risks to health may be prevented or greatly ameliorated. General improvement in the sanitary conditions of textile mills is one of the promising signs of material advancement in industrial hygiene.

The principal conditions which affect health in the textile industries are: The working in a dusty atmosphere which is often kept very moist and usually very warm in order to keep the fiber pliable and workable. The humidity and temperature may be regulated, and by efficient systems of ventilation their ill effects may be minimized or even neutralized. The dust may also be lessened, and the dusty processes isolated.

Much dust is raised during the opening and emptying of the bales of the raw material. This is avoided in the better mills by the use of machinery. Most dust is raised during the process of "carding"; some during "roving," "spinning" the yarn, and "winding" it; and also considerable during "weaving." In linen factories the "hecklers," that is, the men who dress and sort the rough flax (converted into tow by having been passed through a machine), are exposed to considerable amounts of dust and suffer from dryness of the throat and bronchitis, attended by cough and shortness of breath. In the manufacture of sacks, twine, and carpets from jute the processes that are extremely dusty are the preparing and spinning. The dust given off by jute is irritating.

Humidity in Textile Mills.—Working in an atmosphere which is excessively moist and frequently very warm, and, further, containing an excessive amount of organic dust, subjects the workmen to artificial and unnatural conditions which cannot be conducive to health.⁶² Presumably the heat and moisture predispose to rheumatic states and inflammatory conditions of the respiratory tract which are aggravated by the irritation of the fibrous dust. Collis and Greenwood⁶³ found, however, that weavers have a much lower blood-pressure and a more rapid pulse than normal individuals, that they are run down in general health, anemic and suffer from indigestion. This is one of the few industries where low blood-pressure is found as a result of occupation.

The humidity of the air is an important factor in the manufacture of textile fabrics. The former supremacy of certain English localities as textile centers was due to the naturally favorable climatic conditions. This led to the adoption of artificial means of increasing the moisture of the air in mills less favorably located. When the fibers contain a certain proportion of moisture they are elastic and cling closely together, and may be carded, combed, drawn out, and spun into yarn, and woven into cloth more easily than when dry. When the fiber is moist the work in all these processes runs better and smoother; finer grades of goods may be made from the same stock; there is less waste, and the machines may be run at higher speed with less attention from the operators. Yarn spun from dry fiber is harsh and kinky; it does not retain its twist and breaks easily; furthermore, there is more dust and "fly" which menace the health and comfort of the work people.

The temperature and humidity most suitable for obtaining the best results in each process with material of different character and quality have been determined with considerable care, and, generally speaking, have been found to be such as would not be prejudicial to health. Despite this knowledge excessive moisture and high temperatures injurious both to health and to the processes of manufacture are found in some mills—another example of inefficiency through ignorance.

⁶² For effect of heat and humidity upon health see pages 816 to 830.

⁶³ *The Health of the Industrial Worker*, 1921.

The necessary humidity in textile mills is obtained by a number of different devices. The methods which depend upon the introduction of moisture directly into the mill rooms are more objectionable than the humidification of the air forced into the rooms by some system of mechanical ventilation. In any case, the water used to moisten the air should be clean and free from odor or objectionable impurities.

The simplest method of producing artificial humidity in mill rooms is by sprinkling water upon the floor and trusting to natural evaporation. This method, known as "degging," was widely practiced at one time, and is still occasionally found in some foreign mills. Degging was replaced by shallow channels in the floor for the water, or by the placing of pans of water about the room, and later by introducing steam directly into the room. Steam is objectionable for the reason that it unduly increases the temperature. The modern types of humidifying apparatus depend upon moistening the air by passing it over water surfaces or through water curtains. The spray moisteners are made in a large variety of patterns. Some are constructed on the principle of the common household atomizer.

In Massachusetts there is a law regulating the amount of humidity and temperature in the textile mills which is based upon the English schedule contained in the Weaver's Act of 1870. The conditions in Massachusetts, however, are so different from those found in England, especially in the summer time, that the schedule has not been found practical. Much of the ill effects in the textile industries may be neutralized by good ventilation, abundant air space, cleanliness, sufficient light, and the use of improved machinery. Special rooms should be provided for the clothes, in order that the moist garments may be changed for dry ones before the work people go into the open air, thus avoiding the chilling effects of damp garments.

MINING

Mining is one of the dangerous and unhealthy occupations. The dust, the unnatural conditions under which the miner is compelled to work underground, the high temperature and humidity, the poor air, and sometimes exposure to poisonous gases all conspire to make this occupation one attended with unusual risks.

Poor or deficient illumination may cause a disease of the eyes known as nystagmus. Pure water, the disposal of sewage and wastes and other problems of hygiene and sanitation do not differ materially from those discussed in other parts of this volume. The unsatisfactory methods for disposal of feces often found in mines favor the spread of hookworm and other parasites. To this must be added the danger of accidents and explosions.

The magnitude of this form of labor is not ordinarily realized.

There are over one million miners in the United States, distributed as follows:

NUMBER EMPLOYED IN MINES IN THE UNITED STATES, 1923 *

Metal		Quarrying		Coal	
Copper	32,477	Cement rock	13,378	Bituminous	702,817
Gold, silver, miscellaneous	30,525	Granite	11,658	Anthracite	157,743
Iron	38,419	Limestone	46,325		
Lead, zinc	10,226	Marble	5,351		
Non-metallic mineral	11,632	Sandstone and bluestone	4,972		
		Slate	4,329		
		Trap rock	6,442		
Total	123,279		92,455		860,560

* Official reports of the U. S. Bureau of Mines, 1923.

The abnormal conditions of the air in mines are both physical and chemical. The air of mines, unless thoroughly ventilated, is still and stagnant with a high humidity and increasing temperature depending upon depth. The air is also apt to be dusty. The dust may be poisonous, as in the mining of ores containing lead, arsenic, zinc and mercury; or non-poisonous but irritating in varying degrees, such as dust from silica, quartz, limestone, coal, gypsum, or silicates. Siliceous dust causes the most trouble. The most dangerous gases commonly found in the air of mines are CO (white damp), hydrogen sulphid (stink damp), sulphur dioxide, methane, marsh gas or fire damp. The prevention of poisoning by the various toxic gases depends chiefly upon good ventilation or the use of adequate protective equipment. Those who find themselves in the presence of poisonous gases should try to keep calm, avoid hurry and get out with as little exertion as possible.

The conditions of mines have been greatly improved, especially through better systems of ventilation, through the use of safety lamps, through reduction of the amount of dust, the regulation of the hours of occupation, and devices to detect poisonous and explosive gases. The sanitation and cleanliness of mines have also shown development. As an illustration of some of the complications and difficulties of this subject, reference is made to the fact that moisture will prevent explosion in mines. Moisture was, therefore, introduced into some of the German mines with good results, so far as explosions are concerned, but the moisture favored the development of the hookworm larvæ and hence caused such a great increase in the amount of hookworm infection that it became necessary to seek other methods.

The effects of dust vary with the nature of the material mined. The harmful effects of silica and certain metallic dusts have been given above. Coal dust deserves special consideration. Although coal is a vegetable product the result largely of microbial action, fresh coal is free from microorganisms. Oliver points out that in some of the mining centers colliers not only suffer less from pulmonary tuberculosis than persons in other occupations, but that they also suffer unequally in different mining centers. Why this is so was a puzzle, until Collis⁶⁴ pointed out that it may be accounted for by the

⁶⁴ *J. Indust. Hyg.*, 1922, 4, Nos. 6 and 7.

varying amounts of silica dust to which miners are exposed. While the death rate from pulmonary tuberculosis in miners is in some places low, that due to non-tuberculous affections of the lungs is, comparatively speaking, high.

The prevention of dust and the elimination of silicosis among miners depends upon wet mining methods—wet drilling, wetting sides, roof and bottom, wetting muck and rock piles; and by the use of sprays and water blasts to allay the dust after blasting. It also depends upon good ventilation. All miners should be examined physically before employment and periodically thereafter with special reference to tuberculosis.

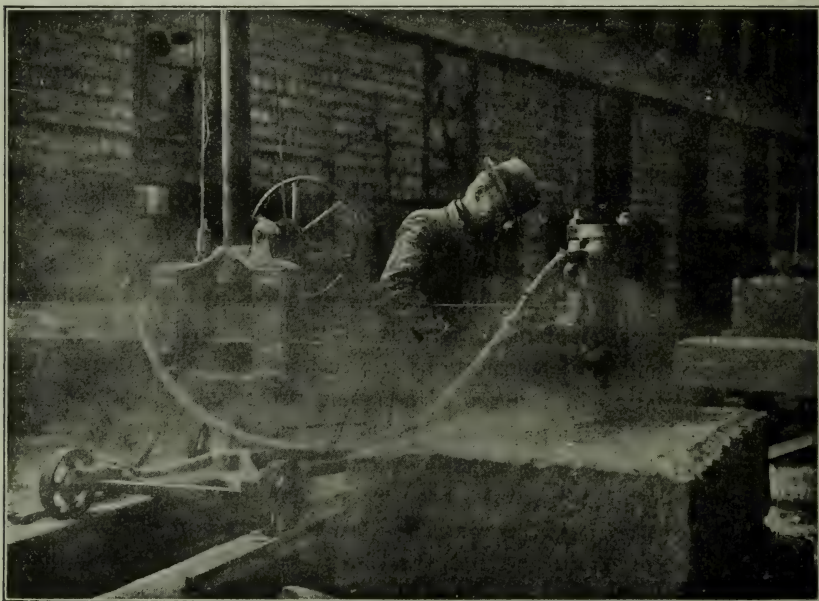


FIG. 142.—THE STONE INDUSTRY.

The workman is using a surfacing machine operated with compressed air. The strong blast of air keeps the granite clean, but gives rise to a great amount of dust. Of the mineral dusts granite is generally considered as most irritating.
(Mass. State Board of Health.)

DeCrocq speaks of the rarity of phthisis among Belgian coal miners. Arnold reports that in Germany tuberculous diseases are rare among coal miners and that there is a prevailing opinion that anthracosis is antagonistic to tuberculosis. Goldman attributed the freedom of the coal miner from pulmonary tuberculosis to an antiseptic action of the coal dust. See page 1264.

Other diseases to which coal miners are subject are "beat hand," as a consequence of using the pick and friction of the handle. The skin of the palm over the bases of the fingers of both hands, also the skin over the fleshy ball of the thumb and that of the other side of the hand, becomes extremely hard and horny. In addition to the enormous thickening of the epithelial layers of the skin there is inflammation of the subcutaneous connective tissue.

Occasionally suppuration takes place in the deeper layers of the hard skin. The suppurating areas are called "keens" by the miners. Beat hand is a painful affection and unfits the individual for work for some time. A similar condition sometimes occurs on the knees and elbows, hence the term "beat knee" and "beat elbow." Miners also frequently complain of backache, largely the result of the peculiar mode of sitting while at work. Dyspepsia, miner's nystagmus, and ankylostomiasis are other conditions to which miners are prone.

The student is referred to the splendid reports issued by the United States Bureau of Mines, particularly studies by R. R. Sayers and D. Harrington, for details concerning the problems of sanitation and hygiene with special application to the peculiar conditions found in mines. These reports may be had from the Superintendent of Documents, Government Printing Office, Washington, D. C.

MISCELLANEOUS

Excessive Heat.—In many trades workmen, more particularly firemen, stokers, workers in foundries and steel mills, are exposed to high degrees of heat. Edsall⁶⁵ has recently called attention to the ill effects of exposure to unusual degrees of heat. The symptoms are acute, violent muscle spasms. The acute effect may be heat-stroke and heat prostration; there may be nervous lesions such as focal meningitis, as well as more or less serious circulatory weakness, anemia, acute and chronic disturbances of digestion, acute and chronic nephritis. Respiratory diseases and skin lesions appear to be unduly frequent in persons exposed to high degrees of heat. There is more than a suspicion that cataracts, retinal and choroidal changes, or chronic conjunctival lesions are brought on in glass-blowers and perhaps also in iron puddlers and other persons whose eyes are exposed to very intense heat and light. De Schweinitz states that he can often tell whether men working at puddling furnaces are right-handed or left-handed by studying the effects of this exposure on their eye grounds.

Unnecessary Noise.—Unnecessary noise may become a nuisance, and under certain conditions is a menace to health, especially high-pitched sounds long-continued, which lead to deafness; hence the deafness of boiler makers and others is a true occupational disease. Siebenmann⁶⁶ and others have demonstrated that long-continued exposure to high-pitched sounds causes degenerative changes in the organ of Corti in the internal ear.

Noises also disturb rest and sleep, irritate the nervous organism, and induce unpleasant results. The susceptibility to noises varies greatly. Many unnecessary noises can be stopped in shop and street with a corresponding saving of energy and increase of efficiency. It is now realized that unnecessary noise represents misspent energy, and hence so much avoidable waste. Quiet

⁶⁵ *J. Am. M. Ass.*, 1908, 51: 1969.

⁶⁶ *Ueber gesundheitliche Schädigungen durch Lärm*, Basel, 1910.

zones at least should be established around schools, hospitals, churches, courts, lecture and music halls, etc.

COMMUNICABLE INFECTIONS

There are several infections to which workmen in certain industries are specially subjected. Of these the best known are: anthrax, or woolsorter's disease from hides and hair; and hookworm disease, or miner's anemia, from polluted soil; also glanders from horses. Tuberculosis sometimes results directly, but more often indirectly, from occupation. Infection occurs through wounds, from oil and in other ways.

Woolsorters' Disease.—Woolsorters' disease is an infection with *Bacillus anthracis*. The spores cling to the hides of animals that have died from the disease or have been slaughtered on account of it. Spores also remain attached to wool and horsehair and to pig's bristles used in brush-making. The infection may be taken in through the slightest scratch or any open wound or through inhalation of dust containing the spores, or may be ingested in the food. Woolsorters' disease most often appears in the wool-sorting, wool-combing, and spinning industries, in the manipulation of horsehair for stuffing chairs and mattresses, and the preparation of bristles for brush-making. Anthrax has also been met with in persons employed in tanyards and in warehouses that connect with docks. The subject is fully discussed by Legge in his Milroy lectures.⁶⁷

The prevention of anthrax is first and foremost a problem in animal husbandry which, in this country, comes under the purview of the Bureau of Animal Industry. Animals having anthrax should be killed and all anthrax carcasses should be buried, incinerated, or tanked in such a manner as to destroy the infection and prevent its dissemination. This is one of the questions for international sanitary agreement, for the wool from Persia, the hair and mohair from Asiatic Turkey, the horsehair from China, the bristles from Siberia, and the hides from India may carry the anthrax spores from these far-off lands and cause infection among our workmen. It is exceedingly difficult to disinfect hides so as to kill the anthrax spores without damaging the hides for commercial use. The methods of disinfecting hides, as well as wool, bristles, hair, etc., are discussed on page 403.

Glanders.—See page 395.

Hookworm Disease.—Miners are specially subject to hookworm disease. The parasite enters through the skin from the polluted soil of the mines. The outbreak which called attention to this danger was the epidemic which occurred among the workmen on St. Gothard's tunnel in 1892. Since then the disease has been called "miner's anemia." Gunn⁶⁸ found that from 50 to 80 per cent of those working in the mines of California and the neighboring

⁶⁷ *Lancet*, March 18, 1905.

⁶⁸ *J. Am. M. Ass.*, 1911, 56: 259.

state of Nevada were infected with hookworms. For a full discussion of hookworm disease see page 142.

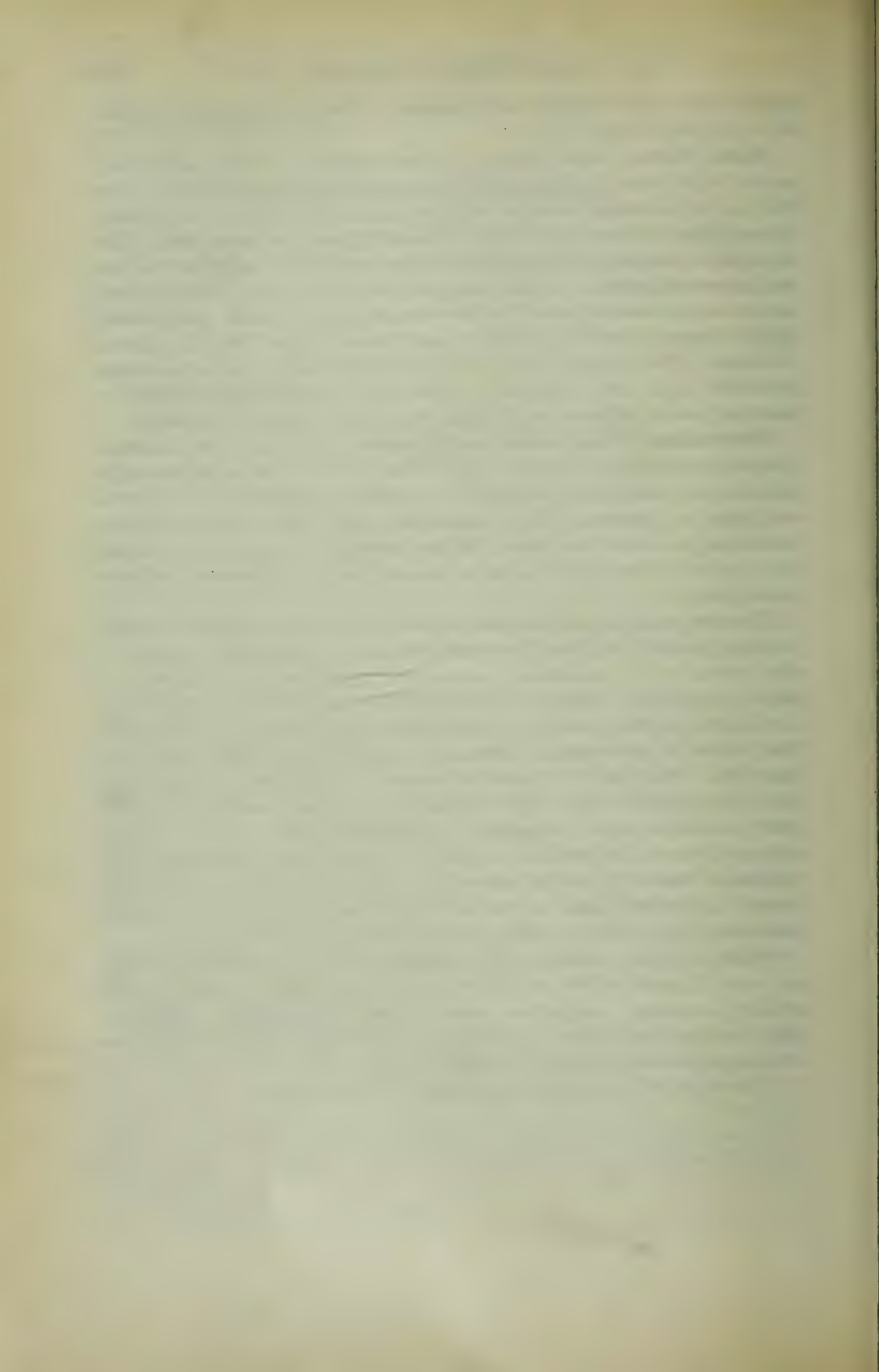
Mouse Favus.—Mouse favus, or favus herpeticus, in man has recently assumed significance in the United States because of the possibility that those who handle Australian wheat, or who are engaged in milling it, may become infected from the bags, dead mice, or possibly from the wheat itself. Men employed in resacking the grain and women employed in mending the torn and gnawed bags often contract favus of the glabrous skin. This is favus herpeticus of Quinke, caused by the dermatophyte *Achorion quinckenium*, which is quite distinct from the typical human favus due to *Achorion schanleinii*. The disease in man develops clinically much as herpes tonsurans, or barber's itch. The infection is usually not serious, yielding readily to treatment, and is far more readily cured than favus vulgaris of the scalp.

Tuberculosis.—Tuberculosis is often spoken of as the most important disease of occupation. There is no doubt that it is the most important single infection in industry, but whether it is commonly contracted as a result of occupation is a question. It is certain that dusty trades, poorly ventilated workrooms, sedentary occupation, fatigue, irritating fumes, long hours and the grind of routine, as well as other factors found in industries, predispose to the disease.

The view generally accepted now is that the infection is usually contracted in infancy or early childhood, but manifests itself clinically later in life. In other words, while tuberculosis is sometimes contracted in industry, it is usually contracted in infancy. It is prominent in industry because 25 per cent of all deaths between twenty and forty years of age are due to tuberculosis. The problem of tuberculosis is intimately bound up with personal habits and home life. Therefore, it is quite as proper to consider it a house disease as an occupational disease. Bad sanitary and hygienic conditions light up latent infections, and it is hopeless to expect arrest or cure of the process so long as the victim continues to work under unfavorable conditions. The problem of what to do for the tuberculous workman and his family, with the "cured" and arrested cases, as well as to find suitable occupation for the pre-tubercular types, is often a difficult puzzle for the social worker.

Statistics plainly show that tuberculosis, as well as bronchitis, empyema, and other diseases of the respiratory tract, is unusually prevalent among grinders, engravers, compositors, stone workers, millers, bakers, plasterers, brass workers, glass cutters, furriers, weavers, and other trades in which there is undue exposure to dust and irritating vapors.

The subject of tuberculosis is discussed in full on page 157.



SECTION XV

SCHOOL SANITATION AND CHILD HYGIENE

It took a long time to realize that the whole child goes to school—his body, mind, and soul; that education of the mind alone is one-sided and may be hurtful; finally, that the hygiene of the child and his teacher, as well as the sanitation of school buildings and their equipment, is of fundamental importance. The combination of compulsory education and schools having an unbalanced curriculum or impure water or vitiated air or improper sanitation is nothing short of a crime by the state against the state. The child profits directly from attendance upon a school which has due regard for the child's physical well being and the development of his character; the state profits indirectly from the lessons in sanitation and hygiene which are carried into the child's home, and are applied as a matter of course in the home of the future citizen. Thus the principles of personal hygiene and sanitation become second nature, and in this way the conquest of the preventable diseases may be materially hastened. It is an economic waste to educate children and then permit them to die of some preventable infection before they have reached the period of maturity and productivity.

The school furnishes abundant material for the physiologist and the psychologist to study growth and development. The effect of the nature and order of the studies for each school year; the hours of work, rest, play, and physical exercise should all be studied and regulated according to the requirements and capacities of each school period, and should be based upon accurate observations extending over long periods of time. Both the immediate effects and the remote influences upon adult life should be taken into consideration. Youth is the time of unrest and activity, and it is part of the school work to direct these energies so as to obtain the best development; youth also requires generous nourishment and sufficient sleep. A child who comes to school tired and worn from disturbed slumber cannot profit in body or mind. The child who comes to school hungry or who does not have a judicious luncheon at the recess period is seriously handicapped physically and mentally. The quality of the food offered for sale at recess should be under close scrutiny. The hot lunches and nutritious food furnished some of the school children in Boston, New York and other cities at reasonable prices are a practical and wise innovation. See page 1278.

School Age.—The child should not be sent to school too young. Children must first learn to walk, run, talk, and coördinate muscles before they undertake reading, writing, and arithmetic. Children should rarely be permitted to start school life until they have passed their sixth birthday. Few are sufficiently developed or sturdy enough properly to endure, either mentally or physically, the discipline and exactions of application and study before that

age. Parents should not hesitate to send a healthy child to school at the age of six.

Pupils should not be graded according to their ages, but according to their mental capacity. Individual aptitudes should be encouraged. The work should be as individual as practicable, and special classes made for backward as well as for forward minds. Special facilities should be afforded for the progress through school of those showing unusual ability.

School Hours.—For the elementary schools one short morning session is enough, but city circumstances often demand two sessions. The general tendency is to reduce the hours of compulsory school attendance and increase the optional time through elective systems which encourage and foster native talents.

First-grade pupils should not spend more than one-third of their school time in their seats. The child on beginning school life enters an environment radically different from the free and active life which was his before school days began. When unduly restrained he soon manifests symptoms of fatigue. Teachers should know that a fidgety young child is often a tired child. Exercises of various kinds that call into play muscular activity are most important at this age not only for mental development, but for physical growth, as well as for relief from the fatigue occasioned by sitting at desks.

The rural school is a problem of magnitude, for approximately 50 per cent of the children of school age in the country are in hamlets and towns of 1,000 and less, and about 39 per cent of them come from farm homes. The difficulties of the "little red school house" require special consideration.

Ungraded Special Schools.—Ungraded or special schools should be provided for backward and defective children (page 564) and for those having favus, ringworm, rachitis, or other conditions requiring either special pedagogical methods or particular medical treatment. Open-air or fresh-air schools for children who have or are threatened with tuberculosis serve a very useful purpose. Ungraded schools for backward and defective children should emphasize vocational training. These children present a serious problem for society. See page 552.

The high school is the time of puberty—one of the most difficult periods in the development of the child. This is discussed in the chapter on Sex Hygiene.

College life is beset with the problems of adolescence—Venus, Bacchus and Mars. Some of the dangers of this age are the venereal peril, tuberculosis, intemperance and drug addiction. The sudden freedom of university life after the strict restraints of preparatory schools is sometimes more than youth is able to withstand.

Object.—Finally, the whole school program should demonstrate that the object is not to teach the child to be a child, but to direct his development so as to become a useful man or woman. The school system should therefore be carried out with due regard for future events and should be correlated with the adult life of the child.

Child Hygiene.—Child welfare is the older term and is used especially by social workers, teachers and philosophers interested in the development of children. Child welfare concerns itself especially with the care of sick, dependent, delinquent and defective children. Child hygiene concerns itself primarily with child health.

Helen Putnam of Providence introduced a resolution in the American Academy of Medicine at Chicago, 1908, advocating the establishment of a national organization for the study of infant mortality; its causes and prevention. The American Association for the Study and Prevention of Infant Mortality was formed to crystallize the country-wide, but unorganized sentiment, demanding protection of infancy. Before this time only sporadic and scattered attempts had been made through milk stations and private agencies.

In December, 1908, the Bureau of Child Hygiene was formed in the Health Department of New York City by Josephine Baker. This was the first bureau of its kind in the world and it was the first time any municipality declared itself responsible for preserving the health of children as well as preventing disease. Dr. Baker blazed the trail and her pioneer work has been copied far and wide. In 1912, the Federal Children's Bureau of the Department of Labor was organized under Miss Lathrop to investigate and report all matters pertaining to the welfare of children and child life among all classes. By 1922, 45 cities and 38 states had departments dealing with child hygiene.

Much of the child welfare work is still done by private organizations, with varying efficiency. It is part of a community problem and should be regarded as a governmental function as well as a community obligation. Child welfare is only one part of a cycle from mother through infancy, and the child to the adult. It includes every phase of health and sanitation in the whole realm of preventive medicine.

Childhood is the best time for preventive work. It presents the greatest opportunities, gives the best results. Childhood is the most plastic period.

There are four stages of child hygiene: (1) prenatal, (2) infant, (3) pre-school, and (4) school.

The prenatal clinic demands examinations and advice in maternal hygiene. This subject and infant mortality are discussed on page 506.

Birth registration is all-important for the second stage. It is the only way to trace and keep track of the babies. In 1908 the birth statistics were satisfactory in only a few large cities. Now the birth registration area includes 23 states and 90 per cent of the population. In 1910, there were only 43 milk stations in 30 cities, according to a survey of the United States Public Health Service. Now they are too numerous for reliable data.

The preschool child is often neglected from a public health point of view. There has long been a gap between infancy and school age, during which time the child runs wild. At this period children are most susceptible to many diseases. Nutrition is of vital importance for growth and development. This is a difficult problem, for children at this age are hard to reach. It

can be solved by extending the activity of baby health centers to the school age, thus establishing continuity. Unless the coöperation of the mother is secured in the program, it will fail. The fundamental problem is one of education.

Fatigue.—Fatigue, prolonged and oft repeated, may injure the development and health of the child. Fatigue is favored by poor ventilation, compulsory sitting upon hard and ill-fitting seats at improperly constructed desks, prolonged tension of a strict discipline, studies that are too intensive, and insufficient relaxation or inconsiderate treatment of the little ones. Discipline, obedience, and regard for the human rights of others are among the most important things learned at school.

Many a child is unjustly disciplined and his little soul harassed through no fault of his own, but perhaps on account of defective eyesight or hearing, or some other physical handicap, or as a result of mental deficiency, or even an unusual mental proficiency.

The question of home work should be carefully regulated in accordance with the capacity and age of the child. Children should not be kept busy at prescribed work most of the hours of the day. Some time should be left for quiet play and the encouragement of personal inclinations during which time the best development unconsciously occurs. Initiative, self-reliance, and self-help are submerged by lack of free time. The amount and nature of the work, both in and out of school, must be judiciously considered and should be based upon long years of careful study and observation. The immediate as well as the remote effects should be taken into consideration. Many an ill-tempered child is simply overwrought and chronically tired out through excessive application of a conscientious and studious nature to tasks beyond the physiological capacity of his little brain and body.

Nutrition and Education.—School age is the period of growth and development, physical and mental. To meet the needs of the growing child, the diet should be generous and varied by judicious selection. Milk, cereals, eggs, fresh fruits, vegetables and greens, bread and butter and meat should be the staple articles of food. See page 619.

The school child is at a serious disadvantage when he is unable to return home for the midday meal, which, in agricultural districts, is the principal meal of the day. Evidence is accumulating of the beneficial effect on the child's physical condition of the addition of milk and a hot dish to the lunch the child may bring with him. Undernourished children should have milk, or milk and graham crackers as a mid-morning lunch. The same should be provided for those who suffer from frequent colds, are chronically pallid, or who do not satisfactorily gain in weight. Some children of this type are probably victims of "latent" scurvy, for they often make remarkable improvement following the taking of orange juice at the morning recess period.

A healthy child at the age of six does not lose weight on entering school life; if so, something is wrong and should be investigated.

An acceleration (skipping grades) in the elementary schools is apt to be associated with a poor nutritional status. Hence caution should be observed in accelerating young children. There is no hurry. At the junior-high-school age the effect of acceleration on nutrition is not so evident.

Many children come to school hungry—in fact without any breakfast. Often the breakfast consists of carbohydrates only. Clarke found in an agricultural community that 57 per cent of the children drank coffee for breakfast to the exclusion of milk, and that only 15 per cent had milk, a remarkable condition in a dairy-farm region. The need of nutritional classes and teaching of food values is evident.

The common causes of malnutrition in children are insufficient food (not enough calories per day); unbalanced diets and avitaminosis; fast eating; sugar habit; omission of morning and afternoon rest periods; overfatigue from a full-day school program and from school examinations; overfatigue due to late hours; activities outside of school, such as clubs, music, and dancing lessons, etc.; too many home studies; failure to gain because of obstruction to breathing such as adenoids, tonsils, deviated septum, etc.; the use of tea and coffee; the omission of cereals and milk from the diet; and other bad health habits. Only a small percentage are undernourished from poverty, tuberculosis, hereditary syphilis, and other diseases. The problem is individual and needs careful diagnosis and special treatment for each case.

A malnourished child is not capable of concentrated work for more than an hour or so without overfatigue.

Absences.—Over 70 per cent of all absences are due to medical problems. More than a fourth of all absences are because of the common cold, and all respiratory disturbances constitute 40 per cent of all absences. Truancy is not a large problem in the younger age groups. Other common causes are: exposure to contagion, weather, and religious holidays. A record of absenteeism with its causes should be kept and each case investigated.

Weighing and Measuring.—Every child attending school should be weighed and measured at least twice during the school year, preferably at the time of general inspection when the schools first open and again near the termination of the school year. Undernourished children particularly should be weighed at least once a month in order to determine whether improvement is taking place as a result of the special measures which should be taken in such cases. Weighing and measuring occupies a unique position in school health service because it is a ready means whereby the interest of the child may be stimulated with regard to his own physical condition, and the coöperation and interest of the classroom teacher in the health of her charges secured. Interest is the first law in pedagogics.

Height-weight tables should be interpreted with regard to race, climate, heredity and other factors.

Health Scoring.—A score chart has been devised to tell at a glance the physical condition of the children in a room. The charts are designed to meet the need of a permanent record and also to impress upon the child the

ideal of health. "It is a record in the language to which the children are accustomed." Charts and stars are now in general use in the schools all over the country. The desire to have a gold star before his name obtains the child's coöperation and helps to carry the health message into the home. The Health Score Charts (Child Hygiene Form No. 12) and the classroom weight charts may be purchased from the Superintendent of Documents, Government Printing Office, Washington, D. C.

Health Education.—Health education is fundamental and should be included as part of the required work of all grades. It is comparatively easy to teach and influence children, very difficult to change the habits of adults. All children should learn something about the structure and functions of the body in addition to the principles of hygiene and sanitation. The teaching of health and the prevention of disease should be made an essential part of education in all schools. The program should furthermore seek to improve health and efficiency by including the practice of the art of hygienic living. It requires constant instruction by example on the part of the teacher in personal hygienic habits and cleanliness. It is necessary to teach the teacher in order that children may receive sound instruction. Teachers, nurses, parents and the children themselves need education concerning adequate nutrition and especially the importance of rest and the evil of chronic fatigue for growing children. The program for health education should coöperate with medical school inspection. Defects which interfere with growth or otherwise handicap children should be removed or corrected.

The best methods are simple and direct. A graded series of textbooks should be used. Interest may be aroused by the picture man, the health fairy, attractive literature, group competition, classroom drills, and above all by the example of the teacher in health habits. In this way advantage is taken of the imitative tendency of children—good example is contagious.

School Building.—The school must be centrally located, so as to be convenient especially for the primary and grammar grades, and the school building should be modern, artistic, clean, and sanitary in all its appointments. Every school building should have playgrounds connected with it. Playgrounds should be level and located on the sunny side of buildings; about 30 square feet for each pupil is necessary to meet the demands of play. Thus 1,000 pupils require 300×100 square feet for playgrounds alone. In cities, roofs may be utilized for play. A limited play area is best utilized by organizing recess play by sex and grades. Schoolhouses should be built in places that are quiet and free from traffic and nuisances, dangers of various kinds, and on ground that is either naturally dry or made so by subsoil drainage. The building should be solidly constructed and should stand apart, so that sun and air may reach it from all sides. A substantial and artistic structure well placed has an important influence upon the young mind and character. Trees and judicious landscape gardening should provide shelter and shade and add to the attractiveness. The foliage, however, must not interfere with the light and ventilation of the schoolrooms. If the building faces north,

with corridors and stairs on this side, all the rooms will have sunlight at some time during the day. The best general arrangement of the plan of the building is that in which the schoolrooms are all placed on the sunny side of the building, with the corridors, halls, stairways, and wardrobes on the other. Built in the old way, with rooms around a central well, schoolhouses have dark central halls and staircases, and favorable lighting cannot be had in some of the schoolrooms.

A school building whose sides face northeast, southeast, southwest, and northwest will have a more equable distribution of light and solar heat on all sides, in the latitude of the United States, and each side will receive some sunshine for at least a short time every day in the year when the sun is shining.¹

The basement should be under the whole building and carefully protected against dampness. Further, the basement should be well lighted, sunny, and kept clean.

School buildings should have at least two entrances, with doors opening outward; the halls and corridors should be generous and well lighted, and the stairs have easy risers and treads for children. The risers should be about 6 inches and the treads no greater than 12 inches. Inclines may be substituted for stairs.

The Schoolroom.—The schoolroom is the unit in planning a school building; that is, the building should be a number of schoolrooms properly disposed, and not a building cut into schoolrooms whose size and arrangements are dependent upon the size and shape of the building.

Some of the important considerations in the schoolroom are the number of pupils to be accommodated, its size and shape, the amount and direction of the light, the ventilation and heating.

The minimum floor space for each pupil should be 15 square feet. If 18 square feet are allowed all exercises are made easier both for pupil and teacher. Two hundred cubic feet of air space is the minimum commonly allowed; therefore a standard schoolroom designed to accommodate 30 pupils should be 20 feet wide by 24 feet long, with a ceiling 13 feet high. The best shape for a schoolroom is that of an oblong, the width being to the length about as 3 to 4. No teacher should be required to have classes exceeding 30 pupils. The rooms, floor space, and air space should be at least as capacious for the primary as for the grammar grades.

BOSTON STANDARD—ELEMENTARY GRADES

Size: 20 ft. by 28 ft. elementary grades.

20 ft. by 30 ft. for upper elementary grades.

12 ft. high in clear.

About 10 or 20 per cent of rooms should be of a size to seat 50 pupils.

¹H. H. Kimball, "Variation in the Total and Luminous Solar Radiation with Geographical Position in the United States," *Month. Weather Rev.*, November, 1919, Vol. 47.

KINDERGARTEN

800 to 900 sq. ft. and capable of having a circle 16 feet in diameter painted on floor with at least 4 ft. all around it.

HIGH SCHOOLS

26 ft. by 32 ft. for 42 pupils.

33 ft. 8 in. by 43 ft. for 60 to 80 pupils.

16 ft. by 26 ft. for recitation rooms.

3,750 to 4,000 sq. ft. with a height of not less than 24 ft. for high school gymnasium.

The color of the walls should be such as to absorb the least light and prove least taxing to the eyes. A light green-gray is favored for the walls, and white or cream for ceilings so as to reflect the light. The surface should not be glossy and should either be coated with an oil paint, so that the walls may be washed, or, better, calcimined with a water paint that may be readily renewed.

The School Furniture.—The most important articles of school furniture, considered from the view of hygiene, are desks and desk chairs, for the reason that the pupil spends during school hours so much time at work at his desk. Unless, therefore, desks and chairs are constructed with full regard for certain well-known laws of hygiene they produce defects of eyesight, injurious effects as to posture, and wrong habits of carriage which are borne through life and, sadly enough, become more pronounced as the years increase.²

Bowditch³ of Harvard University carefully measured and weighed 25,000 school boys and girls of Boston and found surprising variations. Taking ages on their last birthdays Bowditch found the variations in height indicated in the following table:

VARIATIONS IN HEIGHT OF BOYS AND GIRLS

	Boys	Girls
6 years of age	47.13 *	47.36
	40.66	40.57
Difference	6.47	6.79
11 years of age	57.50	57.96
	49.47	49.33
Difference	8.03	8.63
15 years of age	67.90	65.00
	56.55	57.39
Difference	11.35	7.61

* All figures are inches.

² Edward R. Shaw, *School Hygiene*, The Macmillan Co., New York, 1902.

³ Twenty-second Annual Report, State Board of Health of Massachusetts, 1890, pp. 479-522.

More recent data seem to indicate that geographical location, including climate and other attendant circumstances, may determine the age of most rapid growth.⁴

AN AVERAGE ANNUAL INCREMENT IN INCHES OF HEIGHT OF 14,335 SCHOOL CHILDREN IN VIRGINIA, MARYLAND, NORTH CAROLINA, AND SOUTH CAROLINA

	6 to 7 Years	7 to 8 Years	8 to 9 Years	9 to 10 Years	10 to 11 Years	11 to 12 Years	12 to 13 Years	13 to 14 Years	14 to 15 Years	15 to 16 Years
Boys...	1.4	2.0	1.9	1.9	1.7	1.9	1.8	2.3	2.6	1.7
Girls...	1.8	1.9	2.0	2.0	2.0	2.5	2.3	1.8	1.4	0.8

Besides the variations in height there is also variation in the rate of growth, and provision for this difference must therefore be made in the construction and adjustment of the desk and seat. The growth of girls is more rapid from twelve to fourteen years of age, while boys grow most rapidly from fourteen to sixteen years of age. The annual growth during the maximum period is often an inch more than the annual growth at other periods. Further, there exist certain anatomical differences of proportion between boys and girls. The sitting height of girls is greater proportionately than their standing height in comparison with boys.

In the more southern section of the country, it would seem, the growth of girls is more rapid from eleven to thirteen years of age, and of boys from thirteen to fifteen years of age.

The Desk and Seat.—The desk and seat must be adjusted so as to provide for differences of height and differences of growth. The desk must not be a prison stall, but should be comfortable and roomy. It must not favor the development of myopia and must not force a pupil into wrong postures. The matter is of greater importance than school men generally recognize.

The chair and seat should be of such a height that the thigh of the pupil when seated will be perfectly level, the lower leg being in an exactly vertical position, with the foot resting wholly upon the floor; that is, the thigh and the lower leg will, when the chair is of a proper height, form a right angle with each other. The seat must therefore be adjusted accordingly. The seat itself should not be flat, but somewhat concave, the lowest part of the concavity being where the tuberosities of the ischium rest. The concavity has the additional advantage of counteracting the tendency to slide forward on the seat when the pupil leans back. Its width from front to back should not be greater than two-thirds the length of the thigh of the seated pupils in order that the vessels and nerves behind the knee may not be constricted by pressure against the edge. The seat should have a back rest that will support the small of the back properly without leaning back excessively. Whether or not it supports the rest of the back is of small consequence. Support of

⁴ Heights and Weights of School Children, Reprint 750, *U. S. Pub. Health Rep.*, May 19, 1922.

the back carried to the level of the shoulder blades is likely to do more harm than good.

The distance between the seat and the desk should be such that the scholar may read at the desk and write on it without leaning forward more than a little and without entirely losing the support of the back rest. The desk should not be so close as to press against the abdomen, nor near enough to interfere with easy rising from the seat. This means a distance of $10\frac{1}{2}$ to



FIG. 143.—FAULTY POSTURE.

(Shaw's *School Hygiene*, The Macmillan Co.)

$14\frac{1}{2}$ inches from the edge of the desk to the seat back. It also means that the seat must not project under the desk more than an inch at most. The desk should be high enough for the arm to rest comfortably without much resting on the elbow; not, however, so low that the pupil must bend down to write on it.

If the desk top is made to slide backward and forward it will give the pupil more freedom of movement while at the desk and will also permit him to sit down at the desk and rise from it with greater ease. One of the important considerations of a school desk is the proper slope of the top. It is well known that the line of light which least taxes the eyes should fall upon the printed page perpendicularly to its plane. To accomplish this some writers recommend a slope of 45° for the desk top, others 30° . These angles, however, are not practicable. The Vienna Expert School Desk Com-

mission recommends an angle of 15° for the desk top, which is also approved by the experiments of Shaw. Such a slope permits a correct posture in vertical writing.



FIG. 144.—GOOD POSTURE FAVORED BY SUITABLE CHAIR.

A foot rest is sometimes attached to desks. The weight of opinion is now against foot rests, as they restrict the free movement of the pupil's feet while at the desk and interfere with opportunity to shift his feet and legs for relief from inactivity, and further interfere with the thorough cleansing of the floor under the desk. Shaw recommends the Heusinger desk, Fig. 145, and also the Ideal desk. The desk and seat shown in the accompanying photo-

graph, Fig. 146, are known as the Boston school desk and chair. There are now many thousands in use in the Boston schools, and they are being adopted elsewhere.

The seat and chair should be adjusted for each pupil when he enters school or is transferred to another room. Desks and seats should be adjusted



FIG. 145.—THE HEUSINGER DESK.

(Shaw's *School Hygiene*, The Macmillan Co.)

at least twice a year: at the opening of school in September and again in February or March.

The Blackboard.—The blackboard should be placed upon the wall opposite the principal light. The board should not have a shiny, reflecting surface, and should never be placed between windows or near them.

The best blackboards are made of slate, as they can be washed, which lessen the dust nuisance. The best slate for this purpose has a greenish or

strong black color, which is to be preferred to the grays and brownish-blacks. The loss of light by absorption can be reduced greatly by reducing the blackboard area, and also by covering the blackboard with adjustable curtains when not in actual use. Colored crayons made with arsenic or sulphid of mercury carry danger and should be prohibited. Dustless crayons may now be found on the market.

Posture.—Every condition must be eliminated and every care exercised to prevent the acquiring of physical defects in school, as well as to prevent the accentuation of those physical defects which the child may have possessed before entering school. Posture during sitting is of greater consequence than posture during standing, on account of the longer time the child sits and the muscular fatigue caused by the inactivity of a great number of muscles of the body for a long period. Stooping over the desk may be due to myopia. It contracts the chest and interferes with free respiration, and puts additional labor on the heart; it leads to round shoulders and curving of the spine backward and a carriage in which the head is pitched forward; it also tends to displacement of the internal organs, both of the abdomen and pelvis.

In order that the pupil may be in a proper physical condition to maintain an erect posture while in his seat, and thus form correct habits which he will carry through life, he must be given periods of relief from sitting at the desk and corrective exercises at different times during the day. In the first year the child

should not be confined at his desk more than one-third of the time. In the succeeding years the total amount of time occupied at the desk may be gradually lengthened. In addition to the regular recesses there should be frequent short intervals of respite from sitting at the desk devoted mostly to some form of physical exercise. Vertical writing is conducive to a better position of body than slanting script.

The problem of posture is complex. Posture affects health and health affects posture. Poor bodily mechanics often explain why a child is not enjoying the health and development it should. Good nutrition is a contributing factor to good posture. Defective vision, adenoids and bad tonsils tend to have an unfavorable effect on a child's posture. Both congenital and acquired defects are common. In childhood the tissues are plastic and the correction of faulty habits and bad posture is comparatively easy, but often requires skilled orthopedists.

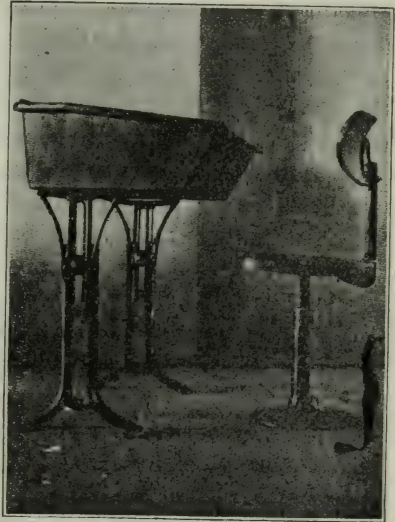


FIG. 146.—BOSTON SCHOOL DESK AND CHAIR.

Recess.—A recess of not less than twenty minutes during the morning session and again during the afternoon session, when all pupils, if the weather and climate permit, go out of doors and engage in some form of active play, is of incalculable value in its results upon physical health and mental development. In addition there should be given to each grade every school day at least two short periods of systematic physical drills for pupils and teacher with the windows open.

Lighting.—The light must be of sufficient intensity, equally diffused, and come from the right direction. For the want of a more exact standard, the general rule is that the amount of transparent glass surface admitting daylight should not be less than from $\frac{1}{5}$ to $\frac{1}{4}$ of the floor space. The necessary amount of window-glass area will depend on the location of the building, direction from which the light is admitted, size and shape of the room, and the proximity of other buildings or objects which might obstruct or reflect the light. A school building should never be located nearer another building than twice the distance represented by the height of the opposite building. Foliage may also diminish the amount of light admitted to classrooms, more usually to those situated on the first floor. Trees should not be planted at a distance less than fifty feet from the nearest window, and must be kept trimmed so that their maximum height should not exceed one-half this distance.

The amount of transparent glass surface required for adequate illumination must be great enough to afford sufficient light on rainy, overcast and cloudy days. Excessive window space is scarcely possible, for glare and direct sunshine may be regulated by shades.

The amount of illumination (the amount of light received on a surface) is measured in foot candles. A foot candle is the amount of light from a standard candle falling on a surface at a distance of one foot. Originally the standard candle was of sperm wax, one-sixth pound weight, and burned 120 grains an hour.⁵ It is now recommended that the daylight illumination of classrooms in use during daylight hours shall not be less than 5 foot candles (preferably 10 foot candles) on desk tops, or 3 foot candles (preferably 6 foot candles) on charts and blackboards.⁶

A unilateral arrangement of the windows to the left of the seated pupils will secure the best illumination of classrooms, provided the farthest desk is not more than 20 feet from the window. By this arrangement annoying cross shadows while writing are avoided. However, windows may also be placed at the right in classrooms of excessive width, assembly halls and study halls, in order to provide additional illumination. In addition, this arrangement may be utilized for ventilating purposes or for admitting direct sunlight while the pupils are not engaged in study. Windows may also be

⁵"Studies in Natural Illumination in School Rooms," *Pub. Health Bull.* No. 159, 1925.

⁶"Revised Code of Lighting School Buildings," American Illuminating Engineering Society and American Institute of Architects, June 16, 1924.

placed at the rear of the pupils, but this arrangement has the disadvantage of requiring the teacher to face the light. Skylights have been advocated for illuminating classrooms but, when they are the sole source of the illumination, they produce an objectionable shut-in or prison-like effect that reacts unfavorably on the pupils.

The window sash should be at the level of the horizontal visual plane above the floor of the seated pupils and should reach as near the ceiling as the construction of the building will permit; for the higher the windows, the greater the intensity of the illumination, the more uniform its distribution, and the more complete the elimination of glare. Light should never enter from the front and shine in the pupil's eyes. Window shades should be translucent. In general, window shades that transmit colored rays, other than those from near the middle of the spectrum, are undesirable. Opaque shades are rarely, if ever, needed. The shades should be capable of adjustment to any position in the window, either by the use of two rollers both at the middle of the window, or one at the middle and one at the bottom, or a single roller that can be adjusted to any position. Teachers should be impressed with the importance of properly regulated window shades to the conservation of the vision of school children. The upper fourth of the window furnishes the best light, hence it is obvious that shades should not be hung from the top.

Sight-saving Classes.—A certain proportion of children, though not blind, are unable to take advantage of their educational opportunities by reason of marked defects of vision. To these should be added children with slight defects that may diminish visual acuity or become worse from the strain occasioned by the ordinary school routine. According to Bishop Harman,⁷ in a series of histories gathered of myopes of over three diopters, aged twenty to sixty years, and classified according to whether engaged in close work all day, or in customs or occupations which did not entail habitual close work, in course of time no less than 53 per cent of the close workers experienced unmistakable failure or ability to continue this work because of serious eye strain, and of those not engaged in continuous close work, only 9.4 per cent. In other words, there is unmistakable evidence that neglect of certain types of defective vision will result in loss in the efficiency of the individual and restricted enjoyment of life. See page 461.

In general, the children who require special care are those who may continue in the ordinary school under certain restrictions; those who should be educated in sight-saving classes; and those who are educationally blind. The majority of the children admitted to sight-saving classes are myopes, a less number have some form of keratitis, and the remainder will be suffering from congenital defects. The conditions of sight-saving classes outlined by Kerr⁸ are:

⁷"Standards of Vision for Scholars and Teachers in Council Schools," *Brit. M. J.*, January 13, 1923.

⁸James Kerr, *School Vision and the Myopic Scholar*, 1925.

1. For all lessons that can be learned by listening to the teacher the children in the myope class go into the ordinary school.

2. Lessons that need reading or writing are given in the myope class. Instead of using books, pens, and paper, each child has a large blackboard, on which it writes with chalk just as the teacher does.

3. In the myope class a specialty is made of handicraft, various forms of carpentry, bent-iron work, model-making, printing from blocks, and drawing. The children enjoy the work immensely, and the instruction is most profitable.

4. The children learn to dance and drill, and for these lessons most of them are able to join in with the ordinary children.

A study of the promotion records of 100 sight-saving class pupils in Cleveland, Ohio, shows a reduction of 85 per cent in the proportion of failures after the work in the sight-saving classes is well begun, as compared with the proportion of failures of those pupils prior to entrance in the sight-saving classes. The proportion of failures among the sight-saving class pupils is 60 per cent less than the proportion of failures in the entire public school system.⁹

The Eyes.—Errors of refraction are exceedingly common, and if not corrected may be the cause of headache, nervousness, reflex pains, and a great variety of symptoms. They are also a great handicap to the mental and physical development of the child. The vision of all children should be tested annually, and at least once for color-blindness. It is believed that the unnatural strain of accommodating the eyes to close work (for which they were not intended) leads to myopia in a certain type of growing children. The eyes should therefore be tested and errors of refraction corrected at least once a year. There are certain children who show normal vision by the ordinary tests (Snellen test type), yet whose eyes should be examined by an expert if they habitually hold the head too near the book (less than 12 to 14 inches); or if they frequently complain of headache, especially in the latter portion of school hours; or if one eye deviates even temporarily from the normal position. The following symptoms also indicate trouble with the eyes, viz., scowling and wrinkling of the forehead when reading or writing, watery eyes, reddened or granular lids, twitching of the face, inattention, and slowness in book studies in a child otherwise bright.

The conditions which are especially hard upon the eyes are dim light, improper angle of vision, small print, and prolonged focusing at close range. Type for books should not be smaller than the following:

Grade	Type	Width of Leading
First year	2.6 mm.	4.5 mm.
Second and third years	2.0 mm.	4.0 mm.
Fourth year	1.8 mm.	3.6 mm.
Above this grade	1.6 mm.	3.0 mm.

⁹ "Sight-Saving Classes in the Public Schools," R. B. Irvin, Supervisor Department for the Blind, Cleveland, Ohio, *Harvard Bulletins in Education*, No. 7, November, 1920.

In addition to the size the characters should be simple, the ink black, and printed upon paper with unreflecting surface that is free from gloss. Paper of a grayish tone is to be avoided and the paper should be thick enough or of such quality that the print does not show through from the back. Pupils should be taught that it is advisable while reading or during other close focusing of the eyes occasionally to look away and accommodate for distance to relieve the tension and counteract the tendency to myopia. See *Conservation of Vision*, page 451.

Ventilation.—The mandates for satisfactory ventilation are fulfilled if the air is kept cool with slight fluctuations in temperature, in motion but not excessive, and free from odors. Either plenum fan-gravity or window-gravity ventilation will give satisfactory results if properly designed, installed and operated, but the window-gravity method is more generally promising.¹⁰ The subject is discussed in the chapter on Ventilation.

Ventilation of the schoolroom is of paramount importance. There is a great waste of time and energy of both the teacher and pupil working in a vitiated atmosphere, for pure air properly conditioned is favorable for good mental work. Bad air means sluggishness, headache, listlessness, inattention, lack of energy, and a disinclination to mental and physical exertion; further, bad air lowers resistance to certain diseases. In cold climates ventilation and heating go hand in hand.

The first responsibility in a poorly ventilated schoolroom lies with the building committees, because of their failure to see that the sum allowed for the ventilating plant is sufficient to give the best that science can devise. The architect often skimps the ventilating system in order to provide a larger and more ornate building.

In favorable climates and during mild weather the windows should be kept open. Even during cold weather the windows should be opened periodically and the room thoroughly flushed out with fresh air. The windows should always be thrown open at recess and also during calisthenic drills and physical exercises and also at the close of sessions. The experience of the open-air and fresh-air schools teaches that cold is a fine tonic for mind and body.

Satisfactory ventilation by means of windows at all times is impossible on account of varying winds, and weather conditions, but more especially on account of the impossibility of securing proper attention on the part of the average teacher to the matter of ventilation in addition to other duties. Window ventilation is simplest, cheapest, and, all things considered, frequently the best. The Fairchild system of window ventilation is described on page 899.

Every schoolroom with a proper equipment and a good janitor may be kept well ventilated at all times. Many good ventilating systems are rendered inefficient through the employment of cheap janitors. A good janitor not only means greater efficiency, but a saving in fuel cost.

¹⁰ Report of the New York State Committee on Ventilation, 1923.

Heating.—Heating by direct radiation from stoves or steam coils or hot-water pipes is inadvisable for schoolrooms. The hot-air furnace may be used, provided the air is sufficiently moist, but the direct indirect system of steam or hot-water pipes is to be preferred. The Massachusetts law requires 30 cubic feet of pure air every minute to a pupil (1,800 cubic feet per hour). A statutory limitation of air flow is undesirable because under existing statutory provisions it will be difficult, if not impossible, to regulate the air flow to a rate compatible with the greatest comfort.

Special attention should be given to the question of humidity, so that the warmed fresh air shall not be excessively dry.

The temperature commonly accepted as proper for a schoolroom is between 60° and 68° F. The children would probably work to better advantage if the temperature were kept a few degrees lower and the humidity kept so that the wet bulb never goes above 70° F. (see page 826). A thermometer should hang at about the breathing line in every schoolroom and the teacher should take hourly readings and keep a record. The temperature of schoolrooms is usually too high, and those heated with the hot-air furnace are usually also too dry. Both extremes are prejudicial. If the air of the neighborhood is smoky and dusty it may readily be filtered before it is pumped into the schoolroom. The combination of the plenum and vacuum systems, the air being driven by rotary fans, is one of the best methods of artificially ventilating schoolrooms. (See chapter on Ventilation.)

Open-Air Schools.—Children suffering from malnutrition, rickets, anemia and tuberculous glands of the neck; from blepharitis and other chronic non-infectious eye diseases associated with malnutrition; nervous and highly strung children; certain types of crippled children; frail children living in the same house with an open case of tuberculosis, and children convalescent after debilitating diseases should be enrolled in open-air classes or schools.

Most children in open-air classes gain in weight, color, appetite, attendance, deportment and scholarship. The results are uniformly good and progress in grades is often remarkable, even though the children work little more than half the time. Open-air schools must give particular attention to clothing and diet, and also to recreation and rest. The benefits to the subnormal and pretubercular child are proved, and it is difficult to explain why it should not be more widely adapted to the average child. See pages 184 and 617.

Special classes should also be organized for the blind and deaf, for speech improvement, eye conservation, and cardiopathic children. In countries where favus prevails special schools are provided, and the same principle might be applied to other communicable infections that are prevalent and chronic.

Water-Closets and Urinals.—Separate accommodations must be provided for the sexes; privies in country districts should be in entirely separate buildings. The urinals should be constantly and automatically flushed and water-closets and urinals should be made to allow complete inspection and

use of the scrubbing brush. Thorough ventilation of the toilet-rooms should be planned for and they should be kept clean and sweet at all times. See page 1087.

The water-closets may be in the basement if properly constructed and independently ventilated. The floors should be asphalted to facilitate cleaning and flushing, and should be hosed daily, and scrubbed at least once a week. The toilet-room should be well lighted. Deodorizers should not be used, for if toilet-rooms are kept clean and water-closets well flushed they will not be necessary. Urinals should be made of slate or hard asphalt or other non-absorptive material, and one urinal should be provided for each fifteen boys. The out-houses in country schools should be properly constructed and under supervision. In fact, a matron should be in attendance to assist the little tots in the kindergarten and lower elementary grades, and a watchful eye on the part of the master of the school and those he delegates for this duty should be kept to prevent misbehavior in toilet-rooms.

Cloakrooms.—There should be one cloakroom for each classroom, and it should connect both with the hall and the classroom. Cloakrooms should be lighted from the outside, heated, and thoroughly ventilated to carry off odors and to dry the clothing. Hanging the clothing in the halls is undesirable, for obvious reasons. Each pupil should have a shelf on which to lay hats and small articles, hooks upon which to hang overcoats, and a space for rubber shoes and umbrella.

Teachers should see to it that the pupils do not sit in wet shoes and stockings or in wet clothes. Each school should have some provision for drying wearing apparel, such as a drying chamber which may be in charge of the janitor, to dry the wet clothing during school hours.

Dressing-rooms should also be provided for the teachers. All such rooms and lockers should be kept scrupulously clean.

Cleanliness.—Schools should be kept scrupulously clean and every precaution should be taken to prevent dust. Cleanliness of person and surroundings should be one of the most important lessons which the pupil learns at school. Through example and discipline pupils should be taught to love order and neatness and to abhor untidiness and slovenliness. Cleanliness is the keynote of sanitation.

One of the duties of the school is to teach and to require at all times cleanliness of person and clothing. The example of clean school rooms, corridors, lockers, toilets, basement, and grounds will, in time, influence the young citizen. Floors especially should be kept clean and the child be required to use the door mats before entering the building. Dust must be discouraged in all ways. In some schools in poor districts it is a good plan to have shower baths for those pupils who do not enjoy good bathing facilities at home. A toothbrush drill is the means of teaching many a child the first principles in dental prophylaxis. The teacher should be constantly on the lookout to impress upon the pupils the elementary facts in hygiene, such as turning aside the head and holding the handkerchief before

the mouth and nose when coughing or sneezing. The teacher should discourage the habit children have of carrying their fingers to their mouths and noses. The anti-spitting rules should be reiterated and strictly enforced. The danger of mouthing toys and pencils and the habit generally of placing things in the mouth should be discouraged; "swapping" partly eaten articles of food should be prohibited, and the reasons explained. Cleanliness is not instinctive in children; it must be learned. The significance of modern biological cleanliness can come only through education and example. Progress in these matters cannot be made without an intelligent understanding on the part of the teacher. It is therefore important to teach the teacher.

Some of the requirements for schools are: clean drinking water; bubbling fountains and the abolition of the common drinking cup; discontinuance of the roller towel, cake of soap, brush, comb, or other toilet articles used in common; cleanliness of floors, desks, corridors, cloakrooms, toiletrrooms, basement, and grounds; the prohibition of dry sweeping or dusting. Blackboards should be washed frequently to avoid the dust nuisance, and the floors may be treated with one of the dustless floor oils. The windows should be kept clean, and each child should have his individual books, pencils, and other accessories. Youth is the time to form good health habits. Health Day, observed annually, gives a special opportunity to emphasize the importance of hygiene and cleanliness.

Medical Inspection of Schools.—The medical inspection of schools is no longer an experiment, but a pressing necessity. It is founded on a recognition of the close connection which exists between the physical and mental condition of children in the whole process of education. It seeks to secure ultimately for every child, normal or defective, conditions of life compatible with that full and effective development of its organic functions, its special senses, and its mental and spiritual powers which constitute a true education.

The object of a medical inspection of schools is not primarily the treatment of diseases, but rather their prevention. More than this, it aims to improve health, thereby enabling the child to become a better citizen. One of the principal objects is the early recognition of physical defects such as errors of refraction, imperfect hearing, malformations of the body from abnormal positions, caries, adenoids, enlarged tonsils, and other obstructions of breathing, and sources of inflammation, etc. An important object of the medical inspection of school children is to determine their fitness to enter school and to recognize mental and nervous disorders; also the early recognition of the communicable diseases and measures to prevent their spread; the supervision of vaccination, and disinfection; the teaching of personal hygiene to pupils and teachers, and the sanitation and cleanliness of the school building and its surroundings; the adjustment of the seat and desk, and the medical supervision of the mental and physical work of the child. One of the important functions of medical inspectors of schools is to determine

and diagnose the causes of undernutrition. Medical inspection of school children has infinite possibilities little realized.

Medical inspection of schools has made very slow progress in this country. It is only within the last decade that any marked advances have been noted and even now there are but few places where it is carried out with the thoroughness and completeness that its importance demands. At Newton, Massachusetts, each child is examined by a physician and pediatrician, in the presence of a physical director, expert in the problems of posture, and in the presence of teacher and parent. The honor of first beginning school health supervision belongs to France, where this duty was imposed on the school authorities in 1837, by royal ordinance. Systematic school medical inspection was started in Brussels in 1874 and in Paris in 1884, since which time the movement has become world-wide. The first school medical officer in America was appointed by the city of New York in 1892. The first systematic inspection of school children was begun in Boston in 1894, after four years' effort, by Samuel H. Durgin, Commissioner of Health, who is regarded as the father of the system throughout America. Fifty physicians were appointed for the fifty school districts to inspect children for the contagious and other diseases.

The first scientific and extensive examination of school children was made by Henry P. Bowditch,¹¹ whose essay upon "The Growth of Children Studied by Galton's Method of Percentile Grades" has become a classic in the subject. In 1908 there were only seventy cities outside of Massachusetts having medical inspection of schools. From this beginning the evolution of school medical inspection legislation has been gradual, but there is still a very serious lack of uniformity and completeness in these laws. The ideal school medical inspection law yet remains to be written in the statutes. In 1924, thirty-one states and territories had enacted school medical inspection laws which designated some state agency to administer the law. The important thing is how it is done, rather than under whose auspices it is done. In eleven states the health and educational authorities coöperate in the administration of the law; in eight it is administered by the department of education; and in twelve states and territories, by the board or department of health. In twelve states no authority is designated to administer the law, though certain duties are imposed on the local authorities; in two states authority is given to local units to employ public health nurses, but no special reference is made to school medical inspection, which they make in some cases. In six other states there is no special school medical inspection law, but some cities have instituted a system of inspection under their general public health powers. In a number of instances the wording of the law is so indefinite that it is impossible to decide whether the prescribed coöperation between the health and educational authorities is real or merely nominal. In twenty-three states and the District of Columbia school medical inspection is mandatory in all districts, and in three states for certain districts only,

¹¹ *Twenty-second Annual Report, Bd. Health, Mass., 1890*, pp. 479-522.

as in Maine, in cities over 40,000; in New York, in all the counties except first-class cities, and in Wyoming, in incorporated cities and towns.

Physical defects are not equally significant either from the medical or from the pedagogical standpoint. Each kind of defect should be separately studied, and classification should not include pediculosis with defective vision; club-foot with defective hearing; adenoids with ringworm. The problem is essentially individual and needs separate consideration, examination, diagnosis, treatment and care, with efficient follow-up of each child.

The medical inspection of schools may be made more real and valuable by teaching the teachers. Virginia, by act of Assembly March 19, 1920, requires every normal school of the state to give a course, approved by the State Superintendent of Public Instruction and the State Health Commissioner in health inspection. No applicant may receive a certificate to teach in the schools of the state who does not present satisfactory evidence of having covered creditably an approved course.

Medical inspection of schools was organized in this country for the purpose of controlling the communicable diseases of childhood. It must at once be admitted that it has been a failure so far as this object is concerned, for it has had very slight influence upon the prevalence of measles, chickenpox, common colds, whooping-cough, mumps, etc. Theoretically we would expect a good system of medical inspection of school children to check the prevalence of these diseases. Perhaps it does so to a limited extent. With improvements in the system, correlated with child life outside the school, much may still be accomplished along these lines, especially with diseases such as diphtheria, scarlet fever, and smallpox.

There has been much discussion concerning who shall conduct the inspection. The medical inspector needs the coöperation of the specialist, the nurse, the teacher, and the parent. It is plain that in any system the teacher holds a key position, and teachers are quite competent to carry out simple tests such as determining the acuteness of vision and hearing. In one sense the teacher is the foster mother of the child and frequently knows the child better than its own mother. The teacher should report to the medical inspector children who show any of the following symptoms: loss of weight, pallor, puffiness of the face, shortness of breath, swellings in the neck, general lassitude, growing pains, rheumatism, flushing of the face, eruptions of any sort, cold in the head, especially running eyes, irritating discharge from the nose, evidence of sore throat, cough, vomiting, fever or frequent requests to go to the toilet, nervous twitching, fits, skin eruptions, and difficulties in understanding usual studies, or queerness of behavior.

The good system of medical inspection of schools has especial need of the services of a skilled psychiatrist, orthopedist, dentist, and director of physical training. This field illustrates the fact that economy consists not in saving money, but in spending it wisely. The National Child Health Council gives the following staff as a minimum that is desirable in a city of 25,000 to 30,000 people.

1 director of school health work	\$4,000
1 part-time physician	1,200
1 supervisor of health training and instruction	2,500
1 mental hygienist and supervisor of ungraded classes	2,500
3 school nurses \$1,500-\$1,800	4,500
1 part-time dentist	500
1 oral hygienist	1,500
	<hr/>
	\$16700

The cost per pupil on a basis of

6,000 pupils	\$2.78
5,000 pupils	3.34

The Council states that certain cities of this size are now spending more than \$5.00 per child on school health work. The next most important link in the chain of medical inspection is the school nurse.

The chief value of the school nurse, perhaps, is in establishing communication with the home and securing friendly coöperation with the parents. Parental neglect is rarely due to the lack of parental affection, but rather to ignorance. The nurse is frequently able to gain the confidence of both child and parent when the medical inspector fails. The nurse, further, will assist the medical staff in carrying out treatment. One of the chief duties of the school nurse is "follow up" social service work. The school nurse is able to detect the beginning symptoms of disease and can be trusted to care for simple troubles. Good service requires one school nurse for about 1,500 to 2,000 children.

The success of school medical inspection is measured largely by the coöperation obtained in the homes. The school nurse is the most effective instrument for securing this desirable coöperation. The most successful school nurse will have a background of sound heredity, tactful and equable temperament, good health, and be imbued with the love and understanding of children. In addition to regular training, school and hospital experience, she should have had training in public-health nursing, and practical instruction in the essentials of nutrition and the sanitary requirements of school buildings and grounds, an ideal seldom realized.

The duties of a school nurse will vary according to whether a school physician is employed on full-time or part-time basis, or none at all, and whether her work is in the rural or urban school. In schools where a physician is employed on a full-time basis the nurse's work should supplement that of the school physician and correlate with it. She should be directly responsible to the school physician for the proper discharge of her duties. However, certain duties are required of school nurses in general, irrespective of the type of school or character of the medical assistance, such as the daily inspection, instruction and disposition of children referred by the school physician or members of the teaching staff; routine classroom inspection at frequent

intervals for the purpose of detecting unreported or unnoticed cases of communicable diseases and noting hygienic conditions; giving health instruction to pupils and to teachers; and follow-up work in the homes.

At schools having a volunteer medical service or service of a school physician on part-time basis, the school nurse may properly engage in special work under the physician's direction, in addition to the routine duties already prescribed, with particular reference to the preliminary physical inspection of children for the more obvious physical defects in order to refer handicapped children to the school physician for diagnosis and advice with regard to notifying the parents.

Rural school nursing is a very different problem from that of nursing in urban schools, and is accompanied by many difficulties. Frequently the nurse will be required to perform all the general duties of a nurse in a school system having a full-time or part-time physician and also, in many instances, she may be called upon to act as a representative of the state or local health officer in so far as her work relates to the control of communicable diseases in the school.

The relative great importance of follow-up work by the school nurse as compared with her other duties is shown by the results of studies of sickness and absences among school children by the United States Public Health Service.¹² In the case of approximately 6,000 school children with over 666,000 possible days of school attendance for one year, 5.6 per cent, or 37,321 days, were lost on account of sickness, and 3 per cent, or 19,993, were lost on account of other causes. The school nurse, by giving instruction in personal and home hygiene and care of the sick and in the selection and preparation of food, should be able to shorten the duration of absence from sickness in individual instances, reduce the number of cases of sickness arising during the year, and lessen the number of absences from causes other than sickness. Follow-up work by the school nurse is required for the purpose of:

(a) Explaining the nature of notified physical and mental handicaps, the potential ill effects thereof on the child's health, school progress, and economic efficiency, and advising with regard to their correction;

(b). Explaining the nature of the quarantinable diseases and the necessity and importance of the prompt isolation of the sick for the protection of the community and other members of the family;

(c) Inquiring into the presence of open tuberculosis in the home in the case of children suspected of having tuberculosis;

(d) Inquiring into absence of more than two days' duration from unexplained causes;

(e) Securing the coöperation of the parents in health instruction of children, and enforcing in the home the regimen prescribed for children in special classes and schools;

(f) Distributing pertinent health literature prepared or approved by the state and local health departments; and

(g) Securing the coöperation of the parents in practicing in the home the

¹²U. S. Pub. Health Rep., July 8, 1921, Reprint 674.

principles of health protection and promotion taught in the regular and special classes and special schools.¹⁵

Duties of the Medical Inspectors.—It is the duty of the medical inspector to detect defects, not to treat them. Who shall treat the child is a matter for the parents or guardian to decide. It is not sufficient merely to notify parents that the child needs treatment, for frequently no attention is paid to the notices. The child may be referred to or taken by the school nurse to the hospital or outclinic. In some districts school clinics have been instituted with success.

An ideal system of medical inspection of schools would consist of a corps of trained and competent physicians and sanitarians who would devote their entire time to this special work. The staff should have the assistance of experts in ventilation and heating, experts in sanitary architecture, experts in sanitary engineering, and experts in the various medical specialties.

Specialists should visit all school buildings no less than three times each year in order to investigate all matters of heating, lighting, and ventilation, cleanliness, gymnasiums, bath, and toilets, and the seating arrangements with reference to the size of the pupils; the purity of the drinking water, the quality of the food purchased by the children at the recess period, and the general conditions of the neighborhood that may affect the health of the pupils.

Furthermore, coöperation between the medical and pedagogical departments should be helpful in solving the many difficult problems concerning the curriculum.

In addition to these general inspections all children entering school should be examined medically at least once during each school year. The first examination is for the purpose of establishing whether the child is fit for school and can do the work without injury to either its mental or physical well-being. The second should be a physical examination, which may be made more thorough if the child is required to strip. This, however, should not be done unless the parents of the child are present or give their consent. The third examination consists of special tests of the eyes, ears, nose, throat, teeth, heart, and lungs, weight, growth, nutrition, etc. Permanent and continuous records should be kept.

Aside from these regular examinations, the school physician must respond to every call when a pupil comes to school having an eruption, fever, or other symptoms indicating a communicable disease. The medical inspectors should also oversee disinfection, vaccination, and certify the return to school of any child who has been out of school by reason of a communicable disease.

On account of the tendency for tuberculosis to develop in children who have just suffered from measles, scarlet fever or whooping-cough, as well as to prevent other sequelæ of the acute infectious troubles, medical inspectors

¹⁵ U. S. Pub. Health Rep., September 8, 1922, Reprint No. 783.

should be detailed to visit and keep in close touch with such children for from three to six months, or for as long as may be necessary.¹⁴

The Communicable Diseases of Childhood.—Parents naturally come to regard the school as a veritable pesthouse for the spread of the communicable diseases of childhood—especially measles, whooping-cough, mumps, diphtheria, scarlet fever, chicken-pox, common colds, etc. Childhood is the most susceptible age for several of these infections. These diseases prevail in epidemic form during the summer time, or when school is closed, and under circumstances which show that their prevalence may be independent of school attendance. It is difficult to determine just what part is played by the commingling of the pupils in school in the spread of such diseases and what part is due to other factors. Some diseases take a sudden jump in the autumn with the opening of school because this is their natural seasonal habit. Further, these diseases are family epidemics and are carried home to the other members of the household, thereby creating secondary foci. It is a common experience for the six-year-old school child to bring measles and other diseases home and thereby give it to the younger brothers and sisters. The school is an advantageous place to immunize children against diseases for which we have specific and efficient prophylactics—smallpox, diphtheria, and scarlet fever. This problem of the communicable diseases and the schools is far from solution; the spread of these diseases has not been conquered by medical inspection, and their relation to school attendance is one that needs careful observation and study.

A difference is made between exclusion on account of disease and that due to exposure. In the latter case the period of exclusion is based upon the period of incubation of the disease. Immunes are not excluded. Carriers should be looked for. With regard to the exclusion of individual children, the indiscriminate exercise of this practice often leads to unjustifiable loss of attendance. Thus, in three schools, in two of which there were no actual cases of measles, 3,737 attendances were lost by exclusion of contacts, and yet not a single contact developed measles. Azoulay suggests separate quarters and special classes for contacts, and also for convalescents.

Closing Schools on Account of Epidemics.—The question of closing the schools when some one of these diseases breaks out is often a difficult one to decide. If the children commingle out of school, upon the streets and playgrounds, no useful purpose is accomplished by closing the schools. Hence closing schools is usually more effective in sparsely settled country districts than in cities. Closing schools is economically wasteful and usually has no influence on the course of an outbreak. Children are less apt to infect each other in the classroom than in the home or on the playground. As a rule better results will be achieved by daily inspection of all school children than by closing the schools. At the beginning of an outbreak the schools may be

¹⁴ S. W. Newmayer, *Medical and Sanitary Inspection of Schools*, Lea & Febiger, Philadelphia, 1924.

closed for the period of incubation and then opened, but careful guard must be exercised to discover new cases and a watch kept over the return of convalescents. Under these circumstances a daily inspection should be conducted before, and not after, the children enter school. If closing the schools during the period of incubation is not effective nothing will be gained by prolonging the period. No one thinks of closing a school just for a single case—not until a whole crop develops are aggressive measures taken, and then it may be too late.

The consensus of opinion now is that the successful control of epidemic disease in school children requires keeping the schools open, with careful daily and frequent periodic inspections; the exclusion of cases and contacts; and home visitation.

A special lookout must be kept for carriers, and laboratory facilities provided to detect the same.

The diseases for which children should be excluded from school are: smallpox, scarlet fever, measles (see also page 213), German measles, chickenpox, diphtheria, tonsillitis, whooping-cough, pediculosis, mumps, scabies, trachoma, ringworm, impetigo contagiosa, venereal disease, pulmonary tuberculosis, influenza.

Conservation of Hearing.—It has been found that approximately 15 per cent of school children possess some defect of hearing either in one or both ears. Defective hearing is frequently mistaken for inattention upon the part of the pupil, for which he may be unjustly punished. Practical tests to determine the acuteness of hearing should be made separately with each ear by the use of a watch or by the whisper voice before the child starts school. Discharges from the ears, known as abscesses in the ears, or earache should at once be reported to the proper medical attendant.

There is perhaps no field of hygiene so neglected as that of the conservation of hearing. Deafness may arise from many causes. The trouble is sometimes congenital, which is an inherited deficiency, and such cases are too often regarded as hopeless. Deafness is often the result of epidemic diseases of childhood, especially scarlet fever and measles, of defective tonsils and inflammation of the throat which passes up the eustachian tube into the middle ear.

Children who are hard of hearing should be seated where they can hear readily. The teacher should bear in mind the deficiency and not ascribe it to stupidity. By means of graduated exercises and appropriate treatment, much can be done to improve many cases.

Oral Prophylaxis—The Mouth, Nose and Throat.—The essential features of oral prophylaxis are (1) normal mucous membranes and associated structures, and (2) cleanliness. The health of the mucous membranes of the mouth and throat, including the closely associated glands and lymphoid tissues and the teeth, depends upon the general health of the body. Cleanliness helps avoid the immediate causes of caries and inflammation.

Flushing and rinsing the mouth with pure warm water, with or without a

pinch of salt, helps mechanically to cleanse the surfaces. Antiseptic mouth washes are a snare and a delusion. It is not possible to add any known germicidal substance to the mouth rinse that will sterilize or disinfect the buccal cavity. This is still more true with the mucous membranes of the nose and throat. Most of the germs are protected, and no disinfectant can reach those deeply imbedded, even when applied directly to the infected mucous membrane. Furthermore, any known disinfectant sufficiently powerful to kill bacteria would injure or destroy the delicate tissues and thereby might do more harm than good. Many of the mouth washes on the market vary but slightly in their composition from those described in either the Pharmacopeia or the National Formulary, which are just as tasty and much less expensive.

The indiscriminate use of douches and sprays may be harmful, by forcing infection up into the ears or sinuses. These should not be used, especially in the nose, without medical advice.

All ulcerated, inflamed or diseased conditions in the mouth, including the tonsils, throat, and nose, should be treated without delay and measures taken to prevent their recurrence. Healthy mucous membranes, sound teeth, and normal tissues are our best protection against infection.

The noses and throats of all pupils should be examined for any cause of obstruction to respiration, particularly adenoids, polypi, deviation of the septum, etc. Nosebleed should always be reported and inquiry should be made as to mouth-breathing during sleep. In all cases of acute illness the throat and mouth should be examined for indications of scarlet fever or measles and for the signs of tonsillitis or diphtheria, and a culture should be taken in any suspected case of diphtheria. The presence of a discharge from the nose should be noted, and if it is thick and creamy a culture should always be taken. If the discharge from the nose is only from one nostril a foreign body or local cause should be looked for. Adenoids may be inferred from mouth-breathing, snoring, chronic post-nasal catarrh, or recurring ear trouble. Pupils with obviously large tonsils, recurring tonsillitis, or enlargement of the glands of the neck should be referred to a physician for treatment. Infected tonsils and inflamed nasal and accessory sinus structures are often the site of focal infection. (See Focal Infection, page 1304.)

The Teeth.—The *teeth* are living sensitive structures, lying in sockets which resemble a bony joint. Strong teeth in a healthy body have a much greater power of resisting caries than otherwise. The teeth can even sparkle with the glow of good health. Teeth need exercise by eating the right kind of food in the proper way. A diet of soft, pulpy food weakens the teeth and invites trouble. On the other hand, the public must be taught to have a respect for the teeth, and should know that it is hazardous to use them to take the place of nut crackers or gas pliers.

Dental defects influence growth, resistance to communicable infections, preservation of facial symmetry, and degenerative diseases.

For the cleanliness of the teeth, we can well content ourselves with a

toothbrush, some silk floss and clean water. In young children, silk floss is not advisable if the space between the teeth is filled with soft tissue. In using silk floss, a rubber band or metal strip, care must be taken not to injure the gums. The teeth can also be polished with a piece of gauze or rubber wrapped about the forefinger. A little soap or some dental powder, free of grittiness, helps to clean the surface of the teeth, and will to some extent help to prevent the accumulation of tartar. Children should be taught the necessity of cleansing all the surfaces of the teeth, and the importance of keeping the toothbrush itself clean by occasional boiling and sunning. The eating of fibrous foods and the chewing of gum help keep the teeth clean. Alkaline mouth washes containing a little bicarbonate of soda, lime water, or magnesium oxid are used on the supposition that they help correct acid mouths and check the development of "acid spots," discoloration and tartar formation.

The teeth should be examined and cleansed by a competent dentist at least once, and preferably twice, a year. Dental hygienists can do much of the cleansing and simpler mechanical work. Dental clinics should be provided in which caries of the first set should have at least temporary treatment. In the light of our present knowledge it is an outrage to allow caries of the teeth to develop into toothache before children are taken to a dentist. Irregularities of the teeth, especially those which make it impossible to close the mouth properly, lead to faulty digestion, to mouth-breathing, and other defects. The first permanent molars (six-year molars) are perhaps the most important teeth in the mouth, and are the most frequently neglected because they are so often mistaken for temporary teeth. It is believed that decay of the teeth is hastened if not favored by the acid fermentation of carbohydrate foods and sugars, so that a factor in preventing dental caries is the removal of food particles by frequent brushing and the use of silk floss. Children should be discouraged from eating sugary and starchy foods of a pasty nature between meals, for these are apt to undergo fermentation with the formation of acid which etches the enamel.

Streptococcal and other infections are common about the roots of teeth, especially those that are devitalized or injured. Perhaps the most important structures to maintain the vitality of the teeth are the pulp cavity and the pericemental membrane. It is also important not to injure the margin of the gums where they join the teeth. Streptococcal inflammations and abscesses frequently form about the roots of such teeth, and sometimes give rise to secondary foci, causing neuralgia, stiff neck, rheumatic inflammation of the joints, sciatica, endocarditis, appendicitis, inflammation of the gall-bladder, and other serious complications. (See Focal Infections, page 1304.) These blind abscesses about the teeth should be sought for by radiography and corrected before secondary complications ensue.

Pulling teeth sometimes removes the focus of infection which relieves acute arthritis. When this happens the cure is dramatic, but unfortunately it is only occasional. Pyorrhea may not always be primarily a local infection,

but rather the expression of a general condition of malnutrition. One of the early symptoms of scurvy is swelling of the gums, loosening of the teeth and gradual absorption of the cement membrane of the alveolar sockets; in fact, the teeth are among the parts first affected in scurvy.

Focal Infections.—One of the recent revelations is the fact that a local focus of infection may cause distant, widespread and often serious consequences. The focus may be tiny and obscure, such as an abscess at the root of a tooth or deeply seated in the tonsils. The trouble is caused by a variety of microorganisms, but streptococci predominate. Either the microorganisms themselves leave the primary focus to settle in and inflame distant parts, or they produce toxic products which give rise to the symptoms.

The primary focus may be located in any part of the body. The most common and important sites are the tonsils, peridental membrane, the nasal and accessory sinus tissues. Infections in the genito-urinary as well as the alimentary systems may also act as foci of distribution. Foster gives the following in order of importance: (1) teeth, (2) tonsils, (3) sinuses, (4) gall-bladder, (5) prostate.

The most important types of trouble due to focal infections are arthritis, endocarditis, myocarditis and pericarditis; in fact, many cases of heart disease have their origin in some neglected or apparently insignificant focal infection. This gives the hint for prevention. Next in importance to the heart come the kidneys with nephritis, especially the glomerular type. Just as the primary focus may be located in any part of the body, the secondary effects may strike almost anywhere. Thus, we have cholecystitis, appendicitis, pancreatitis and various skin lesions, which may have their origin in some locus of infection at some distant point. Various "rheumatic" complications, neuralgic pains and vague manifestations of various sorts are also ascribed to focal infection.

The microorganisms associated with focal infections, which in turn are the cause of systemic trouble, are usually one of the streptococci. *S. hemolyticus* is commonly associated with the more acute forms, while *S. viridans* is often found in the subacute and chronic lesions, especially subacute endocarditis. A great variety of other microorganisms have been found, even the lowly colon bacillus. The infection is often mixed.

One of the curious unexplained phenomena in bacteriology is the tendency of bacteria to localize themselves in certain tissues. Some of them have their activity strictly limited by particular structures. These affinities sometimes show selective trophisms for certain tissues with a tendency to specific localization. The subject of elective localization has been studied and emphasized especially by Rosenow with experiments particularly with streptococci and pneumococci. He reports that sometimes the same lesion is produced in the same place in experimental animals as that found in the patient from whom the culture was obtained.

Dental Caries.—Caries of teeth is dissolution and disintegration of the enamel and dentine, often extending into the tissue of the pulp and gums. It

is the commonest of all defects of the body. Caries should not be confused with hypoplasia, abrasion, attrition or erosion. Caries is essentially a decalcification. In the light of our present knowledge, the development of sound teeth, capable of resisting destructive agencies, is fundamentally a dietary problem.

Caries of the teeth is a disease of civilization. Savages in vigorous health and with nutritious diet do not have decayed teeth, even though they do not use a toothbrush or practice oral prophylaxis. An examination of ancient Egyptian skulls shows that even the earliest races were not free from dental caries. It is much more prevalent among some races than others. Mummery has shown that it varies even among primitive races—from 1 to 0.4 per cent in the Eskimo, to 20.8 per cent in the Negro. Among civilized races the figures reach as high as 90 per cent.

Dental caries shows distinct periodicity, and occurs especially in childhood, during adolescence, in pregnancy and old age.

Caries is a disease with marked peculiarities. Usually the process is self-limited, but often it goes on to complete destruction of the tooth. It shows curious localizations. Caries occurs in teeth only during life; it is a decay which stops with death.

Causes of Dental Caries.¹⁵—The causes are complex and perhaps multiple. Diet appears to be the fundamental factor. The critical periods for the making of sound teeth are during fetal existence and the first two years of life. Nutrition and diet of the pregnant mother and the baby are therefore all-important to lay the foundation of strong teeth, which are able to resist the inroads of caries for life. In other words, teeth depend upon an endowment of an immunity which is their most efficient protection.

The causes of dental caries may be conveniently considered in the five following categories: (1) Anatomical factors, (2) bacteria of the mouth, (3) influence of the glands of internal secretion, (4) relationship of saliva, (5) influence of diet.

1. *Anatomical Factors.*—Defects in the structure of teeth may frequently constitute an important predisposing factor in caries. Nutritional disorders of the mother during the latter months of gestation may interfere with calcification of both the temporary and the permanent teeth. Instead of normal enamel or dentin there may be areas of hypoplasia on the surfaces or in the substance of the tooth, and these are liable to decay after the tooth erupts through the gum. Similarly, the exanthematous diseases of childhood, which usually occur during the period of tooth formation and growth, may leave their mark upon the enamel surface as pits, honeycomb parts or deep fissures. These hypoplastic areas are susceptible to caries.

Not only the structure of the teeth, but their position and relation to

¹⁵ References to the literature and all references noted in the discussion on the causes of dental caries will be found in the admirable review of John Albert Marshall, "The Etiology of Dental Caries," *Physiol. Rev.*, 1924, 4: 564, which contains a complete bibliography on the subject.

each other in the mouth is a predisposing factor. Ill-advised early extraction of deciduous teeth delays or modifies the development of the dental arches. Crowding or overlapping, particularly of incisors, cuspids and bicuspids, results. This favors the collection of food particles between the teeth.

The mechanical appliances used by dentists, such as the clasps on artificial dentures, a filling incorrectly placed or a poorly fitting crown may hasten the disintegration process. However, it is claimed that these latter causes may be regarded as comparatively insignificant when scrupulous care in hygiene is observed. On the other hand, it is sure that when malformed or malposed teeth are attacked the process may be more intensive than in sound teeth in proper alignment.

Cook suggests that differences in the structure of teeth may be due to the amount of magnesium, calcium and other inorganic salts in drinking water. He reports a noticeable increase in the prevalence of caries in school children where the drinking water contains only a trace of these salts, whereas in those waters with either temporary or permanent hardness, the percentage of diseased teeth is somewhat lower. Forberg in Sweden and Rose in Baden, Germany, have reported similar results. It will be recalled that in Baden goiter is very prevalent. The relationship between caries and abnormal thyroids, on the one hand, and iodine and drinking water, on the other, deserves more attention. Von Beust claims that there is difference in "vascularity" of dentin and enamel, and more recently Mummery has shown that there exist definite gradations in the degree of permeability of stains in young teeth as contrasted with the older ones. This is interpreted to mean that the calcification process may continue even after the eruption of the tooth.

2. *Bacteria of the Mouth and Their Relation to Dental Caries.*—Miller was the first to announce that decalcification of enamel and the subsequent invasion of the decalcified mass by acid-forming bacteria may be produced experimentally when teeth are immersed in cultures containing certain types of acid-producing microorganisms. He pointed out as early as 1883 that decalcification of tooth structure precedes the liquefaction of the organic matrix. Miller made it very plain that he did not wish to be understood as saying that bacterial action is the sole and only cause of decay of the human teeth. He made it clear that in his opinion there is not a single case of caries in which microorganisms do not play some part, and that in most cases they play a very important part. Miller's theory has long been the accepted viewpoint, namely, that acids are produced in the mouth, as a result of fermentation of food detritus and also that softened dentin owes its degree of decalcification to the local elaboration of acids by bacteria.

Following the work of Miller, a great literature developed concerning the particular organisms responsible for caries. Goadby and others agreed that they were unable to identify a specific microbe of dental caries, although most bacteriologists focused their attention upon *B. acidophilus*, the *Cladotrix placoides* and the *Leptothrix buccalis*. Lothrop and Gies investigated the

"influence of acid-producing bacteria in the focal precipitation of salivary mucin under relatively large masses of unaffected colloidal matter." Gies points out that all these researches do not explain immunity to caries, localization of caries or the sudden halt in the lesion. A reparative process may occur, in which there is an apparent "recalcification" of areas previously decalcified.

Eckermann presents a somewhat novel theory of the etiology of dental caries and ascribes a rather minor rôle to microorganisms. He thinks the process occurs in two stages. The first he considers may be produced by osmosis between saliva and blood-serum, assuming that the osmotic "membrane" consists of dentin and enamel. Eckermann states: "The hypothetical knowledge of the origin of dental caries being caused by lodging fermented carbohydrates has in no direction helped us to stop the increasing wave of decay. It is a singular fact that the teeth offer for many thousand years a resistance against decomposition when buried in the earth, even though some may be carious. Death stops instead of hastening decomposition of the teeth." We are, however, concerned primarily in eradicating decay of teeth in children rather than in skeletons.

MacIntosh, James and Lazarus-Barlow, after an exhaustive examination of selected carious material, differentiated a definite type of bacillus, which is capable of producing a high degree of acidity by fermentation of carbohydrates. The average P^H value of nine strains was 2.75, which is sufficient to decalcify teeth. It was also shown that teeth left in contact with pure cultures over prolonged periods show changes almost identical with those found in natural caries. Howe and Gillette agree, in spite of the findings of Kligler that the uncared-for mouth, regardless of the absence or presence of caries, contains a greater number of organisms than the clean mouth, that the type of organism is apparently the most important factor.

In summing up, it is apparent that bacteria are not the only factors concerned in dental caries. *B. acidophilus* is demonstrable in every mouth irrespective of the presence or absence of caries. Further, the acidophilic properties of different strains obtained from carious and non-carious conditions are the same. It appears, therefore, that both the environment and the tooth structure are more important factors than the types or numbers of organisms concerned. The kind of food, fibrous or non-fibrous, the efficient cleansing of tooth surfaces, the general health of the individual, the degree of nutrition—all of these factors are concerned in an etiology, formerly considered almost exclusively from the bacteriological standpoint.

3. *The Influence of the Glands of Internal Secretion.*—It has been suggested from time to time that malfunction of the "ductless glands" exerts an influence upon the formation of teeth, and in this way may be a factor in dental caries. In 1906 Erdheim destroyed portions of the parathyroid glands in rats and the surviving animals developed horizontal grooves on the surfaces of the upper incisor teeth. Afterwards, it was shown that these grooves were caused by an interference in calcium metabolism occurring

at the time when these particular teeth were being formed in the jaw. Howard from the human clinic contributes additional evidence in discussing hyperthyroidism by noting that the teeth show rapid decay and marked fragility. The pluriglandular therapy has at present scant support from the experimental evidence at hand. Gies and his co-workers observed deficient calcification of the incisors after removal of the parathyroids in rats. The literature, in fact, is replete with various views, but unfortunately the observations dealing with dental defects are scant and unconvincing; in fact, the dental aspect has not received the attention which it merits. There seems *a priori* reason for believing that the teeth are affected by endocrine influences.

4. *The Relation of Saliva to Dental Caries.*—Researches dealing with the changes produced by saliva upon the development or the retardation of dental caries were reported by Magitot in 1878. He considered the variable reaction of the saliva is due entirely to abnormalities in glandular activity. He also differentiated between those conditions in which the saliva is viscous and the mucin abundant and those in which the opposite obtains. It has often been observed that overactivity of the salivary glands is a symptom of various forms of stomatitis. In acute and chronic febrile disorders there is generally a variation both in the amount and composition of the saliva. Quinquerot states that an unclean mouth constitutes a predisposing factor in the production of dental caries and that dry mouths are immune. Furthermore, that mouths in which there is abundant saliva, especially if the latter is viscous, will surely exhibit signs of caries. There is little experimental evidence to support these statements. Kirk believes that the composition of saliva may possibly depend upon food habits and that the desired immunity may be obtained only through a more intelligent and rational diet.

Much attention has been given to the sulphocyanate content in saliva. Lohmann claims that sodium sulphocyanate prevents decay of deciduous teeth and allays pain. Bunting found that dental caries is present when the salivary analysis indicates a deficiency of potassium sulphocyanate. Gies, in 1913, doubted that the compound possesses any inhibitory action upon dental caries and also emphasized the unscientific and dangerous practice of its indiscriminate administration by dentists. The practice has fallen into disrepute.

Jeanneret studied the uric acid content of saliva and Felder the amount of nitrogen and albumen. Prinz discusses the problem from the standpoint of enzyme content of saliva. Brown calls attention to the fact that it is not only the acidity of saliva but its buffer index which is important, for the buffer index is the sum of reserve acidity and reserve alkalinity. The hydrogen-ion concentration of saliva is much emphasized nowadays but Prinz and others point out that the buffer value is the index to be sought rather than the actual acidity.

The saliva is a complex mixture and it is quite evident that it may well play a part in the production and the stopping of caries, but it is also quite clear that it is not the sole or even the important factor in this disease.

5. *The Influence of Diet on Caries.*—Both the physical and chemical properties of food have been shown to influence the function and health of teeth. Soft diets relieve the teeth of the usual amount of exercise in chewing. Under these conditions the teeth and the supporting structures suffer from lack of sufficient use, and this results in under-development of the jaws with a subsequent crowding of teeth. It is now well known that growth of the jaws is favored by well-directed exercise in order to prevent malocclusion. The physical characteristics of food therefore bear an indirect relation to caries, for a soft diet promotes disease, whereas a fibrous one in addition to its stimulating effect upon the process of mastication serves also as a cleansing agent on the surfaces of the teeth. Refining foods, such as flour and white sugar, has brought about the adoption of an unnatural diet, whereas bran, the wheat germ, and brown molasses, so scrupulously removed in processing, contain substances of value.

Frick early called attention to the fact that the artificial feeding of infants may be the main cause of dental degeneration as a result of faulty nutrition. In fact, he goes so far as to conclude that the predisposition to dental caries increased at the same ratio as bottle feeding.

It is apparent that there is uncertainty at this time concerning the relation between caries and the chemical composition of food. The added impetus to the study of nutrition which has been given by the discovery of vitamins and their attendant effects has thrown new light upon the dental aspect. The bacterial views of Miller upon the disease have not been overthrown, but his concept of caries is gradually being modified by the fact that experiments have been reported in which "caries-like lesions" have been produced experimentally.

Howe has produced abnormal teeth in rats and monkeys through restricted diets, consisting mainly in lack of antiscorbutic vitamin. He even found that recalcification occurred when green food in the diet was increased. Mrs. Mellanby describes unusual conditions of tooth formation and tooth position as a result of diet deficiency in vitamin A. An exhaustive study of the effects of different dietary elements on the teeth has been made by McCollum and co-workers. They produced "caries-like lesions," pulp exposure, osteo-dentin, and fracture and overgrowth of teeth. The type of diet which produces the greatest percentage of dental abnormalities was one in which there was a deficiency of protein, calcium and vitamin A.

SUMMARY.—The insidiousness of the onset of dental caries, the difficulties which are frequently experienced in controlling it, and the unknown factors which influence its cessation all are still matters of conjecture. Until the complete etiology has been worked out, the treatment will be one of repair rather than one of prevention. It appears, therefore, that caries is at least a twofold process in which defective structure of enamel and dentin is closely correlated with deficiency in diet and physiological abnormalities. Further than that, certain types of bacteria found in the mouth elaborate locally a sufficient amount of acid to decalcify tooth structure, and in this manner form

cavities. The factors tending toward an arrest of caries, however, have not as yet been recognized.

The importance of adequate nutrition during the prenatal period and early babyhood is repeated for the sake of emphasis. Heredity probably also plays a rôle here, as it does in so many other conditions affecting health and disease. Finally, among the accessory causes should be mentioned neglect of dental attention through ignorance of parents, cost of dental service, lack of dental facilities, especially in rural sections, failure of the child to call attention to the condition of the teeth either because it is too young or because of fear. The prevention of caries has been indicated and depends upon a better understanding of its causes.

Diseases of the Skin.—Apart from the exanthemata the diseases of the skin of school children which are of importance because communicable are: scabies, pediculosis, ringworm, and impetigo.

Scabies.—All children who are scratching or have an irritation on the skin should be examined for scabies (the itchmite). The parasites are usually contracted by direct contact, and also from animals. The staphylococcus causes the most annoying symptoms in scabies. It is important that all infected members of the family be treated until cured, else the disease is passed back and forth from one to another. It is also important that all clothing, bedding, towels, etc., and similar things that come in contact with the body be boiled each time they are washed. All cases of scabies should be excluded from school until cured. Sulphur ointment is usually efficacious.

Head Lice.—*Pediculi humani*, var. *corporis* are extremely common among children, and are communicated directly and also by wearing each other's hats or hanging them on each other's pegs, or from combs and brushes. Each child should have its own brush and comb. These should be cleaned by immersion in boiling water. No person should be blamed for having lice, only for keeping them. The condition may be suspected by the teacher in children who show indications of irritation of the scalp, and the cause is easily detected by looking for the eggs (nits), which are small white objects adhering to the hair. Head lice are best treated by killing the living parasites with crude petroleum and then getting rid of the nits. With boys this is easy; a close haircut is all that is needed. With girls whose hair is not bobbed a fine-tooth comb wet in alcohol or vinegar, which dissolves the attachment of the eggs to the hair, may be used. All combs and brushes used should be carefully washed and disinfected. Children with pediculi should be excluded from school until their heads are clean. For further prevention and discussion see page 370.

Ringworm.—Ringworm of the skin yields readily to treatment, but upon the scalp is usually chronic. When the disease attacks the scalp the hair falls off or breaks off near the scalp, leaving areas the size of a dime or dollar nearly bald. The scalp in these areas is usually dry and somewhat scaly, but may be swollen and crusted. The disease spreads at the circumference of the area and new areas arise from scratching, etc. The diagnosis is made by

looking for the fungus. The infection is not only transmitted from child to child but also from horses and cats.

Favus.—Favus is a disease somewhat allied to ringworm, more common in Europe than in America. In this disease quite abundant crusts of a yellowish color are present when the process is active. The roots of the hair are killed by the *Achorion schönleinii*, so that loss of hair from this disease is permanent, a scar remaining when the condition is cured.

Children with ringworm or favus should not be allowed to attend school unless the lesions are properly dressed and covered with collodion. Children should be taught to use their own brushes and combs and not to wear each other's hats, caps, etc. In some districts special schools are maintained for favus and for ringworm of the scalp, where the pupils are successfully treated with x-rays.

Impetigo.—Impetigo is a disease characterized by pustules which appear on the face, neck, and hands, less often upon the body and scalp. The size of the pustules varies very much and they often run together to form on the face large superficial sores covered with thick, dirty, yellowish, or brownish crusts. The disease is contagious and spreads by scratching as well as by using common towels and other things. Children having impetigo should not be allowed to attend school until all the sores are healed and the skin smooth.

Nervous Diseases and Mental Defects.¹⁶—A sharp lookout for indications of diseases of the nerves and of mental defects should be kept and especial notice taken of suggestive symptoms in a child who did not formerly show them. The teacher should be taught to report instances of restlessness or inability to stand or sit quietly in a previously quiet child, especially if to this are added irritability of temper and loss of self-control, such as crying for trifles or inability to keep the attention fixed.

Chorea.—Twitching of the muscles, the result of disease, may cause the child to drop things, render his work awkward, or interfere with writing or drawing. Such children are too often scolded for being inattentive or careless. The indications of chorea (St. Vitus's dance) should not be confounded with habit-spasms such as blinking of the eyelids or the slower twitching movements of the face or shoulders or other parts of the body, which may be due to defects of vision, adenoid growths, or other reflex causes. Cases of chorea should be removed from school at once, both for the child's safety and to prevent an epidemic of imitative movements such as sometimes occurs. Children with habit-spasms need not be withdrawn from school work, although these conditions often require treatment.

Epilepsy.—Mild epileptic attacks (*petit mal*) are frequently overlooked or misunderstood by the teacher. They may be mistaken for fainting. Usually these attacks are only momentary, in which the child stares fixedly and does not reply to questions or in which he suddenly stops speaking or whatever he is doing and is unaware of what is going on about him. The lapse

¹⁶These subjects are discussed in detail pages 435 to 439.

of consciousness is one of the characteristic features of epilepsy. The attack may be accompanied by rolling up of the eyes, drooling, or unusual movements of the lips; an epileptic fit often appears like a choking attack. Teachers very frequently misunderstand epileptic attacks and cannot be expected to distinguish them from hysterical convulsions and other diseases. It does not necessarily follow that cases of epilepsy should be withdrawn from the school, but medical advice should always be had.

Neurasthenia.—Neurasthenia or nerve fatigue may be shown by irritability or sleeplessness and other indications threatening a nervous breakdown. This may be due to irregular habits, want of proper sleep, lack of suitable food, poor hygienic conditions, or simply from the child being pushed in school beyond his physical or mental capacity. Excessive fear or morbid ideas, bashfulness, undue sensitiveness, causeless fits of crying, morbid introspection, and self-consciousness may also be symptoms of a neurasthenic condition, and call for investigation and for the teacher's sympathy and winning of the child's confidence to prevent developments of a more serious nature. Excitability is often the first stage of fatigue and is frequently mistaken for brightness and therefore encouraged.

The teacher should know that forgetfulness, loss of interest in work and play, desire for solitude, untidiness in dress or person, and like changes of character are sometimes incidental to the period of puberty.

Defectives.—Mentally defective children in the public schools exhibit certain common characteristics which soon become evident. The typical incorrigible child of the primary grades often is a mentally defective child of the excitable type. They are destructive, cruel to smaller children, and often precocious sexually. Certain cases show marked moral deficiency. Mentally defective children must be distinguished from those who are only temporarily backward as a result of some removable cause such as defective vision, impaired hearing, adenoid growths, or as a result of unhappy home conditions, irregular habits, want of proper sleep, lack of suitable food, bad hygienic conditions, etc. Teachers should refer to the medical inspectors for examination children who, without obvious cause, such as absence or ill health, show themselves unable to keep up in their school work, who are unable to fix their attention, or are incorrigible.

A careful lookout should be kept for children showing sexual perversion, for one sexual pervert may demoralize a whole school.

Vaccination.—Vaccination should be required of all children before they are permitted to attend school. The evidence of a successful vaccination usually accepted is a physician's certificate or a characteristic scar. For the indices of a successful take, see page 3. School children should be vaccinated before entering school and again before entering high school.

SECTION XVI

DISINFECTION

CHAPTER I

GENERAL CONSIDERATIONS

Disinfection.—Disinfection means the *destruction of the agents causing infection*. An object is said to be infected when contaminated with pathogenic microorganisms. It is disinfected by destroying these organisms, whether they are in the substance or on the surface of that object. Disinfection, then, deals only with destroying the vitality of those minute forms of life which cause disease. It does not mean the destruction of all the lower forms of animal and vegetable life that may be in or upon an object—this is sterilization.

Disinfecting procedures properly applied have an important part in the prevention of communicable diseases. Their efficiency decreases as the distance between the place of origin of the infection (the patient) and the point of their application is increased. Hence the emphasis upon concurrent rather than upon terminal disinfection.

Sterilization.—*Sterilization means the destruction of all microbial life on or in an object*. A sterile object is a lifeless object. All processes which sterilize necessarily disinfect, but all disinfecting processes by no means sterilize. The distinction between disinfection and sterilization arises principally from the fact that spores have a much greater resistance to all influences which destroy the vegetative cells. Fortunately, none of the pestilential diseases of man which occur in widespread epidemics, so far as known, are caused by microorganisms with resistant spores; therefore the usual processes of disinfection may be thoroughly efficient, yet leave many harmless and hardy forms of microscopic life alive. In other words, sterilization is rarely necessary in public health work, except in the case of anthrax, tetanus, botulism and other spore-bearing infections, such as malignant edema and the gas bacillus group.

Antiseptics.—Antiseptic substances *prevent decomposition and decay*. Such substances retard or prevent the growth and activity of microorganisms, but do not necessarily destroy them; that is, antiseptics delay or prevent fermentation and putrefaction without destroying the microorganisms which cause the processes. There is a great difference between the antiseptic and the disinfecting power of most substances. For instance, a solution of formalin will restrain the development of most bacteria in the proportion

of 1 to 50,000, but it requires a 3 to 10 per cent solution of this liquid to kill the bacteria in a reasonably short time. As weak a solution of bichlorid of mercury as 1 to 300,000 will sometimes prevent the germination of anthrax spores, whereas it requires a 1 to 1,000 solution to destroy them. Saturated solutions of salt or sugar will preserve meat, vegetables, and other organic substances; that is, they are antiseptic in their action but not germicidal, as they have small powers of destroying microorganisms. Cold is also antiseptic or preservative in action, not germicidal.

Asepsis.—Asepsis means *freedom from or absence of living pathogenic microorganisms*. Surgical asepsis may be achieved through physical cleanliness without the use of active germicidal substances.

Germicide.—A germicide is *a substance or agent which destroys germs*. Germicides and disinfectants are interchangeable terms, as both are used to indicate the destruction of microorganisms. Some germicidal processes used in public health work are potent enough to sterilize objects with which they come in contact.

Deodorant.—A deodorant is *a substance which has the power to destroy or to neutralize unpleasant odors*, such as those arising from organic matter undergoing fermentation or putrefaction. Such substances must be distinguished carefully from disinfectants. Deodorants destroy smells; disinfectants destroy germs. Many of the disinfecting agents are also deodorants, but all deodorizing substances are by no means disinfectants. For example, charcoal will absorb the malodorous gases arising from putrefying and fermenting materials, but it is inert so far as its power to destroy the cause of these processes is concerned. Formaldehyd, chlorin, and ozone, on the other hand, are true deodorants and disinfectants, as they combine with the organic matter to form new compounds which are both odorless and sterile. Bichlorid of mercury, while a very potent germicide, has practically no immediate effect upon odors. The volatile oils and other substances having a pungent odor are not deodorants; they simply cover up one smell with another. Deodorants have small value in public health work. For the most part they are a snare and a delusion and a waste of money. The best deodorant is cleanliness (see pages 920 and 1315).

Fumigation.—Fumigation consists in *liberating fumes or gases with the object of destroying vermin, insects, rats, mice and other small animals acting as carriers of infection, and sometimes also germs*. The chief fumigants used in public health work are formaldehyd, sulphur dioxid and hydrocyanic acid gas; to a less extent carbon monoxid, funnel gases, pyrethrum, carbon disulphid, chlorin, etc. Most of these are very poisonous to higher forms of life, but have little or no germicidal power—excepting formaldehyd, which is a good germicide but feeble insecticide. Fumigation does not take the place of disinfection; gases at best have but the merest surface action. As they lack the power of penetration they cannot be depended upon to disinfect even thin fabrics, or surfaces soiled with sputum or similar discharges in which the virus is protected against the fumigating gas.

Fumigation is chiefly useful in preventing the spread of insect-borne diseases. See page 268.

The terms fumigation and disinfection have been much confused. Because *fumigation* is ineffective as a germicide and unnecessary after some of the bacterial diseases—the word has gone forth that *disinfection* is unnecessary and useless. No mistake could be greater. Fumigation has its place and its limitations in public health work—so has disinfection.

Nature's Disinfecting Agencies.—In nature many forces are constantly at work to destroy infection and thereby limit the spread of the communicable diseases. It is the duty of the sanitarian to encourage the use of these natural disinfecting processes; they are *dilution, sunlight, dryness, time* and *antibiosis*. Sunlight is a great destroyer of germ life. Few microbes, especially the pathogenic ones, can live in the direct bright sunlight many hours. Dryness is another natural condition that is destructive to many of the minute forms of life with which we have to contend. The combination of dryness and sunlight is quite as good, if not better, than the ordinary fumigating processes which are commonly used in practical disinfection against surface contamination. Dryness, sunlight, and cleanliness are the keynotes of sanitation in the modern acceptance of the term.

We now know that most of the pathogenic microorganisms do not grow and multiply in our environment. For the most part they soon die when wafted into the air, deposited on surfaces, conveyed in water or placed in the soil. It is only occasionally that some of them find conditions favorable for development in foods such as milk and meat, and exceptionally in water. Further, it is to be noted that ordinarily it requires a certain number of microorganisms to produce infection. It is quite likely that a single typhoid bacillus or a single tetanus spore may kindle a conflagration, but experimental evidence with the infections upon laboratory animals teaches the lesson that ordinarily an animal is capable of taking care of minute amounts of infection, particularly if attenuated. It requires at least 10 tubercle bacilli to cause tuberculosis in a guinea-pig. Dilution, attenuation, and the conditions of our environment, unfavorable to most germs harmful to man, therefore protect us in no small measure against the communicable diseases. This is one reason why so many of the communicable infections are usually contracted rather directly through close personal contact.

Cleanliness.—Cleanliness is a very important adjunct to the work of disinfection. In fact, cleanliness lies at the base of all our sanitary measures. The mere act of cleaning removes some of the adherent microbes from the surface and the ordinary scrubbing and washing result in the final destruction of many more. Dry dusting and sweeping serve only to stir up dust and infection, which settle down again upon the same or other surfaces. Cleanliness serves another important purpose, so far as infection is concerned; it removes the organic matter on which and in which bacteria may find favorable conditions for prolonging life and virulence. The modern conception of cleanliness has expanded with the growth of the sanitary sciences. We now

aim at biological cleanliness as well as æsthetic cleanliness. This includes not only the removal of organic matter, but the destruction of insects and vermin, and their feeding and breeding places (see page 267). So far as personal cleanliness is concerned, the two important acts to prevent infection are: (1) Washing the hands before eating, before handling food and after leaving the toilet; and (2) keeping the fingers away from the mouth and nose.

The surfaces frequently used or handled by the public, such as woodwork, seats, floors, desks, door handles and the like in schools, stores, factories, shops and public conveyances and assembly places should, when practicable, be frequently scrubbed with hot soapsuds and strong soda solution. This also includes the seats of water-closets and privies, wash-basins, and other objects used in common.

In the wholesale disinfection which must be practiced to check widespread epidemic diseases due to bacterial infection we are largely limited to the use of the agents which nature has constantly at work to destroy such infection. Against a single case of communicable disease or against a limited infected area we may employ aggressive measures such as steam and strong chemicals; but when a disease, due to bacterial infection, has spread over an extensive district, these methods must be supplemented by all the resources of nature. The people must be educated so as individually to employ intelligent measures to avoid the infection. Cleanliness must be more scrupulously practiced than ever, sunlight and dryness must be given their fullest opportunity to operate even at the expense of a few faded carpets or colors. Cleanliness from another standpoint is discussed on page 920.

Antibiosis and Symbiosis.—Many pathogenic microörganisms are destroyed in the process of putrefaction and fermentation. They die in the fierce struggle for existence going on in the process of decomposition. For the most part the hardier saprophytic forms of life overpower and kill the disease-producing microörganisms which have comparatively feeble powers of resistance. The fact that infected carcasses, sewage, and putrid organic matter generally purify themselves by the very processes that destroy them is a fortunate provision of nature.

Ordinarily, only one major infection is active in the same individual at one time. Symbiosis is noted in the case of tetanus, which is favored by other microörganisms. Influenza and measles invite infection with the pneumococcus and predispose to tuberculosis. On the other hand, there are curious instances of antibiosis; thus, sarcoma may disappear after erysipelas; malaria influences parietic dementia.

When and Where to Disinfect.—It is much better to prevent infection than to be compelled to destroy it after it has become disseminated through ignorance, carelessness, or negligence. It is the duty of the disinfecter to destroy infection wherever it is found; it is the ideal of the sanitarian to prevent its broadcasting.

Man is the fountain-head of most of the infections to which he is heir; hence, the best place to apply disinfection is at the seat of origin of the

infection. The excretions, especially those from the mouth, nose, and bowels, as well as discharges from eruptions and wounds most frequently need attention. When proper precautionary measures have been taken at the bedside with a case of cholera, typhoid fever, or diphtheria there is little need of subsequently disinfecting the sick room, but when a diffusion of the infection results then a general purification becomes necessary.

Qualifications of the Disinfecter.—The disinfection of any given place is a complex operation, and should not be attempted by anyone not familiar with the peculiarities of the particular infection with which he has to deal and a thorough knowledge of the disinfecting agents employed. In other words, it is quite as important to know *what* to disinfect as *how* to disinfect and *when* to disinfect. A thorough understanding of the causes and modes of transmission of the communicable diseases is the most useful weapon the disinfecter has in his fight against the spread of infection.

Success depends upon personal attention to minute details. Germs are little things, and it is little things that count in this kind of work. The disinfecter who is satisfied to leave the process in the hands of an inexperienced person with a few words of instruction cannot expect to obtain trustworthy results. The disinfecter must give personal surveillance to the whole process—the materials, the strength of solutions, modes of application—and must guide and direct every step of the operation. He may profit by the example of the conscientiousness and thoroughness with which the surgeon assures himself of every detail of asepsis in his operating clinic.

Much of the routine fumigation by departments of health is probably ineffective, although the procedure be faithfully carried out. The average fumigating squad does not understand the effect of temperature, humidity, outside winds, porosity of walls, shape and size of enclosures, and the rate of application and other factors of the gases employed.

Controls.—Disinfecting processes should be controlled from time to time. Cultures containing spores and also non-spore-bearing bacteria placed in various parts of the apparatus should be exposed as a guide and check to the thoroughness of the process. Thermometers should be in duplicate and occasionally standardized. An admirable method of controlling each run is by means of mechanical regulation and automatic registration of the time and temperature in both steam and hot-air processes. Emphasis should always be placed upon the point that the vital temperature is the temperature of the object to be disinfected rather than that of the apparatus.

For the control of gaseous disinfection, saturate threads with an active culture of *B. prodigiosus*. These threads are attached to little slips of paper which are then exposed in various portions of the room to be treated. After the completion of the operation the threads are inoculated into Dunham's peptone medium. If the *B. prodigiosus* has survived the characteristic red color appears in the culture medium.

Disinfection Must Be in Excess of Requirements.—The disinfection of rooms, bedding, ships, and objects that have been exposed to infection must

of necessity be greatly in excess of the actual requirements. This is one of the difficulties met with in attacking an invisible foe. A sick room might readily be disinfected and rendered safe by applying a gill of germicide to the infected spot. But, as we cannot see the germs, it is necessary to apply our disinfecting agents to all parts of the room and its contents that have been exposed to the infection, in order not to miss that particular spot. In practical work a large factor of safety is sound and sensible. At first disinfection was directed by a shotgun process in a general sort of blunderbuss way against everything, but now that we know more about the habits and habitat of each one of the particular microorganisms we can concentrate our efforts with some exactness upon the particular objects liable to transmit infection, and with greater assurance of eradicating danger.

Specificity of Germicides.—There are few universal poisons and few if any general germicides. Most disinfecting substances are more or less specific in action. Germicidal agents often show marked selective action towards bacteria, spirochetes, protozoa, algæ or the “ultra microscopic” viruses. Thus, phenol and the cresols have comparatively feeble action against the virus of smallpox, vaccinia, and other filtrable viruses. Copper sulphate is a specific poison against algæ. Sodium oleate favors the growth of Gram-negative cocci of the *Micrococcus catarrhalis* group and of staphylococci, while pneumococci and streptococci of the hemolytic and *S. viridans* variety fail to develop.¹ Fuchsin (Endo’s medium) suppresses *B. coli*, but does not inhibit *B. typhosus*. Glycerin kills non-spore-bearing bacteria, but preserves filtrable viruses. Gentian violet and other para-rosanilin dyes kill Gram-positive, but not Gram-negative microorganisms. Proflavine, acriflavine and mercurochrome are specific for Gram-positive bacteria. Bile salts dissolve pneumococci and restrain the growth of staphylococci and streptococci, but favor the growth of typhoid and colon bacilli. Ethylhydrocuprein (optochin) is specific for the pneumococcus. Acridin with mercuric chlorid has remarkable properties of penetrating tissues. There are many other examples of selective action. Hence, the killing of *B. typhosus* or any other test organism is not a proof of general disinfecting power. There is, therefore, a tendency towards the development and use of specific germicidal agents.

Chemotherapy.—Most cells of the body are more easily killed than bacteria. All the ordinary germicides used in public health work are quite destructive, even corrosive to the tissues of the body. For a long time the only specific remedies in practical use were quinin for malaria and mercury for syphilis. Since Ehrlich, in 1910, found that “606” (salvarsan) will kill spirochetes without serious injury to the body, the search has been to find specific substances that will kill the parasites without harming the host. Chemotherapy goes on with rewardful results. We now have tartar emetic for leishmaniasis and atoxyl for trypanosomiasis (sleeping-sickness), and other, specifics useful in prevention and cure.

¹ *J. Am. M. Ass.*, 1918, 71: 1050.

The Ideal Disinfectant.—The ideal disinfectant must first and foremost possess a high germicidal power. It must not be rendered ineffective by the presence of organic matter; it must be reasonably stable, so as not to deteriorate under ordinary conditions; it must be soluble or readily miscible in water; if it forms an emulsion the emulsion should be permanent; it should be harmless to man and the higher animals; it should have the power of penetration; it should not corrode metals, bleach pigment, rot fabrics, or stain surfaces; and, finally, it should be reasonable in price.

The stress of modern activities demands disinfecting processes that are instantaneous in their action, all-pervading in their effects, cheap, harmless, and free from unpleasant odors that might be offensive to the fastidious. Such perfect disinfectants are not known. It requires money and the expenditure of well-directed and intelligent energy to accomplish satisfactory disinfection. *No one germicide is applicable to all diseases or to all substances, or even to the same disease or the same substance under different conditions.*

Concurrent Disinfection.—Concurrent disinfection signifies the immediate disinfection and disposal of all infected material during the course of the illness. It implies the prompt disinfection or destruction of all infected discharges and of all articles soiled by them. Furthermore, it includes the purification and cleanliness of the immediate environment of the patient so as to check the spread of infection.

Terminal Disinfection.—Terminal disinfection signifies the measures taken to destroy or purify infectious material after the recovery or removal of the patient or the termination of isolation or quarantine. Terminal fumigation finds its chief usefulness in fighting the insect-borne diseases.

Uses of Disinfection and Fumigation.—The distinction between fumigation and disinfection must be kept clearly in mind (page 1314). Terminal fumigation for measles and certain frail and short-lived viruses is not necessary. Furthermore, gases such as formaldehyd are uncertain in practice, and have the merest surface action. They cannot be depended upon against tuberculosis or diphtheria. New York and several other cities have omitted terminal fumigation since 1913 in cases of scarlet fever and in several other diseases. Both concurrent and terminal disinfection of discharges and objects likely to convey infection will always remain an important measure of prevention.

Terminal fumigation for the bacterial viruses has been discontinued as a public health measure because it has little effect upon the control of such infections and the cost of such "disinfection" appears to be disproportionately large to the benefits.² The evident limitations of terminal fumigation have cast doubt in the minds of some health officers upon the value of disinfection in general. This is an unfortunate attitude. No one can question the

² We are indebted to Charles V. Chapin, Health Officer of the City of Providence, R. I., for early and vigorously pointing out the futility of much of the terminal "disinfection." See *Sources and Modes of Infection*, New York, 1912.

great value of disinfection properly applied. It is, of course, much more important to destroy the infective discharges throughout the course of a case of typhoid fever than to trust to one final disinfection of the sick room and its contents. The same holds with about equal force for most of the communicable diseases. We now know that fomites play a comparatively minor rôle in the transmission of disease. The disinfection of rooms and objects does not now, therefore, hold the importance in the minds of sanitarians that it once did. However, if terminal disinfection prevents the occurrence of only a small number of cases it would still seem to be worth while. Moreover, what health officer would willingly allow his child to occupy the bed or handle the objects in a room soon after a case of typhoid, scarlet fever, tuberculosis, or diphtheria without first applying some effective method of purification? The greater the care and cleanliness exercised during the progress of the disease the less the need of terminal disinfection. The boiling of fabrics and a good cleansing of surfaces with soap and water, followed by an application of bichlorid, carbolic acid, or one of the cresol preparations is a more effective method of disinfection than formaldehyd gas, which is the best of the gaseous agents. Cleansing, renovating, airing and sunning of the room should always be the final process.

The principal *objects that need disinfection* are the discharges from the body; towels, bedding, handkerchiefs and fabrics; food, tableware and other objects that have been mouthed, and finally the hands of the nurse, physician and others who come in contact with the infection.

Penetration.—In practical disinfection a certain amount of penetration is almost always called for. Most germicides penetrate poorly and slowly. As a rule, substances in solution penetrate better than substances in emulsion. Gaseous substances cannot be depended upon to penetrate at all. They have only the merest surface action. Feces and sputum are not only the most difficult to penetrate, but also the most important because so apt to contain virulent pathogenic microorganisms throughout their mass. No germicidal agent can be depended upon to permeate a fecal mass under ordinary conditions in a reasonable time. It is, therefore, most important that such substances be thoroughly disintegrated and the germicide incorporated through the mass. Bacteria in nature are usually imbedded in various substances which differ greatly in consistency and composition, and therefore practical disinfection calls for stronger solutions and longer times than indicated by laboratory tests upon the naked germ cells. In certain instances, where penetration is required, trust should be placed only in steam or boiling. Dry heat has poor powers of penetration compared with steam. The penetration of steam and of gases is facilitated by a prior vacuum. None of the ordinary chemicals can be trusted to penetrate upholstered furniture, mattresses, pillows, thick blankets and the like.

Organic Matter.—Organic matter seriously interferes with the efficiency of almost all germicidal agents. Chlorinated lime, ozone, hydrogen peroxid, potassium permanganate, and other active oxidizing agents attack organic

matter with avidity and are thus soon used up. The metallic salts coagulate organic matter, thus automatically impeding further ingress. Formaldehyd and phenol show less reduction of power in the presence of organic matter than perhaps any of the other germicides.

In nature, bacteria are usually imbedded in organic matter. The way in which organic matter handicaps germicidal action has been shown by many investigators. Behring found, for example, that anthrax bacilli suspended in water are killed in a few minutes with bichlorid of mercury 1:500,000; in bouillon it required 1:40,000; while in blood-serum 1:2,000 was not always effective.

Time.—Time is an essential factor too frequently disregarded in disinfecting with liquids—suspension or solution. Very few chemical disinfectants act instantly, even in strong solutions and under favorable conditions. The microorganisms are so often in clusters or are surrounded by mucoid films or are so imbedded in colloidal albuminous masses that a considerable time is required for the disinfecting solution to penetrate to the germ. If the microbes are dry it takes a certain time to wet them before the chemical can act. These and other factors must be added to the time actually necessary for the substance to destroy the life of the germ after it comes in direct contact with it.

It is well known that some germicides, particularly the metallic salts, if given sufficient time, will eventually kill in exceedingly weak dilutions. Mercuric chlorid, according to Chick and Martin, will act as a germicide in a dilution of 1:1,000,000 if given sufficient time. The action of copper sulphate in exceedingly weak dilutions on algæ is also of interest in this connection. Some disinfectants, like hypochlorite, chlorin, permanganate, and hydrogen peroxid, on the other hand, exert their most useful action promptly, and are then rapidly used up by being oxidized or neutralized and thus lose their power.

Chick found a logarithmic ratio between concentration of disinfectant and the time taken to disinfect. She found, furthermore, that the phenol coefficient of mercuric chlorid with *B. typhosus* varies greatly with the prolongation of exposure. Thus:

Phenol coefficient of HgCl_2 2.5 minutes exposure = 13.5 coefficient.

Phenol coefficient of HgCl_2 10 minutes exposure = 175 coefficient.

Phenol coefficient of HgCl_2 30 minutes exposure = 550 coefficient.

This is an interesting side light on the Rideal-Walker technic which, until recently, permitted a latitude of time of comparison varying from two and one-half to fifteen minutes. This wide variation of the coefficient, however, is found mainly in the case of the metallic salts.

Time is an exceedingly important element in disinfection. It is not sufficient simply to dip the hands in and out of a bichlorid solution, to rinse fabrics in carbolic acid solution, or to pour formalin over feces. It takes time to penetrate and then to kill. Further, the speed of the reaction varies

with each substance, and depends upon the concentration and also the temperature.

Speed of Disinfection and Stability of Disinfectants.—A knowledge of the speed with which a disinfectant acts is essential to an understanding of the conditions under which it may be used to best advantage. The speed of disinfection is an important factor, for it varies enormously with different types of substances. Germicides of the chlorin group and iodine are among the most rapid, while dyes and some metallic salts are relatively slow. As a general rule, germicides that act promptly are quickly decomposed or neutralized. Stable germicides act slowly, unstable germicides act quickly. A list of germicides with reference to these factors follows:

INORGANIC DISINFECTANTS

Hydrogen peroxid and some of its derivatives	Unstable, easily decomposed during disinfection
Chlorin	Unstable, easily decomposed during disinfection
Hypochlorous acid and its salts	Unstable, easily decomposed during disinfection
Bromine and iodine	Less unstable than chlorin
Boric acid and its salts	Stable
Mercury salts	Often inactivated by precipitation or otherwise
Bismuth salts	Often inactivated by precipitation or otherwise
Zinc salts	Often inactivated by precipitation or otherwise

ORGANIC DISINFECTANTS

Alcohol, ether, etc.	Stable
Iodoform	Fairly stable
Formaldehyd	Unstable
Hexamethylenetetramine and its derivatives	Mostly stable
Aromatic chloramins	Unstable, easily decomposed during disinfection
Phenols, naphthols and derivatives	Mostly stable
Dyes, such as malachite green, acriflavine, etc.	Mostly stable, though sometimes reduced to leuco-forms and often adsorbed by tissues

Temperature.—There is a complete analogy existing between a chemical reaction and disinfection, one reagent being represented by the disinfectant and the other by the protoplasm of the bacterium. Chick states that the velocity of disinfection increases with the rise in temperature in a manner similar to the curve of a chemical reaction. In fact, the temperature so greatly influences the disinfecting power of liquids that it is strongly recommended *always to use warm or even hot solutions in actual practice*. Slight changes of temperature make a great difference. Feeble antiseptic solutions become strong germicides when warmed. Phelps claims that as the temperature increases arithmetically, the velocity of reaction increases geometrically.

This is not a general rule applicable for all disinfectants, for Chick showed that the germicidal power of the metallic salts increases 2- to 4-fold for each rise of 10° C., while phenol usually rises 7- to 8-fold for each similar change in temperature.

A good instance of the effect of temperature is given by Heiden, who found that anthrax spores which survived the effects of a 5 per cent carbolic solution for thirty-six days at room temperature were destroyed in half an hour in the same solution at 55° C. At 75° C. it took only three minutes to kill them. A 3 per cent carbolic acid solution killed the same spores at this temperature in fifteen minutes and a 1 per cent solution in from two to two and one-half hours.

Temperature of Disinfecting Apparatus.—It is important to remember that *the temperature registered by the thermometer of the apparatus is rarely a true index of the temperature of the things to be disinfected.* This is a common source of error in the use of autoclaves and sterilizers where large objects or masses are heated, such as cans of food, bundles of bandage, bales of fabric, or quantities of clothing. The temperature recorded on the thermometer of the sterilizer is usually higher than the actual temperature within. Good sterilizing technic is essential and a factor of safety desirable. The time necessary for penetration must be taken into account. Thermometers should be standardized and rechecked from time to time.

The critical temperature in the center of bales, bags, cans, etc., can be determined with the thermo-couple or special recording thermometers. In the use of steam under pressure, the temperature is a more reliable guide than the pressure.

Emulsions and Solutions.—As a rule an emulsion has greater germicidal power than a solution. Thus soapy and resinous emulsions of the phenols may accentuate the germicidal power of these substances. Chick and Martin have observed that the particles of an emulsion or soapy preparation of the coal-tar acids exhibit active Brownian motion. The bacteria are considerably larger than the mean diameter of the emulsified particles. The bacteria may plainly be seen to be bombarded by these particles. In this way the bacteria are frequently brought into intimate contact with the undiluted particles of pure coal-tar acids. The maximum effect may therefore be obtained and the death of the bacteria is inevitable. Such concentration about the bacteria is not likely to occur with substances in solution. The coal-tar acids in suspension act upon the bacteria first through adsorption, and then through chemical combination. The bacteria rapidly become surrounded by the disinfectant in a much greater concentration than actually exists within the liquid. Other particulate matters present have the same power of adsorption, and their presence therefore interferes with the germicidal value of substances in emulsion. Thus the value of phenol in solution is barely impaired by the presence of organic matter while emulsified disinfectants are reduced to one-third or one-half their original value. That germicidal substances in emulsion fail to penetrate may be demonstrated by pouring one of the coal-tar emulsions

upon a fecal mass; a layer of the coal-tar creosotes soon collects upon the surface, plainly visible as a film.

Dilution.—There must be a sufficient concentration of the substance used so that it shall be present throughout the whole mass in the proportion required. Thus an agent that is effective in a 2 per cent solution cannot be used in that strength to disinfect an equal volume of an infected liquid, since the mixture would then contain but 1 per cent. This is particularly important in the disinfection of urine, feces, sputum, vomitus and other infected liquids.

Reaction.—Some germicides are acid, others alkaline; the substances to be disinfected also vary in reaction. Thus lime is an alkali, and if used to disinfect an acid substance enough must first be added to neutralize the medium and then an additional amount of lime must be added necessary to accomplish the disinfection. In the same way, if mercuric chlorid is added to solutions containing sulphids, caustic alkalies, or certain metallic salts, sufficient must be added in order to first precipitate these substances and then enough more added to exert its disinfecting action. Likewise, the greater the number of germs to be destroyed the greater the amount of the disinfectant required to accomplish the purpose. Bacteria and their spores are much more sensitive to heat in either an acid or alkaline solution than in a neutral medium.

The Mechanism of Bactericidal Action.—Chemical substances and physical agents act in a great variety of ways to bring about the destruction of bacteria. Just how the microbes are poisoned is, in many instances, an unsolved problem in toxicology. It must be self-evident that disinfectants act in different ways, especially when we consider such widely diverse substances as acids, alkalies, metallic salts, phenols, ozone, bleaching powder, and chloroform.

Krönig and Paul,³ as early as 1897, established the important known facts in a classic contribution. They showed that the toxicity of the metallic salts depends upon:

1. The concentration of the metallic salt.
2. The specific property of the salt.
3. The type of solvent.
4. The degree of dissociation of the salt.
5. The effect of the cation.
6. The effect of the anion.
7. The effect of the undissociated salt.

With regard to the acids, the toxicity depends upon:

8. The electrolytic dissociation, that is, the concentration of hydrogen ions in solution. In a few instances, the anions have a specific toxic effect.

³ *Ztschr. f. Hyg.*, 1897, 25: 1.

The toxicity of the bases is in accordance with:

9. Their dissociation power, that is, with the concentration of hydrogen ions in solution.

The disinfecting power of the halogens:

10. Increase with their atomic weight.

The oxidizing chemicals are toxic:

11. In proportion to their oxidizing power.

With regard to negative and positive catalysis, they found that:

12. Anything that will increase the dissociation will increase the toxicity.

13. The alcohols are positive catalyzers when acting on the metallic salts, and negative catalyzers when in solution of phenol and formaldehyd.

14. Bouillon, gelatin and the body fluids are negative catalyzers in metallic salt solutions.

Krönig and Paul recognized the probability of the existence of certain general arbitrary laws between the concentration and toxicity of the metallic salts.

In 1908, Chick,⁴ using the figures obtained by Krönig and Paul in 1897, and those later obtained by Madsen and Nyman⁵ in 1907, and supplementing these results with a number of her own obtained with anthrax spores, was able to show a close similarity between the process of killing and the equation for a unimolecular reaction embodying Guldberg and Waage's law. Her experiments with *B. paratyphosus* showed a departure from this simple law which she explained as due to permanent differences in resistance to disinfectants among individual organisms.

In 1918, Brooks,⁶ using the rate of hemolysis of blood cells, concluded that disinfectants do not follow the unimolecular law. He believes that the fundamental reaction may be either a simple process or the expression of a complex series of changes, whose rate is at all times governed by that of the slowest of the series.

Barnett Cohn⁷ finds the mortality at constant temperature of bacteria in unbuffered media like distilled or tap water is variable and coincident with apparently insignificant P^H variations. Controlling the P^H by means of M/500 buffer solutions stabilizes this variability. He concludes from his interesting and instructive studies that the mortality of bacteria whether by strong disinfectants or by milder agents follows the laws of logarithmic decline. He has shown that the course of the disinfection process can be

⁴ *J. Hyg.*, 1908, 8: 92; 1910, 10: 238.

⁵ *Ztschr. f. Hyg.*, 1907, 57: 388.

⁶ *J. Gen. Physiol.*, 1918, 1: 61.

⁷ *J. Bact.*, 1922, 7: 183.

expressed by mathematical relations comparable to those used in dealing with monomolecular chemical reactions.

The conception that disinfection resembles a chemical reaction, the disinfectant representing one reagent and the bacterium the other, is of great importance since the *cardinal points of efficient disinfection*, namely, *adequate active mass*, or concentration of the germicide, *time of action*, and *perfect contact* are thereby experimentally established.

The Choice of Germicide.—The choice of the germicide depends somewhat on the nature of the substance to be disinfected, as well as upon the resistance of the virus. Ordinarily germicidal solutions, such as bichlorid of mercury, 1:1,000, or carbolic acid, 2½ per cent, cannot be trusted to kill tetanus spores; emulsions are not serviceable for the disinfection of feces; a weak chlorinated lime will disinfect water, but a strong solution is necessary to disinfect fabrics, but the strong solution bleaches and rots the fiber. Certain chemicals have a selective action and appear to be specific poisons for some organisms as, for example, copper sulphate for algæ. On the other hand, carbolic acid is particularly ineffective against the virus of smallpox. Taken altogether, therefore, the choice of the chemical, its strength, and time of application, the temperature of the solution, and its method of employment, are all problems which must be solved for each particular class of infection, and each particular group of substances.

THE STANDARDIZATION OF DISINFECTANTS

There is no accurate standard by which the power of disinfecting agents may be measured. There are conditions influencing the life of the bacterial cell which we are unable to control. It is for this reason that the strengths of solutions necessary to disinfect are variously stated by different authorities, and the time of exposure is for the same reason not always definitely decided. The difficulty in this connection is to determine the minimum conditions which will furnish trustworthy results and still provide a coefficient of safety necessary for general practice. Of still greater importance is the fact that our laboratory tests do not imitate the natural conditions under which bacteria are commonly found in nature. The requirements of practical disinfection are therefore usually much more severe than the conditions of our laboratory tests.

While the results of scientific work in the laboratory must be our guide as to the value and efficiency of any disinfecting process, we cannot ignore the results of experience gained in actual practice in combating the communicable diseases. This is especially true of disinfectants used against a disease the cause of which is only surmised or the mode of transmission not definitely known. We have had a lesson on this point in the case of sulphur dioxid, long used empirically, but discredited when it was shown to be a feeble germicide; and finally reestablished as an efficient insecticide.

On the other hand, laboratory experiments have established with great

accuracy the value and reliability of certain disinfectants which otherwise would have been overlooked. Thus the value of corrosive sublimate, chlorinated lime and formaldehyd was established, while on the other hand some substances, such as zinc chlorid and sulphate of iron, have been robbed of the high place in which they were formerly held, and placed near the bottom of the list of disinfectants. Even carbolic acid has been shown to have less germicidal power than was formerly supposed.

METHODS OF STANDARDIZING DISINFECTANTS :

Pringle⁸ as early as 1750 attempted to standardize the then known anti-septics by determining their power to preserve (*i. e.*, prevent decomposition in) a mixture consisting of 2 grains of meat and 60 grains of sea salt in 2 ounces of water. Following Pringle's work little was accomplished for over 100 years in standardizing disinfectants until Koch started a new era by the use of pure cultures and the "thread method."

The Thread Method.—Koch⁹ in 1881 used pure cultures of *B. prodigiosus*, *B. pyocyaneus*, and *B. anthracis*, both with and without spores. He soaked threads in a culture of the test organism and afterward dried them for various periods and then exposed these infected threads to the action of the disinfectant to be tested. The threads were then washed and laid on the surface of a solid nutrient medium and incubated for growth. This method, although characterized by greater scientific accuracy than the technic previously used, lacked perhaps those broader features of the older, rougher experiments. Koch's reports, so favorable to bichlorid of mercury, gave a great impetus to its use. Geppert,¹⁰ however, soon made it plain that Koch's high regard for bichlorid of mercury was partly due to an overestimate of its destroying power, inasmuch as the thread may carry over a sufficient amount of the chemical to inhibit growth. Geppert used ammonium sulphid to precipitate the mercury and thereby demonstrated a lower figure for its germicidal power.

Sternberg's Method.¹¹—As early as 1881 Sternberg described a method that is evidently the precursor of the "carbolic coefficient" and its various modifications. He mixed 5 c.c. of a young culture with equal quantities of a solution of the germicidal agent. Thus 5 c.c. of a 1 to 200 solution of carbolic acid was added to 5 c.c. of a recent culture of typhoid, and after stated intervals 1 or 2 loopfuls would be transferred to a nutrient medium.

The Garnet Method.—The Garnet method proposed by Krönig and Paul¹² in 1897 was an attempt at a more precise method. Small garnets of uniform size are coated with an emulsion containing sporulating anthrax bacilli. These are dried and then dropped into the disinfecting solution. After

⁸ *Phil. Tr.*, London, 46: 525.

⁹ *Berl. klin. Wchnschr.*, 1889, 26: 789; also *Deutsche med. Wchnschr.*, 1891, 17: 797.

¹⁰ "Mitteilungen aus dem kaiserlichen Gesundheitsamte," 1881, 1: 324, abstracted by Whitelegge, in "Recent Essays," New Sydenham Society, London, 1886, 115: 493.

¹¹ *Bull. of Nat. Bd. Health, U. S. A.*, 1879, 1, 219, 227, 237 and 265, and 1881, 3: 23. Sternberg's *Manual of Bacteriology*, New York, 1873, p. 186.

¹² *Ztschr. f. Hyg.*, 1897, 25: 1.

exposure for stated intervals the garnets are removed, rinsed, and the organisms washed off in sterile water, plated, and counted. Krönig and Paul emphasized the necessity of the disinfectant reaching each organism, the subsequent washing of the disinfectant from each organism, and the performance of the test with a constant number of organisms, since the time required for disinfection is dependent upon the number of microorganisms present. This method, along with the thread method of Koch, has been supplanted by the "carbolic coefficient" of Rideal and Walker and modifications thereof.

CARBOLIC COEFFICIENT

Rideal and Walker¹³ in 1903 introduced a method by which they proposed to determine and state in definite numerical terms the value of any disinfectant. This they called the "carbolic coefficient," for the reason that carbolic acid is taken as the unit of measurement against which the germicidal power of all other substances is compared. It was also spoken of as the "Rideal-Walker" method or the "drop" method, because one drop of the culture of *B. typhosus* was used for each c.c. of disinfectant tested.

Rideal and Walker opened a new era in the standardization of disinfectants. They prepared a number of standard conditions for the test, without which comparable results are not possible. The most important conditions are temperature, media, nature and age of the test microorganisms, time of exposure, degree of dilution, etc. The Lancet Commission¹⁴ in 1908 recommended several modifications of the Rideal-Walker technic and this method was further modified by Anderson and McClintic,¹⁵ who, in 1911, proposed certain changes in technic, and a different method of calculating the coefficient, which they termed the "phenol coefficient." Stimson¹⁶ described a machine by which the testing may be facilitated.

Kendall and Edwards¹⁷ in 1911 described an infected agar plug, designed to test the penetrating power of a disinfectant.

Physical-Chemical Methods.—It remains to cite the admirable work of Chick and Martin¹⁸ upon the laws of disinfection. They proposed in 1908 that the time element be established arbitrarily and with this called for two other constants, namely, the number of bacteria initially present, and the temperature. They believed that the killing of bacteria simulates a monomolecular reaction in which the bacteria take the place of one of the reacting substances. Phelps¹⁹ in 1911 made the interesting proposal to determine the germicidal value of a disinfectant at any temperature and concentration by a mathematical formula which would use the findings of a single experiment of comparatively easy technic.

¹³ *J. Roy. San. Inst.*, 1903, 24: 424; *J. Infect. Dis.*, 1912, 10: 254; *Am. J. Pub. Health*, 1913, 3: 575.

¹⁴ *Lancet*, 1909, 2: 1454, 1516, 1612.

¹⁵ *U. S. Hyg. Lab. Bull.* No. 82.

¹⁶ *U. S. Pub. Health Rep.*, 1918, 33:529.

¹⁷ *J. Infect. Dis.*, 1911, 8: 27.

¹⁸ *J. Hyg.*, 1911, 8: 132; 1908, 5: 644, 698.

¹⁹ *J. Infect. Dis.*, 1911, 8: 27.

The Carbolic Coefficient Method.—This test for standardizing disinfectants has been variously modified and improved.²⁰ As modified it is at present the best method we have for comparing the strengths of germicidal substances in solution or suspension. The method, however, has distinct limitations, as it gives information concerning the relative value of germicides only upon the naked germ cells under comparatively favorable conditions of action.

In order to obtain results that may have comparative value and to avoid discrepancies it is of the greatest importance to keep all the factors of the test uniform and to give attention to every detail. The following²¹ are the more important factors and principles upon which this test is based:

Time.—The time is taken as the constant and the strength of the disinfectant as the variant. It is easy to demonstrate that, if reversed, totally erroneous results will be obtained.

Test Organism.—The coefficient will vary with different microorganisms. The culture recommended is a twenty-four-hour-old *B. typhosus* grown in bouillon. It is important always to use the same strain of typhoid, as different races vary in resistance. Further, the culture should be carried over every twenty-four hours on at least three, preferably seven successive days before using it in a test. It is advisable to filter the culture through filter-paper in order to remove clumps just before beginning a test. The culture should always be grown under the same conditions, upon the same medium, so as to insure uniformity.

Medium.—The standard beef-extract broth (reaction + 1.0) recommended by the Committee on Standards of the American Public Health Association for Water Analysis, is used both to grow the test typhoid organism and also for the sub-cultures made after exposure to the disinfectant. Ten c.c. of this broth are placed in each test-tube for the sub-cultures, as this amount is sufficient to avoid any antiseptic activity of the disinfectant carried over.²²

Phenol, first proposed by Rideal and Walker, and now adopted by common consent, is the standard of comparison. Phenol may readily be obtained chemically pure, and exact solutions may be prepared by titration with bromin. A 5 per cent stock solution is usually made and this is diluted for the purpose of the test. Another advantage of phenol is that it is relatively unaffected by the presence of organic matter. Only formalin perhaps is superior to it in this regard.

Temperature of Exposure.—This is one of the most important factors. The germicidal activity of substances increases with the temperature. In this respect germicidal reactions resemble chemical reactions. It is there-

²⁰ S. Rideal and J. S. A. Walker. *J. Roy. San. Inst.*, London, 1903, 24: 424. "The Standardization of Disinfectants," The Lancet Commission, 177, Nos. 4498, 4499, 4500. Anderson and McClintic: *J. Infect. Dis.*, 1911, 8: 1. *U. S. Hyg. Lab. Bull.* No. 82; *U. S. Pub. Health Rep.*, 1918, 33: 529; 1919, 34: 2297.

²¹ The carbolic coefficient here described is not the Rideal-Walker Technic but modified in part from the Hygienic Laboratory Phenol Coefficient, and in part from the Lancet Commission Method.

²² See "Modification of Media," *U. S. Pub. Health Rep.*, 1919, 34: 2297.

fore of the utmost importance that the solutions tested should be always at the same temperature, and for this purpose 20° C. has been selected as most convenient. The solutions to be tested and the typhoid culture itself

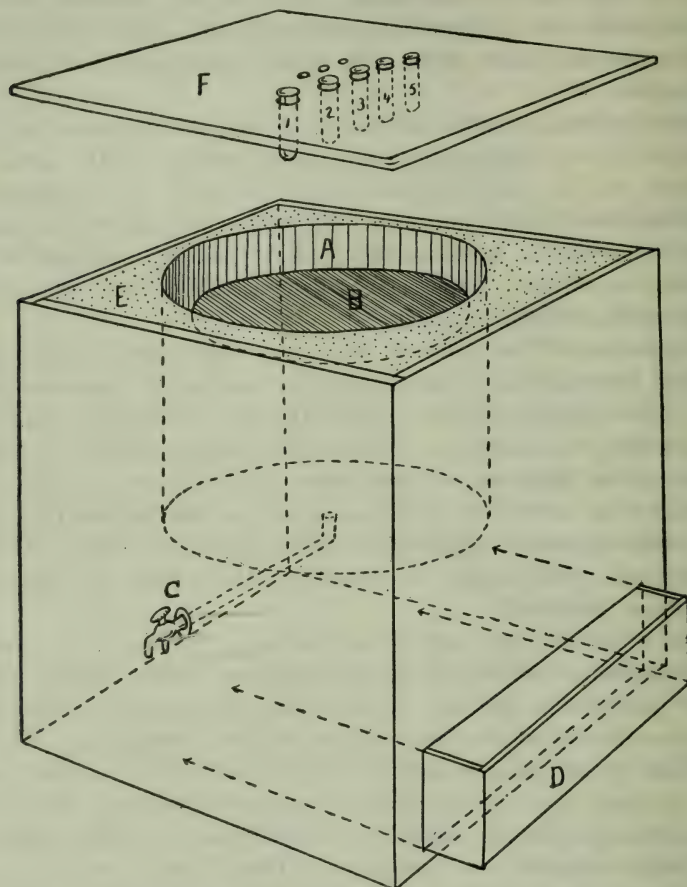


FIG. 147.—DEVICE FOR DETERMINING CARBOLIC COEFFICIENT.

Consists of a wooden box 14 inches long by 14 inches wide by 15 inches high, containing a metal pail (A) 10 inches in diameter and $8\frac{1}{4}$ inches deep. A shelf made of wire mesh (B) is inserted 2 inches from the top of the pail, which is filled with water. A pipe with a faucet (C) from the bottom of the pail will be found very convenient to draw off the water and regulate its temperature. Asbestos packing (E) completely surrounds the pail in order to insulate it. The lid of the box (F), which is raised in the drawing, contains openings for the five test-tubes, and three other openings for cultures and thermometer. When the lid is in place the test-tubes rest upon the shelf (B). A drawer (D) in the bottom of the box is convenient to keep test-tubes, inoculating needles, thermometers, and other parts of the apparatus.

must be brought to this temperature before they are mixed, and then maintained at this temperature in a water-bath.

Proportion of Culture to Disinfectant.—Rideal and Walker first proposed to use one drop of the typhoid culture to each cubic centimeter of germi-

cidal solution. It is more accurate to use a measured amount, say 0.1 c.c. of the twenty-four-hour-old bouillon culture of typhoid to 5 c.c. of solution. These are convenient amounts easily and accurately measured with standardized delivery pipets. It should be kept in mind that the addition of the bouillon culture dilutes the germicidal solutions, but as this is a constant factor it does not affect the comparative values as expressed by the carbolic coefficient, but may be taken into consideration in judging the germicidal values for practical work.

Inoculation Loops.—Precisely the same quantity of fluid from the mixture should be removed each time for the transplants. This is done most readily with platinum loops made of 23 U. S. standard gauge wire and a loop 4 millimeters in diameter inside measurement. This may be made over a No. 14 wire U. S. gauge. Several of these loops should be on hand. They are sterilized and placed upon a rack. As one is used it is flamed and returned to the rack, so that it will be cool when taken in its turn.

Dilutions.—A standard series of dilutions should be made of the phenol standard and also of the germicide to be tested—in accordance with the tables in Hygienic Laboratory Bulletin No. 82.

Technic.—The following method is the one used in my laboratory for carrying out the carbolic coefficient:

A solution of 5 per cent phenol c. p. is made and standardized chemically.²³ The usual dilution of 1 to 90, 1 to 100, and 1 to 110, etc., are made from this stock solution as desired.

The solutions of the germicidal substances to be tested must be made accurately, according to volumetric or gravimetric methods.

The tests are carried out in test-tubes one inch in diameter and three inches long. These are placed in a row in a water-bath. The test-tubes rest upon a bed of sand and are held in place by a wire rack or simply by a board perforated with holes of suitable size. If the water-bath is sufficiently large and the water brought to just 20° C. it may be maintained at this temperature with but slight attention.

Each test-tube receives 5 c.c. of the solution to be tested. Time is allowed for the solutions to reach the temperature of 20° C., then the culture which has previously been brought to 20° C. is added and mixed with the solution in each test-tube in turn. The culture is added to each tube at intervals of just 30 seconds. With a row of five tubes this will make a 2½-minute interval for each tube as the subsequent sub-inoculations are made.

Two and one-half minutes after the phenol and the culture have been mixed together in tube No. 1 a loopful of the mixture is removed and planted in broth; 30 seconds later a loopful of the mixture is taken from tube No. 2, and so on throughout the series at intervals of 30 seconds. The entire procedure of removing the loopful of mixture and planting it into one of the test-tubes containing 10 c.c. of broth requires only about 15 seconds, allowing

²³ By bromin titration. (See description in Sutton's *Volumetric Analysis*.)

plenty of time to flame the loop, replace it in the rack, and pick up another loop which had previously been flamed and has cooled sufficiently for the next operation. The test-tubes holding the mixture of germicidal solution and culture need not be removed from the water-bath, and it is not necessary to keep them plugged with cotton. The loop should always be plunged to the

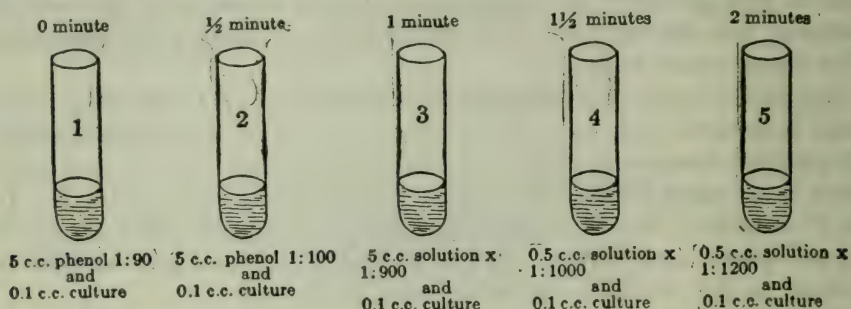


FIG. 148.—ARRANGEMENT OF TUBES IN WATER-BATH AND THEIR CONTENTS.

bottom and care taken not to touch the sides of the test-tube and always to carry away a loopful of the fluid to be transplanted. The test-tubes holding the medium for the transplants are conveniently placed in wooden racks and are incubated at 37° C. for forty-eight hours, when the readings as to growth (+) or no growth (—) are tabulated.

An example of a carbolic coefficient test follows:

Solution	2½ Minutes	5 Minutes	7½ Minutes	10 Minutes	12½ Minutes
Phenol 1: 90	—	—	—	—	—
Phenol 1: 100	+	—	—	—	—
Solution X 1: 900	—	—	—	—	—
Solution X 1: 1,000	+	—	—	—	—
Solution X 1: 1,200	+	+	+	+	+

The carbolic coefficient of solution X is therefore $\frac{900}{90} = 10$.

The carbolic coefficient is determined by comparing the strengths of the unknown disinfectant with phenol that “show life” in two and one-half minutes—or practically the strengths that kill in five minutes. Previously it was permissible to compute the coefficient upon any of the periods of the test up to thirty minutes. However, it was found possible in this way, either by chance or intent, to obtain an extravagantly high coefficient. Thus Chick and Martin showed that the phenol coefficient of certain metallic salts varies from 13.8 to 550, depending on whether the period of comparison is two and one-half or thirty minutes.

Anderson and McClintic²⁴ have modified the calculation employed by the Lancet Commission to determine the coefficient to be the mean between

²⁴ U. S. Hyg. Bull., No. 82.

the strength and the time coefficient; that is, the figure representing the degree of dilution of the weakest strength of the disinfectant that kills within two and one-half minutes is divided by the figure representing the degree of dilution of the weakest strength of the phenol control that kills within the same time. The same calculation is done for the weakest strength that kills in fifteen minutes. The mean of the two is the coefficient.

An example of the "phenol coefficient" as determined by the Hygienic Laboratory method follows:

PHENOL COEFFICIENT: HYGIENIC LABORATORY METHOD

Sample	Dilution	Time Culture Exposed to Action of Disinfectant for Minutes						Phenol Coefficient
		2½	5	7½	10	10	15	
Phenol	1:80	—	—	—	375 650
	1:90	+	—	—	—	— + —
	1:100	+	+	+	—	—	—	80 110
	1:110	+	+	+	+	+	—	—
								2
Disinfectant "A" ...	1:350	—	—	—	4.69 + 5.91
	1:375	—	—	—	—
	1:400	+	—	—	—	2
	1:425	+	+	—	—	—	—	—
	1:450	+	+	—	—	—	—	—
	1:500	+	+	—	—	—	—	—
	1:550	+	+	+	—	—	—	—
	1:600	+	+	+	+	—	—	—
	1:650	+	+	+	+	+	—	—
	1:700	+	+	+	+	+	+	—
	1:750	+	+	+	+	+	+	—
								= 5.30

Temperature of medication, 20° C.

Culture used, *B. typhosus*, 24-hour, extract broth filtered.

Proportion of culture and disinfectant, 0.1 c.c. + 5 c.c.

Kendall and Edwards²⁵ have devised an ingenious method to determine the penetrating power of germicides in the presence of organic matter. The method consists essentially of cylindrical molds of agar impregnated with the test organism. Sections of these cylindrical molds or "artificial feces" are exposed to the germicide solutions and plants made after proper intervals of time from a core taken from the center of the cylinder.

The carbolic coefficient of any substance should be based upon the average of a large number of tests.

Interpretation of Results.—A low coefficient means a weak germicide and a useless disinfectant. On the other hand, a high coefficient is not a true indication of a favorable agent in practical work. There are many factors still to be considered. Thus a useful disinfectant should not be very poisonous to higher animals; should not corrode metals or rot fabrics; should not stain or bleach; should not have an unpleasant smell; should be reasonably cheap; should be readily miscible with water and not deposit from

²⁵ *J. Infect. Dis.*, 1911, 8: 250.

THE PHENOL COEFFICIENT OF SOME COMMERCIAL GERMICIDES

(Determined by Thomas B. McClintic*)

Germicide	Without Organic Matter	With Organic Matter
Bacterol	1.58	1.34
Benetol	1.23	0.92
Cabot Sulphonaphthol	3.87	2.33
Carbolene	1.36	0.65
Carbолоzone	1.48	0.48
Car-Sul	2.00	1.75
Chloro-Naphtholeum	6.06	3.21
Cremoline	1.26	0.69
Creo-Carbolene	4.03	2.26
Creolin-Pearson	3.25	2.52
Cresoleum	2.90	1.75
Crude Carbolic Acid	2.75	2.63
Dusenberry's Liquid Creoleum	1.00	0.40
Germol	2.12	1.79
Hycol	12.30	9.37
Hygeno A	3.56	1.81
Kreosota	1.26	0.65
Kreotas	1.10	0.30
Kreso	3.92	2.32
Kresolig	2.18	1.48
Lincoln Disinfectant	1.48	1.10
Liquor cresolis compositus (U. S. P.)	3.00	1.87
Lysol	2.12	1.57
"Naphthalene Disinfectant"	2.50	1.36
Phenoco	15.00	9.86
Phenol liquid (U. S. P., 1890)	1.77	1.76
Phenosote	3.43	2.31
Phinotas	1.37	0.53
R. R. Rogers Disinfectant	3.03	2.05
Rudisch's Creolol	1.24	0.75
Saponified Cresol	1.03	0.57
Tarola	3.12	1.93
Trikresol	2.62	2.50
Zenoleum	2.25	1.64
Zodone	1.62	0.51
Zonol	2.37	1.57
Antozone †	nil	...
Creola Disinfectant	0.52	...
Dioxygen	weak	...
Electrozone	0.90	...
Formacone Liquid	weak	...
Killitol	weak	...
Kretol	0.92	...
Listerine	weak	...
Phenol Disinfectant and Cleansing Liquid	0.61	...
Phenol Sodique	weak	...
Pino-lyptol	0.27	...
Platt's Chlorides	weak	...
Public Health Liquid Disinfectant	0.48	...
Sanitas	0.30	...
The Twentieth Century Disinfectant	0.13	...
Veroform Germicide	0.43	...
Worrell's Insect Exterminator and Disinfectant	weak	...
Zodane No. 3.	weak	...

* U. S. Hyg. Lab. Bull. No. 82.

† The following disinfectants have a coefficient of less than 1. Most of them are so weak that it was impracticable to determine the coefficient.

solution or suspension; should be reasonably stable; should act both in alkaline and acid media; should not be greatly influenced by the presence of organic matter, and should possess a fair power of penetration. Further, it should not be specific, but be a general poison for microscopic life. It must at once be evident that no one test can determine all of these factors, so that a thorough and comprehensive study of the substance to be used should be made upon many different parasites under many different conditions before we can have a satisfactory knowledge of its power and limitations. This is one of the reasons that makes us conservative about taking up new germicidal substances until thoroughly tested under different conditions, *and inclines us to adhere to well-known chemicals such as bichlorid of mercury, carbolic acid, cresols and phenol, lime, the hypochlorites, chlorin, and formaldehyd, the advantages and limitations of which have been thoroughly established.*

CHAPTER II

PHYSICAL AGENTS OF DISINFECTION

Sunlight.—Sunlight is an active germicide. It destroys spores as well as bacteria. Unfortunately, the sunlight is so uncertain and the force of the sun's rays so variable and their disinfecting powers so superficial that it cannot be depended upon as an aggressive measure in attacking infection. In rooms, ships, and confined spaces sunshine comes more under the purview of the sanitarian than under that of the disinfecter, but the latter can always use it to advantage in supplementing his other methods. Rooms and objects should always be sunned and aired after disinfection.

The different rays of light have very different effects upon germ life. The blue-violet and ultraviolets, that is, the more refrangible chemical rays of short wave length, are the only ones possessing germicidal power. The red and yellow rays are practically inert in this regard. The source of light seems to have little influence upon the result; it is more a question of intensity and nature of the rays. Even diffused light retards growth and development of bacteria, and if strong enough will finally kill them. Electric light containing the proper rays is efficient. The Roentgen rays have no bactericidal properties. The sun is the natural generator of ultraviolet rays.

The time required for sunlight to destroy bacteria varies with its brightness and with conditions such as moisture, temperature, transparency, and composition of the media, which aid or hinder the effect of the rays. The time also varies with the different microorganisms; plague bacilli and cholera vibrio usually die more quickly than tubercle bacilli. Spores are much more resistant to the influence of the chemical rays than the bacterial cells themselves. Thus it usually requires about thirty hours' sunning to kill an anthrax spore, while the anthrax bacillus is killed in one or two hours when subjected to the same conditions.

Ultraviolet Rays.—Ultraviolet rays are invisible rays which lie beyond the violet end of the spectrum. In general it may be said that they include those rays of short wave lengths between 2,900 and 2,250 angstrom.¹ (See Plate I.)

The wave lengths of the visible spectrum are from about 7,610 angstrom units (red) to about 3,970 (violet). According to Nogier² the ordinary ultraviolet rays extend from 3,920 to 3,000 units, the average rays from 3,000 to 2,200, and the extreme ultraviolet rays from 2,200 to 1,000 units. He states that rays from 3,920 to 3,000 are not bactericidal to any extent

¹ One angstrom unit is $1/10,000,000$ of a millimeter.

² *Rev. d'hyg.*, 1910, 32: 421.

but produce sunburn after long exposure. Rays between 3,000 to 2,200 have a strong action on bacteria while those from 2,200 to 1,000 are still more powerful but are of little value since they are easily absorbed by air and other substances.

Roentgen rays are not absorbed by bacteria and therefore have no germicidal action, probably because they are the shortest wave length known.

Ultraviolet rays from the Cooper-Hewitt lamp, produced by an electric discharge through mercury vapor contained in a quartz lamp in vacuo, have an exceedingly powerful germicidal action, killing spores as well as bacterial cells. Ordinary glass is opaque to these rays of short wave lengths and it is therefore necessary to use quartz globes.

Cernovodeanu and Henri³ have shown that the action of ultraviolet light is greater near the lamp and decreases as the square of the distance from the source of the rays. Ultraviolet rays act independently of temperature between 0° C. and 55° C. They also act equally quickly in the presence or absence of oxygen. Pure cultures of non-spore-bearing microorganisms are killed in from five to sixty seconds. Molds, however, are only partially destroyed.

Ultraviolet light kills bacteria by some direct action upon the protein which it changes physically and chemically, and not, as was formerly supposed, indirectly, through the formation of ozone, peroxids, heat, etc. Whether the rays of short wave length are germicidal depends upon whether they are absorbed. Roentgen rays, for example, the shortest known, are not absorbed and are not germicidal. The bactericidal action of light is confined to the ultraviolet region of the spectrum, beginning at 350 millimicrons and extending with increased intensity to 185.6 millimicrons. These limits coincide with the absorption of ultraviolet light by bacteria.⁴

On the whole, very satisfactory results have been obtained in disinfecting clear water with ultraviolet rays, and in several cities in France and elsewhere the municipal water supply is treated by this process. It is also used to purify swimming pools. See pages 1037 and 1072. Proteins and other bodies of high molecular weight interfere with the action of the rays. Turbidity and color have a similar action.

Ultraviolet rays cannot be used successfully to pasteurize milk for the reason that milk is opaque, and furthermore the rays act upon the protein, causing unpleasant odors and tastes to develop. Attempts to kill the bacteria in turbid water, beer, wine, or vinegar have been only partially successful, because the organic matter interferes with the penetration of the rays.

Ultraviolet light possesses no therapeutic value so far as direct destruction of bacteria within any of the tissues of the body is concerned. Verhoeff⁵ has shown that ultraviolet light will not destroy bacteria within the cornea, even when the latter is perfectly transparent, without at the same time severely injuring the corneal tissues. See also page 459.

³ *Compt. rend. Soc. de Biol.*, 1910, 150: 52; also *Ztschr. f. Hyg.*, Feb., 1916.

⁴ Bayne-Jones, *Johns Hopkins Hosp. Bull.*, 1923, 34: 11.

⁵ *J. Am. M. Ass.*, 1912, 62: 762.

Electricity.—It appears that electric currents have little germicidal action in themselves and that the apparent effects noted by some investigators are due either to the heat generated by the current or to electrolytic action. Electricity has very little use in practice as a disinfectant. Hermite used the products of electrolysis for the sterilization of sewage. He added sea-water to the sewage and the electrolytic action caused the formation of hypochlorite, which has well-known germicidal action. The effect of electrical currents upon bacteria seems to be a purely chemical one in the case of germicidal substances being formed by electrolytic decomposition; or a thermal one in the case of the production of heat, which so frequently attends the discharge of electric currents.

Pressure.—Hite⁶ finds that a pressure of 100,000 pounds per square inch, at room temperature, destroys most non-spore-bearing bacteria. Under these conditions, milk containing from 30 to 40 million per c.c. may be reduced to a few hundred by the application of 100,000 pounds for ten minutes. The pressure does not affect the enzymes. Forty-five thousand pounds pressure is sufficient to kill *B. typhosus* in beef broth in ten minutes. *B. diphtheriæ* in beef broth are killed at 40,000 pounds pressure in ten minutes.

Larson, Hartzell and Diehl⁷ found that a direct pressure of 6,000 atmospheres kills non-spore-forming bacteria in 14 hours. A pressure of about 12,000 atmospheres for the same length of time is required to kill spores. Non-spore-bearing bacteria are killed by carbon dioxide of 50 atmospheres pressure in about 1½ hours. Yeast cells withstand the action of carbon dioxide for more than 24 hours, probably because of their ability to transmit the carbon dioxide molecule through the membrane promptly. Nitrogen under a pressure of 120 atmospheres has no effect on bacteria.

Burning.—Fire is the great purifier. Burning has, however, a very limited range of usefulness in practical disinfection. The disinfecter is seldom justified in burning an article against the wish of its owner, for we now possess methods by which any object may be rendered safe so far as its power of conveying disease is concerned. In actual practice, however, the disinfecter often comes across a great amount of rubbish and articles of little value that he will find easier and cheaper to burn than to disinfect. The burning of garbage and refuse is the safest means of disposing of such organic substances from a sanitary standpoint, especially in districts where pestilential disease prevails. From the same standpoint the cremation of all bodies dead of a communicable disease is the safest method of preventing possible spread of infection from this source. Burning is the most satisfactory method of disinfecting and disposing of small amounts of sputum and other infected discharges. Burning of the surface of the ground by means of gasoline torches and petroleum is sometimes used to destroy animal parasites and other infections which find lodgment on the soil. The gasoline torch is also used to fight insect pests of trees and plants.

⁶ Bull. 146, W. Va. Agricul. Exp. Sta., October, 1914.

⁷ J. Infect. Dis., 1918, 22: 271.

Dry Heat.—A temperature of 160° C. continued for one hour will destroy all forms of life, even the most resistant spores. It is easy to maintain this temperature in an apparatus of special construction known as a hot-air or dry-wall sterilizer. Dry heat penetrates slowly, especially through fabrics. Air is also a poor conductor. It must therefore be remembered that the temperature as registered on the thermometer does not necessarily indicate the temperature at the critical place within the sterilizer. A liberal factor of safety is therefore necessary, especially in large-scale operations. In the sterilization of glassware and other objects in laboratories making biologic products, it is customary to expose them for at least two hours to not less than 170° C. to insure penetration and heating of all surfaces. Glassware and many other objects will stand this degree of heat and are sterilized in an oven of this kind in bacteriological laboratories and in surgical clinics.

Dry heat is not as satisfactory a disinfectant as moist heat, as it lacks the power of penetration and is injurious to fabrics. Most materials will bear a temperature of 110° C. without much injury, but when this temperature is exceeded signs of damage soon begin to show.

Scorching occurs sooner with woolen materials, such as flannels and blankets, than with cotton and linen. Over-drying renders most fabrics very brittle, but this injury may be lessened by allowing the materials which have been subjected to dry heat to remain in the air long enough to regain their natural degree of moisture and pliability before manipulating them.

The ordinary household cooking oven is as good as any specially contrived apparatus for the disinfection of small objects by dry heat. In the absence of a thermometer it is usual to heat the oven to a point necessary to brown cotton and expose the objects no less than one hour.

Boiling.—Boiling is such a commonplace, every-day procedure that it is often neglected in practical disinfection despite the fact that it is one of the readiest and most effective methods of destroying infections of all kinds. An exposure to boiling water at 100° C. continued for an hour will destroy the living principles of practically all the infectious diseases with which we have to deal in public health work. To be sure, there are a few spores that have shown a remarkable resistance to boiling water and streaming steam in laboratory experiments. Boiling, therefore, cannot be entirely depended upon where tetanus, anthrax, or other resisting spores are in question. As a matter of fact, a degree of moist heat much lower than the boiling point of water is effective against the great majority of the known viruses. Thus a temperature of 60° C. for 20 minutes will destroy the microorganisms of cholera, typhoid, dysentery, diphtheria, plague, tuberculosis, pneumonia, erysipelas, and practically all non-spore-bearing bacteria. Boiling kills them at once.

Boiling is especially applicable to the disinfection of bedding, body linen, towels, and fabrics of many kinds; also kitchen and tableware, cuspidors, urinals, and a great variety of objects. Surfaces, such as floors, walls, beds, metal works, etc., may be effectively disinfected by mechanically cleansing

them with boiling water. The efficacy of boiling water, especially when used in such circumstances, is greatly increased by the addition of corrosive sublimate, carbolic acid, or one of the alkaline coal-tar creosotes. The addition of lye, borax, or a strong alkaline soap also increases the penetrating and detergent power of boiling water when applied to surfaces soiled with organic or oily matters.

Boiling in 3 to 5 per cent phenol or some similar disinfectant for thirty minutes will kill tetanus and other resisting spores. This method is applicable to rubber goods and other articles.

In using boiling water for the disinfection of bright steel objects or cutting instruments the addition of 1 per cent of an alkaline substance such as carbonate of soda will prevent rusting and injury to the cutting edge. The method advocated by Rebula⁸ is as follows: Two and one-half grams of sodium hydroxid (NaOH) should be added to 1,000 grams of water. The sodium hydroxid should be allowed two minutes in which to dissolve and to combine with the carbon dioxid of the water before the instruments are put in the solution and boiled.

Steam.—Steam is one of the most satisfactory disinfecting agents we possess. It is reliable, quick, and may be depended upon to penetrate deeply if used with proper technic. Further, it does more than disinfect; it sterilizes. Vegetating bacteria are killed instantly and most spores in a reasonable time. It may therefore be used to destroy the infection of any of the communicable diseases.

Either streaming steam or steam under pressure is used in practical disinfection.

Streaming steam has the same disinfecting power as boiling water, and an exposure of half an hour to an hour is sufficient. Steam under pressure is a more powerful germicide than streaming steam. At a pressure of 15 pounds⁹ to the square inch steam has a temperature of approximately 120° C. and may be depended upon to sterilize in twenty minutes. At twenty pounds' pressure it has a temperature of approximately 125° C. and will sterilize in fifteen minutes, provided there is direct exposure. In practical work a factor of safety is usual.

The federal regulations¹⁰ require 121° C. (15 pounds) for thirty minutes for steam sterilization of glassware and rubber tubing. Glassware and rubber tubing must be moistened immediately before steam sterilization and each flask or hollow apparatus should contain one-eighth of its volume of water when put in the autoclave. This is for the purpose of insuring that steam will be in contact with all surfaces.

The following table gives the temperature at various pressures and vice versa.

⁸ *Centralbl. f. Chir.*, 1920, 47: 1297.

⁹ That is, 15 pounds above atmospheric pressure. Barometric pressure is zero. Throughout this book "pounds pressure" means "gauge pressure."

¹⁰ Regulations U. S. Public Health Service, October 1, 1919.

TEMPERATURE		PRESSURE		
Degrees C.	Degrees F.	Millimeters of Mercury	Pounds per Square Inch	Gauge Pressure *
100	212	760.00	14.70	0.0
105	221	906.41	17.53	2.83
110	230	1075.37	20.80	6.10
115	239	1269.41	24.55	9.85
120	248	1491.28	28.85	14.15
125	257	1743.88	33.72	19.02

* That is, pounds per square inch above atmospheric pressure.

The *temperature* is a much more reliable guide than the *pressure*. The temperature of the *object* to be disinfected, rather than the temperature of the *apparatus*, is the critical factor. Many failures are due to neglect of these simple points in the physics of disinfection.

Penetration may be greatly facilitated by a partial vacuum before introducing the steam.

Steam is applicable to the disinfection of bedding, clothing, fabrics of all kinds, and a great variety of other objects, provided certain precautions are taken to prevent shrinking, staining, running of colors, etc. Steam shrinks woolens and injures silk fabrics; it ruins leather, fur, skins of all kinds, rubber shoes, oilcloth, and articles made of impure rubber or containing glue, varnish, or wood.

Food of all kinds is sterilized by steam in the process of canning.

It is important in disinfecting with steam, whether with streaming steam or steam under pressure, to *expel the air from the apparatus*. The air, being a poor conductor of heat, forms dead spaces and prevents the steam coming in direct contact with the articles to be disinfected, thereby defeating the object to be attained. As steam is lighter than air the latter can best be expelled from the apparatus by admitting the steam from above, in which case the descending column of steam forces the air out at the bottom. If the steam is admitted at the bottom it swirls up, making a nearly uniform mixture with the air, and while the temperature quickly rises in the apparatus the air escapes mixed with the steam, so that it takes a long time and an unnecessary waste of steam to drive out the contained air.

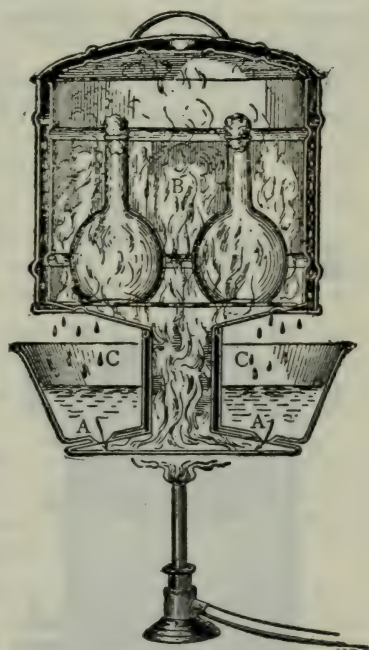


FIG. 149.—SECTION THROUGH ARNOLD STEAM STERILIZER.

Disinfection with streaming steam may be accomplished in many ways without the use of special apparatus. For rough and ready work on the railroad the objects to be disinfected may be hung in a freight-car and the steam brought from the locomotive. On board a vessel one of the compartments above the water-line may be filled with steam from the boiler. Objects may be steamed in any rough structure, such as a freight car or an outhouse, wherever a boiler is found to furnish the steam. Such a structure need not be tight, for the streaming steam escaping from the cracks produces a circulation and favors penetration.

In the laboratory small objects are disinfected in streaming steam in the Arnold steam sterilizer or the Koch steamer.

On account of the great certainty with which steam under pressure acts it is the favorite method in practical disinfection, especially where sterilization is required, and devices for applying this process on a large scale have reached a high degree of perfection. The smaller forms of steam sterilizers under pressure are known as digestors or autoclaves and the larger ones as steam disinfecting chambers.

The Autoclave.—The autoclave, digestor, or steam sterilizer consists of a closed kettle usually made of copper or iron and sufficiently strong to withstand the pressure. Water is placed in the kettle and the heat is applied to the bottom, usually by means of several Bunsen gas jets. The apparatus is surrounded as high as the shoulder, where the lid is attached, with a metal jacket which serves the purpose of bringing the heat of the flame in contact with the entire surface of the kettle. The lid is made to fit tightly by means of screw bolts and a rubber gasket. A thermometer, pressure gauge, safety valve and a small opening with a stopcock for the purpose of allowing the escape of the air are provided. If all the air is not expelled from the apparatus the dead spaces will have a much lower temperature than that registered on the thermometer. For instance, the steam itself may register a temperature of 130° C., while test fluids exposed may only reach 70° to 80° C.

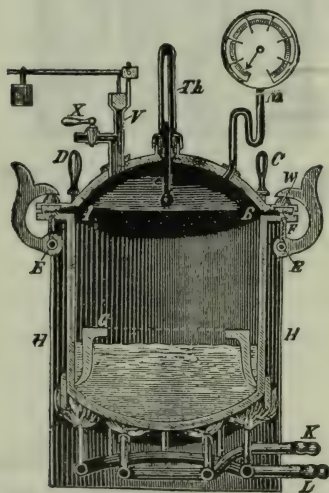


FIG. 150.—SECTION THROUGH AUTOCLAVE.

Therefore, in using this form of sterilizer it is customary to allow the steam to escape in full force for several minutes before permitting the pressure to rise.

In the sterilization of liquids, for which this apparatus is frequently used, it is important, at the conclusion of the process, not to take off the lid or open the valves, or in any other way release the pressure until the apparatus

has cooled; otherwise the condensed steam causes a diminished pressure, in which the heated liquids will boil energetically, resulting in a bubbling over, a blowing out of stoppers, or a bursting of the flasks. It is therefore necessary to wait until the pressure is zero, as registered on the gauge; or, better, until the condensing steam produces a partial vacuum and the air is automatically sucked in through the vacuum valve, which is sometimes fitted in the lid of the apparatus for this very purpose.

The Steam Chamber.—The steam disinfecting chamber has reached a high degree of usefulness through the gradual perfection of the details of its working parts. These chambers are somewhat complicated and their mechanical construction must be mastered in order to insure reliable results. Steam disinfecting chambers may be used with streaming steam or with steam under pressure; with formaldehyd gas alone, or with this gas in combination with dry heat; and, with cyanogen chlorid and other gases, with or without a vacuum. They should always be operated under skilled supervision, and with automatic registration and controls.

The disinfecting chamber itself may be rectangular or cylindrical in shape, the former giving more effective space, the latter being a stronger and cheaper method of construction. The chamber is built of an inner and outer shell forming a steam jacket, as shown in Figures 149 and 150. The steam jacket serves several purposes. By heating the contents of the disinfecting cylinder before the steam is turned in it avoids condensation. During the process of disinfection it helps keep the steam in the chamber "live," thereby preventing the wetting of the objects exposed. After the disinfection is finished and the chamber opened the heat from the steam in the jacket may be used to dry the contents which have just been steamed. Therefore, in using this apparatus for disinfecting with steam, either with or without pressure, the steam is kept circulating in the jacket from the beginning to the end of the process.

In the jacket the steam has a perfectly free circulation, so that the entire disinfecting cylinder, with the exception of the doors, is surrounded by live steam. The outer shell of the jacket is insulated with a covering of sectional magnesia, asbestos, or some other non-conducting substance.

The steam from the boiler passes through the main steam pipe A (Fig. 151) to the pressure-reducing valve (2), and thence to the bottom of the jacket at B, B.

Into the disinfecting chamber itself the steam can be admitted only from the jacket, through the circulating pipes, A. C. B (Fig. 152), and after circulating through the disinfecting chamber in the direction as shown by the arrows is allowed to pass out with the drip through the drain D (Fig. 152). Upon the completion of the process the steam may be blown off through the vacuum pipe F, but this outlet should not be used during the steaming because the desired circulation would not be obtained.

It will be noticed that the steam is admitted at the *bottom* of the *jacket*, and at the *top* of the disinfecting *chamber*, as shown in Figure 151. The

object of admitting the steam at the top of the disinfecting chamber is to favor the expulsion of the air through its outlet at the bottom by means of the descending column of steam. Therefore, in order to expel all the air and fill the chamber with steam it is essential to open the drain D (Fig. 152) while the steam is entering through B, B, and this outlet D should not be closed until steam escapes freely. In using the vacuum attachment to expel the air contained in the apparatus the *modus operandi* is somewhat different.

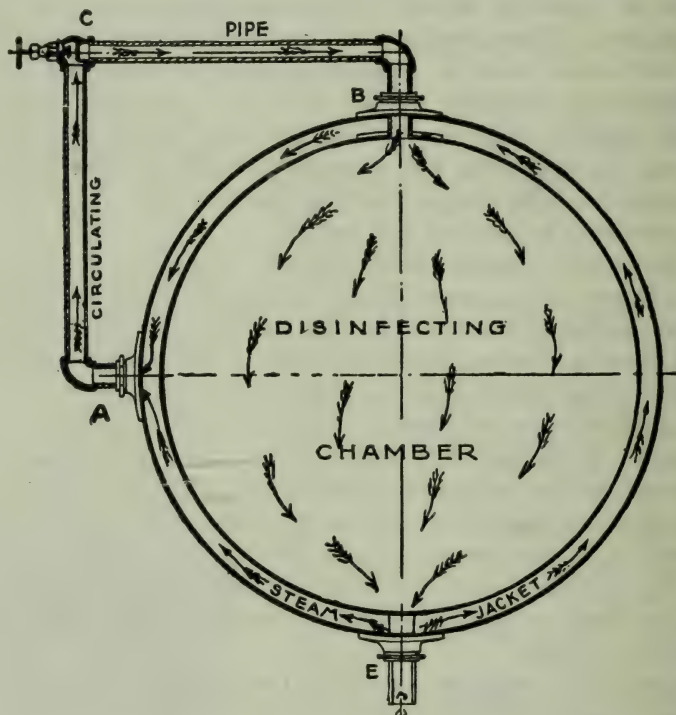


FIG. 151.—CROSS SECTION THROUGH STEAM DISINFECTING CHAMBER.

To determine whether all the air has been expelled, lead a hose from the exhaust to a bucket of water. Air reveals itself as bubbles; steam gives the well-known hammer effect.

A partial vacuum may be obtained in steam chambers of this type with the ejector (4, Fig. 153). The object of the vacuum is to facilitate the penetration of the steam, which rushes into all the interstices of fabrics and inaccessible places, to take the place of the air which has been withdrawn. The ejector works upon the familiar principle of the water vacuum pump, the air being drawn or sucked along with the current. With a pressure of 80 pounds in the boiler and the valve J (Fig. 153) wide open, the ejector will produce a partial vacuum of 15 inches in one of the largest-sized cham-

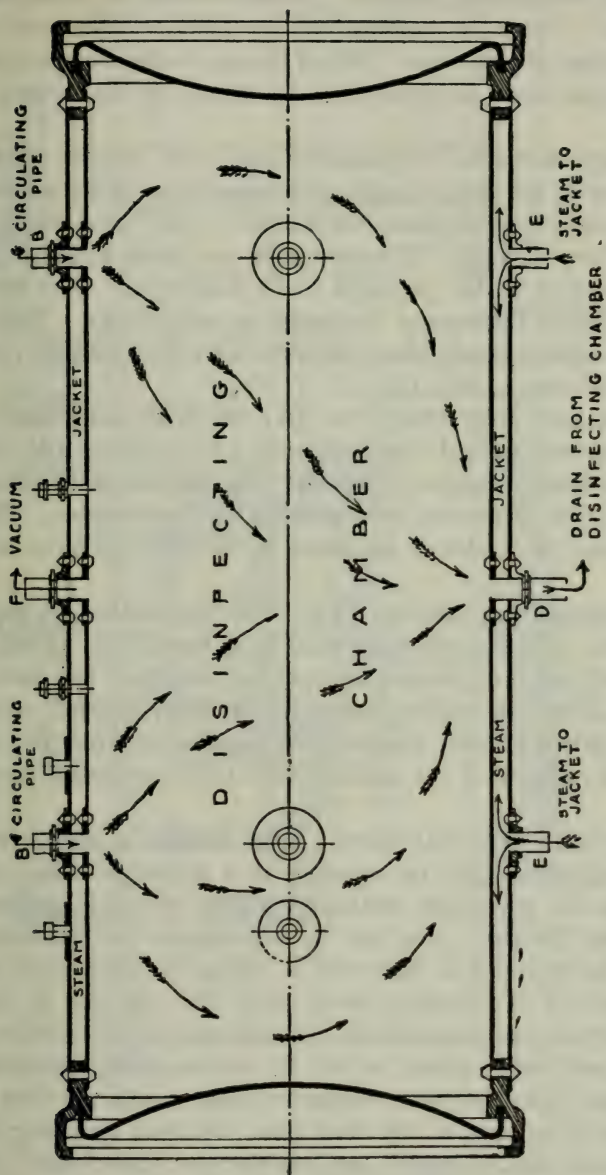


FIG. 152.—LONGITUDINAL SECTION THROUGH STEAM DISINFECTING CHAMBER.

bers in one minute, which is very much quicker than can be accomplished with the ordinary forms of piston pumps.

Any steam disinfecting chamber may have attached to it an apparatus for generating formaldehyd, chlorin, cyanogen chlorid or other gas, so that objects that are injured by exposure to steam may be disinfected and disinfested with the gas, plus dry heat. Before the gas is admitted into the chamber a partial vacuum must be established by means of the ejector, to insure penetration.

The ordinary mercurial thermometers that stick out are often incorrect and furthermore do not always register the temperature of the interior. Steam disinfectors should have duplicate thermometers, and these should be standardized from time to time. Thermocouples are most accurate and can be placed in the center of the packages to be disinfected. The thermometers made by the Taylor Instrument Company are satisfactory. They should be self-recording, upon rotating sheets, so as to have a permanent record of the temperature and time of each run.

Steam chambers must always be provided with galvanized or copper hoods to prevent rust-stained drip from soiling the clothing and other objects exposed to the steam; gauges to indicate both vacuum and steam pressure, and a safety valve to prevent over-pressure in the chamber. The amount of pressure from the boiler is regulated by a reducing valve in the main steam pipe.

In the accompanying diagram (Fig. 154) the method of installing the steam chambers in the disinfecting shed of a quarantine station is shown. It will be noted that the cylinders are open on both ends, and that a dividing wall running across the building separates the receiving end, where the infected objects arrive and are prepared for disinfection, from the discharging end, where the contents of the chamber are aired, dried, and repacked after disinfection.

This separation is essential where a large amount of disinfection is done for a variety of diseases, as, for example, in a municipal disinfecting establishment or at the quarantine station of a busy port. It is true that the virus of certain diseases is not apt to contaminate the surroundings, and in such cases there would be little risk in taking the disinfected articles out of the same end of the chamber from which they are put in, especially if the exposed surfaces are mopped with a disinfectant in the interim. But this is a risk that need not be taken; in fact, all well-regulated disinfecting plants maintain a rigid separation between the two sides, never allowing both doors of the chamber to be open at the same time, and even providing two sets of workmen, one for the "infected" and one for the "disinfected" side.

The chambers must be loaded with care in order to obtain reliable results and to avoid injuring the articles exposed to the process. The packages must not be too large or crowded too closely, for, although the vacuum facilitates the penetration of the steam, there is a limit in this regard; it takes so much longer for disinfecting agents to penetrate dense packages and bundles

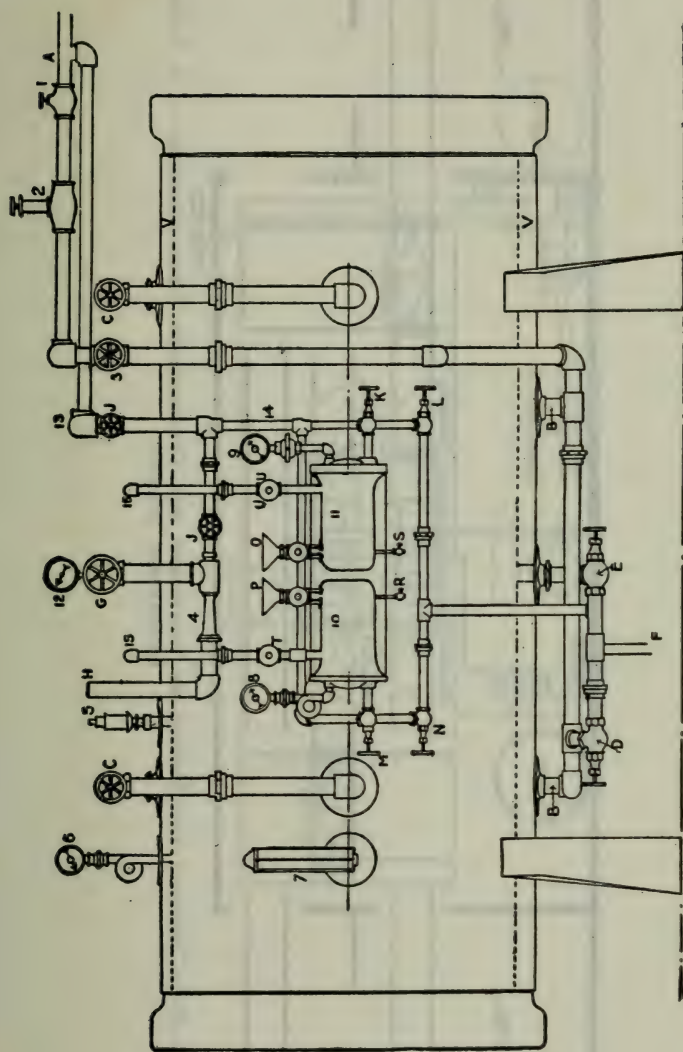


FIG. 153.—KINYOUN-FRANCIS STEAM DISINFECTING CHAMBER.

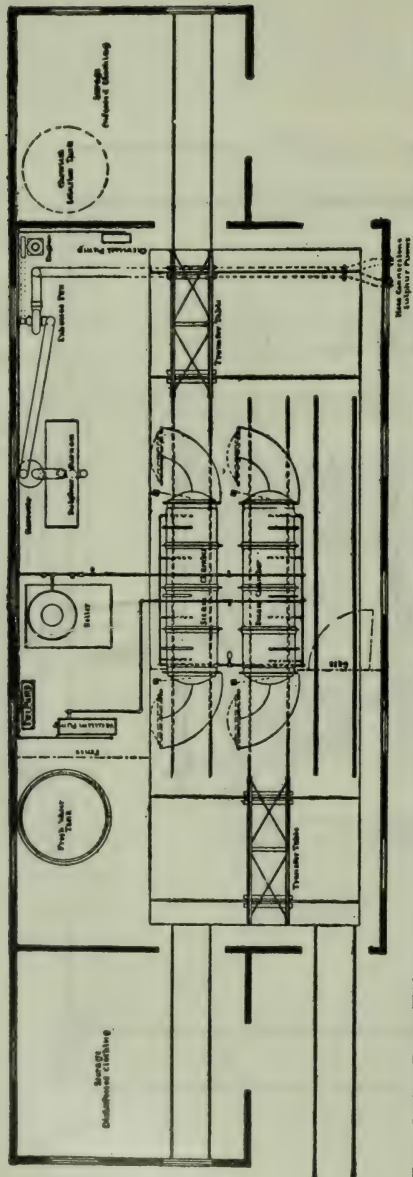


Fig. 154.—Plan showing the method of installing the double-ended steam chambers at a national quarantine station.

that there is little saving of time and a distinct loss in trustworthiness. Steam cannot be expected to penetrate mattresses, compressed bundles of rags, bales of cotton, feathers, hair, or other packages of merchandise which are often presented for disinfection, except by aid of the vacuum and prolonged exposure.

CHAPTER III

CHEMICAL AGENTS OF DISINFECTION

LIQUID DISINFECTANTS

These consist of substances in either solution or suspension. An enormous number of such disinfectants have been exploited, but to be of practical value they must not only be strongly germicidal, but must also meet the many exacting requirements of general practice. Such substances are few in number.

Almost any chemical substance under one condition or another has the power to retard the development or destroy the activity of microbial life. We need only mention the well-known power of common salt or of sugar, both of which in sufficient concentration prevent fermentation and putrefaction. In weaker dilutions these same substances, on the contrary, favor growth of almost all the known bacteria.

The undeserved reputation of many chemical substances depends more upon their vile odor or judicious advertising than upon actual efficiency. Only those substances that have proved their worth by scientific tests and shown themselves to be trustworthy in actual practice will be discussed.

Methods of Using Chemical Solutions.—There are various ways of applying chemical solutions for disinfecting purposes. No method is trustworthy that does not thoroughly wet the object with the solution, so that there may be direct contact between the germ and the germicide.

As a rule this may best be accomplished by immersing the infected object. When this is not practicable the solution must be applied to the object. A favorite way of applying disinfecting solutions to surfaces, such as walls, ceilings, the holds of ships and other rough structures, is by means of a hose. The pressure is supplied either by elevating the tank containing the solution or by means of a pressure pump. As bichlorid of mercury and chlorinated lime are practically the only disinfectants used in this way, the containers must be made of material that is not corroded by these strong chemicals.

In applying the disinfecting solution to the surfaces of a room or the hold of a ship the operator should begin at one corner of the ceiling, wetting that first, and then go over every portion of the walls systematically, from above downward. The floor comes last.

Solutions thus applied remain but a short time in contact with the surfaces to be disinfected. It is therefore an advantage to have the solution

hot and strong and to have sufficient pressure, in order to obtain the mechanical cleansing effect produced by a vigorous stream.

Germicidal solutions are usually applied by hand methods with mops, brushes, etc. The spray atomizer is unreliable.

The above methods with reference to solutions apply also to emulsions. Germicidal solutions and emulsions are much more potent when used hot.

Metallic Salts.—The metallic salts which are used are mainly those of silver, mercury, bismuth, zinc and copper. The germicidal activity of many of these metallic salts when acting upon bacteria suspended in pure water is extraordinarily high, but this powerful action is reduced in the presence of organic matter.

There is good evidence pointing to the belief that the metallic ions in aqueous solutions, owing to electrolytic dissociation, are the chief disinfecting agents.¹

Bichlorid of Mercury.²— HgCl_2 , bichlorid of mercury or mercuric chlorid, commonly called corrosive sublimate, is one of our most valuable and potent germicides. It destroys all forms of microbial life in relatively weak solutions. It kills both germs and their spores. It is not a deodorant.

The disadvantages of bichlorid of mercury are that it corrodes metals, forms insoluble and inert compounds with albuminous matter, and is very poisonous. These disadvantages place distinct limitations upon its use.

Bichlorid of mercury will dissolve in 16 parts of cold water and 3 parts of boiling water. As it is not readily soluble in water, it is convenient to keep a saturated alcoholic solution on hand and use this to make the watery solution. A 25 per cent solution may readily be made in alcohol, and by the addition of hydrochloric acid or ammonium chlorid this solution keeps well without precipitation.

The solution of bichlorid of mercury is rendered more stable by the presence of hydrochloric acid or a chlorid such as ammonium chlorid or common salt. Twice the weight of these substances should be added to the quantity of bichlorid used. If the solution is to be pumped or otherwise come in contact with metals it is better to use the salt than the acid, because the acid solution of bichlorid is very destructive to the metal parts of the pump and to the couplings and nozzle of the hose, particularly if made of copper or brass. Sea-water contains about 4 per cent of salt, and is well suited for making bichlorid solutions. It is extensively used at seaport quarantine stations for this purpose.

The germicidal action of bichlorid solution seems to depend upon the reaction which takes place between the mercury and the protein of the germ. Geppert has shown that in the reaction which takes place between the bichlorid of mercury and the spores of anthrax the vitality of the latter may seem to be lost, but that the bichlorid may be precipitated from its combination by

¹*Arch. f. exper. Path. u. Pharmacol.*, 1893, 32: 456.

²The use of bichlorid of mercury as a disinfectant dates from Koch's experiments in 1881. *Kaisl. Gesundh.*, Vol. I.

the action of ammonium sulphid, thus restoring the viability of the spore. The sulphur acts as an antidote.

Bichlorid of mercury is decomposed by lead, tin, copper, and other metals, and therefore should not be made or kept in metal receptacles. Lead pipes are rendered brittle and worthless. Care must therefore be exercised in using this solution about water-closets and house plumbing.

Corrosive sublimate is precipitated in alkaline fluids containing albuminous substances. The precipitate consists of insoluble and inert compounds; therefore corrosive sublimate should not be used for the disinfection of media containing much organic matter, particularly when the reaction is alkaline. It is not well suited to the disinfection of sputum feces, for it forms an albuminate which retards penetration. It also unites chemically with sulphids and the caustic alkalies, so that it should not be employed as a disinfectant when these substances are present in any considerable amount.

To diminish the danger from accidents in households and hospitals bichlorid solutions should be colored with permanganate of potash or indigo or one of the anilin dyes.

Bichlorid of mercury is usually used in the proportion of 1:1,000, which is ample for the destruction of all the non-spore-bearing bacteria, provided the exposure is continued not less than half an hour and direct contact is obtained.³ To assure this contact a longer time should be allowed in practice. Many bacterial cells are killed almost at once when brought into direct contact with a solution of this strength, and the great majority perish within fifteen minutes. The extra time allows for penetration and provides a factor of safety. Warm solutions are much more potent than cold. For spores a solution of 1:500 is necessary and an exposure of not less than one hour.

For practical work the solution may be made as follows:

Corrosive sublimate	1 dram	1 gram
Water	1 gallon	1 liter
Mix and dissolve		

This is approximately a 1 to 1,000 solution. One ounce of this solution contains very nearly a half a grain of corrosive sublimate.

Silver Salts.—The germicidal action of most silver salts closely resembles that of the corresponding mercuric compounds. Silver chlorid is insoluble and hence ineffective, but silver nitrate appears to resemble mercuric chlorid fairly closely. Silver cyanids, colloidal silver, and various organic compounds which yield free silver ions on solution in water, all appear to have inferior germicidal properties, although some of them find useful application in medical practice.

Silver nitrate is used in public health work chiefly for the prevention of

³ The action of mercuric chlorid upon spores has been most carefully studied by Kronig and Paul, *Ztschr. f. Hyg.*, 1897, 25: 1; by Madson and Nyman, *Ztschr. f. Hyg.*, 1907, 57: 388; and by Chick, *J. Hyg.*, 1908, 8: 92.

gonorrheal ophthalmia. Credé's method consists in the use of a 1 per cent solution as a prophylactic against ophthalmia neonatorum. Argyrol, which is a colloidal silver, is used in 25 per cent solution, and protargol, a silver protein, is used in 3 per cent solution.

Silver nitrate coagulates albuminous matter more actively, perhaps, than any of the metallic salts. It therefore has feeble powers of penetration. It is useless as a germicide to cauterize wounds produced by dog bites for the prevention of rabies.

Sulphate of Copper.—Sulphate of copper (CuSO_4) is about half as strong as bichlorid of mercury. It has a peculiar selective action in that it has a remarkable affinity for many species of algæ which are killed in the proportion of 1 : 1,000,000. Algæ are the most common cause of unpleasant odors and tastes in drinking water, and sulphate of copper may therefore be used to check or destroy their growth. See page 1036. In these great dilutions sulphate of copper will not kill the typhoid bacillus, so that it is not practical to use it as a disinfectant in water.

Ferrous Sulphate.—Ferrous sulphate has long been valued as a disinfectant on account of its cheapness and property as a deodorant, and has been used extensively. Its germicidal power has been shown by laboratory tests to be rather feeble, so that it cannot be depended upon as a trustworthy disinfectant.

Ferrous sulphate (FeSO_4), commonly called green vitriol, iron vitriol, or copperas, consists of large bluish-green crystals which slowly effervesce and oxidize in the air. It is soluble in about twice its weight of cold water, forming a greenish solution. It is a much less powerful germicide than the sulphate of copper, and is limited in use to the destruction of odors, and even for this purpose is not always successful. It is still used with lime for the clarification of turbid waters.

Zinc Salts.—Zinc salts have long been known to have antiseptic properties, and the chlorid especially was used by Lister and other. Its germicidal action is far inferior to that of most mercury and silver salts. Chlorid of zinc (ZnCl_2) was at one time highly valued as a disinfectant and is still extensively used despite the fact that it stands rather low in the list of germicidal agents. It has even weaker power as a disinfectant than ferrous sulphate. Five per cent solutions are effective against anthrax spores, but a 2 per cent solution is effective against most vegetative forms in a reasonably short time when acting in an aqueous medium. This activity is, however, much influenced by the medium in which it acts, since it is very rapidly precipitated by proteins, phosphates, etc., and therefore cannot be recommended as trustworthy. It has some power as a deodorant.

COAL TAR AND ITS PRODUCTS

Coal-Tar Creosote.—Creosote is a highly complex refractile liquid obtained from the destructive distillation of wood or coal. Wood-tar creosote

for medicinal use is obtained from beechwood; it is a complex mixture of phenoloid bodies, the proportions of which differ according to the modes of distillation and purification. It contains phenols, cresols, and higher homologues. Coal-tar creosote, sometimes called creosote oil, contains that portion of the distillate from coal-tar intermediate between crude naphtha on the one hand and pitch on the other. Coal-tar creosote contains phenols, cresols, and higher phenoloid bodies, also naphthalene and other solid hydrocarbons, as well as pyridin and other bodies of basic character. Creosotes vary in composition, and owe their germicidal properties to the phenol and cresols which they contain. They are seldom used as such, but form bases of many commercial disinfectants after purification or the addition of alkalies or soaps. It is the creosote from coal-tar, and not wood-tar, that is used as a germicide in public health work.

Hale⁴ has shown that the coal-tar disinfectants of the phenoloid group are considerably less toxic than either phenol or the cresols, but they are not harmless, non-poisonous substances as sometimes indicated by the commercial labels. On an average the disinfectants of the phenoloid group have a toxicity equal to about 15 or 20 per cent of that of phenol.

There appears to be an intimate relation between the germicidal powers of phenol and its derivatives and their protein precipitating capacity. Many of the various halogen derivatives of phenol are highly germicidal against bacteria suspended in water, but are not particularly active in the presence of blood-serum or other protein matter.

Carbolic Acid.—Carbolic acid is a very useful disinfecting substance with a wide range of application. It should not be depended upon to kill spores. As it does not coagulate albuminous matter as actively as corrosive sublimate it may be used for the disinfection of soiled clothing and bedding, as well as for excreta and sputum.

Carbolic acid is a popular term for an ill-defined mixture consisting largely of phenol and phenolic bodies. Crude carbolic acid was discovered by Runge (1834) in coal-tar. It is a nearly colorless, or reddish to reddish-brown liquid, turning darker on exposure to air and light. It has a strong creosote-like odor, with benumbing, blanching, and caustic effects upon the skin and mucous membrane. Crude carbolic acid is a mixture of phenols and cresols, with coloring matter and impurities. Carbolic acid is often used as a synonym for phenol; in fact, the British Pharmacopeia recognizes the name "*Acidum carbolicum*," but uses "phenol" as the English name for this substance. The term "carbolic acid" should be restricted to the crude mixture of phenols and cresols; whereas phenol is a definite chemical substance C_6H_5OH . (See Phenol.)

Crude carbolic acid is soluble in 15 parts of water at 15° C., making about a 6 per cent solution. The undissolved portion should not exceed 10 per cent of the volume of the carbolic acid. Carbolic acid is a very useful

⁴ *U. S. Hyg. Lab. Bull.*, 1913, No. 88.

disinfecting substance with a wide range of applicability. The cresols which it contains have a higher germicidal value than pure phenol itself. Commercial carbolic acid also contains hydrocarbons, and other impurities of tar oil which are totally lacking in bactericidal properties.

Crude carbolic acid dissolves in water with some difficulty and should therefore be thoroughly mixed. It is used in solutions of 2.5 to 5 per cent, in which strength it may be used for the destruction of non-spore-bearing bacteria. A 5 per cent solution is not dependable against spores. Warm or hot solutions are much more effective than cold. It should be remembered that crude carbolic acid (coefficient 2.75) has a higher germicidal potency than pure phenol (coefficient 1.0). Crude carbolic acid is commonly used for rough disinfecting purposes, such as floors, stables, barns, outhouses, animal pens, etc.

Phenol.—Phenol, C_6H_5OH , has the chemical structure of an alcohol, and is the chief constituent of carbolic acid. Pure phenol crystallizes in long colorless needles; commercial phenol forms a crystalline mass which turns reddish in time, and in contact with moist air deliquesces to a brown liquid. It has a penetrating odor and strong burning taste, and is a corrosive poison. It is soluble in 10.6 parts of water at $25^{\circ} C.$, very soluble in ether, alcohol, chloroform, benzin, carbon disulphid, glycerin, fixed and volatile oils.

Phenol when dissolved in alcohol or ether loses in germicidal value; the addition of 0.5 per cent of hydrochloric acid aids its activity.

McClintock and Ferry⁵ have shown that the large majority of the coal-tar disinfectants (carbolic acid, cresols, and the like) do not destroy the virulence of vaccine virus in $1\frac{1}{2}$ per cent solutions at five hours' exposure, while with this strength and length of time these disinfectants would destroy practically all non-spore-bearing bacteria. The inference, therefore, is allowable that this class of disinfectants is not safe to use for such diseases as smallpox, or others due to filtrable viruses.

The fact that carbolic acid and phenol do not actively coagulate albuminous matter renders them suitable to the disinfection of excreta and organic matters generally. They are not destructive to fabrics, colors, metals, or wood in the strengths used, and therefore may be employed for the disinfection of a great variety of objects. Crude carbolic acid, although it has a stronger germicidal power than pure phenol, has the disadvantage of having a more pungent and penetrating odor and leaves a deposit of coal-tar oils and other impurities.

There has been much disparagement of phenol because laboratory tests have clearly demonstrated that it is not a general germicide, and furthermore cannot always be depended upon to kill spores.⁶ This limits but does not destroy its usefulness, especially as the great majority of the epidemic diseases of man are due to non-spore-bearing bacteria.

⁵ *Am. J. Pub. Health*, 1911, 50: 418.

⁶ Anthrax spores are remarkably resistant to phenol solutions, and may be viable after four days' immersion in a 5 per cent solution.

The time of exposure to a 3 or 5 per cent solution should be not less than one-half hour. Fabrics are usually immersed for one hour.

Spores are killed with certainty by boiling for thirty minutes in 3 to 5 per cent phenol or some similar disinfectant.

The introduction of a halogen atom into the benzol ring greatly increases the germicidal power of the phenols, cresols and naphthols, while at the same time the toxicity of these substances is diminished. Thus Bechhold and Ehrlich⁷ have shown that tri-brom- β -naphthol and tri-chlor- β -naphthol are very powerful germicides; practically odorless, and not very poisonous.

The Cresols.—By far the majority of the disinfectants sold to the public are mixtures of varying quantities of phenolic bodies, especially the cresols, with inert tar oils and an emulsifying agent such as soap or tar, and sometimes rosin, gelatin, or dextrin. These substances possess a smell distinctive of carbolic acid and are effective germicides. The cresols, $C_6H_4(CH_3)OH$, have the advantage over carbolic acid or pure phenol in that they readily form beautiful emulsions, have a higher germicidal value, and are less poisonous. It has already been pointed out that while emulsions may be more potent germicides than solutions, on the other hand, they lack the power of penetration.

Cresol is prepared from coal tar by collecting the distillates coming over between $140^\circ C.$ and $220^\circ C.$, and then purifying these distillates by treatment with solution of sodium hydroxid and hydrochloric acid. Cresol is a mixture of the three isomeric cresols obtained from coal tar and freed from phenol, hydrocarbons, and water. It is also known as cresylic acid.

Cresol⁸ consists of a mixture of ortho-, meta-, and para-cresols. Meta-cresol is a liquid; the other two are solid crystalline bodies having a low melting point. These cresols are found in commercial carbolic acid. The cresol group forms the next higher homologue to phenol, one atom of hydrogen being replaced in the latter by the methyl radical, CH_3 . The cresols are very insoluble in water. Their solution may be facilitated by the use of alcohol or glycerin. Cresol is a clear or pink-colored syrupy liquid. It is soluble to the extent of about $2\frac{1}{2}$ per cent in water. It is somewhat less poisonous than carbolic acid; its uses are the same. It is an effective germicide in a 1 per cent solution, which is as active as 3 per cent phenol.

Liquor Cresolis Compositus.—Liquor cresolis compositus of the United States Pharmacopeia is a substitution compound for "Lysol" and consists of cresol, 500 gm.; linseed oil, 350 gm.; potassium hydroxid, 80 gm.; and water sufficient to make 1,000 gm. This official mixture makes a clear solution in water. The solution is intended as a substitute for the many commercial preparations of cresol on the market. It has practically the same uses as lysol.

⁷ *Ztschr. f. Hyg. u. Infektionskrankh.*, 1909, Bd. 64, p. 113; Hoppe-Seyler's, *Ztschr. f. physiol. Chem.*, 1906, Bd. 47, Hefte 2 und 3, p. 173.

⁸ *Trikresol* is a trade name. It is the same substance as "Cresol" of the United States Pharmacopeia.

Lysol.—Lysol is a brown, oily-looking, clear liquid with a creosote-like odor. It is made by dissolving a fraction of tar oil which boils between 190° and 200° C. in fat, and subsequently saponifying by the addition of alkali in the presence of alcohol. It contains 50 per cent cresols, especially meta- and para-cresols, and 50 per cent of a strong concentrated potassium soap made with linseed oil. The soap contains 68 per cent of fatty acids. Lysol is miscible with water, forming a clean, saponaceous, frothy liquid. It is more powerful as a germicide than phenol, and is usually used in 1 per cent solution. It has a carbolic coefficient of 2.12 without organic matter and 1.87 with organic matter.

Pyxol is a preparation of cresol and soft soap analogous to "Lysol."

Creolin.—Creolin is a preparation similar to lysol. It is a proprietary preparation patented by Pearson and consists of an emulsion of cresols and certain other products contained in tar oil, with rosin soap. Many other similar preparations are on the market, such as cresolin, cyllin, disinfectol, sanatol, ixal, creosapol, sylphonathol, etc. The tar oil is brought into solution either with rosin soap or by means of concentrated sulphuric acid. Creolin forms a milky emulsion when mixed with water. It is used in 1 or 2 per cent solution. The phenol coefficient is 3.25 without organic matter and 2.52 with organic matter.

Aseptol.—Aseptol is a 33 $\frac{1}{3}$ per cent watery solution of orthophenol-sulphonic acid, $C_6H_4(SO_3H)OH$. It is made by mixing equal parts of phenol and concentrated sulphuric acid in the cold; if warmed, parasulphonic acid is formed, which is a much feeble germicide than orthosulphonic acid. The acidity of the orthophenolsulphonic acid is neutralized with barium carbonate.

Aseptol is a colorless liquid which gradually turns yellowish when exposed to the light, with a weak odor of phenol, and a feeble acid reaction. It is miscible, in all proportions, with water, alcohol and glycerin. Orthophenol-sulphonic acid gradually changes to paraphenolsulphuric acid in watery solution. Aseptol is much used in Germany for the disinfection of barns, out-houses, stables, and woodwork, soil, and the purification of rough substances generally. It is usually used in 5 per cent solution. In this strength it will kill anthrax spores in twenty-four hours.

Asaprol.—Asaprol is the calcium salt of β -naphthol sulphonic acid. It is made by warming 10 parts of β -naphthol with 8 parts of concentrated sulphuric acid in a water-bath until a clear solution is obtained. It is then diluted with water, neutralized with calcium carbonate, filtered, and the filtrate dried to a reddish powder. This powder is soluble in 1 $\frac{1}{2}$ parts of water and 3 parts of alcohol. It turns blue upon the addition of ferric chlorid.

Sanatol.—Sanatol is a dark fluid, readily miscible with water, forming a slight turbidity. It is made from 20 parts of tar oil, containing phenols, and 10 parts of 90 per cent sulphuric acid, and diluted with water sufficient to make 100 parts.

Solveol and Solutol.—Solveol is a solution of sodium cresolate in excess of cresol. Solutol is a solution of cresol in excess of sodium cresolate.

There are a vast number of other commercial disinfectants of similar nature consisting of coal-tar cresotes in combination with alkalies, soaps, resins, etc., such as chloronaphtholeum, sylphonathol, bacillol, saprol, para-cresol, and other trade names.

Naphthols.—Naphthols are found in coal tar though in small amount. They have a high germicidal value about the equal of phenols. Naphthol, $C_{10}H_7OH$, is a hydroxyl derivative of naphthalene; α and β modifications are known. The latter is of especial interest as a germicide. Naphthol itself is insoluble in water, but may be rendered soluble as a sodium salt, or may be emulsified with soaps or resin. Naphthol is used more as a medicinal germicide than in public health work. Tri-chlor- β -naphthol is much more germicidal, but less toxic than phenol.⁹

Ambrine.—Ambrine is a mixture used in the treatment of burns. It is a proprietary preparation, and similar to a product made according to the following formula: β -naphthol, 0.25 per cent; eucalyptus oil, 2 per cent; olive oil, 5 per cent; hard paraffin, 25 per cent; and soft paraffin, 67.75 per cent.

Naphthalene.—Naphthalene, $C_{10}H_8$, is a hydrocarbon obtained from coal tar and purified by crystallization. It is a white, shining crystalline substance, having a strong characteristic odor resembling coal tar, and a burning aromatic taste. It slowly volatilizes on exposure to air. Naphthalene is insoluble in water, but soluble in alcohol. It burns with a smoky flame.

Naphthalene has antiseptic properties but is much less active than either the cresols or the phenols. It is poisonous to most fungi and probably to most insects. Under the name "tar camphor" it has largely supplanted true camphor as a means of preventing the deposition by moths of eggs in woolen clothing. Cold storage for furs and objects of value is effective. Woolen goods may be protected by simply wrapping them in paper, which prevents the moth laying her eggs, for it is the larvæ that do the damage.

OXIDIZING AGENTS

Potassium Permanganate.—Potassium permanganate is a germicide of undoubted value, but of very limited application in general practice on account of the readiness with which it is reduced and rendered inert by organic matter. Despite its limitations it ranks high on the list of germicides for certain definite purposes, more particularly in surgical practice. It has been much used in India and other places for the purification of water.

All the permanganates are strong oxidizing agents, but as soon as they are reduced to manganese salts their disinfecting action ceases, so that their maximum germicidal effects are transitory.

Potassium permanganate ($KMnO_4$) is a dark purple, crystalline substance with a sweet, astringent taste. A few crystals impart to a large quantity of

⁹ See *Phenol und seine Derivate als Desinfektionsmittel*, by Kurt Laubenheimer, Urban and Schwarzenberg, Berlin, 1909.

water a rich purple tint which is destroyed by organic matter and deoxidizing agents. It is soluble in 16 parts of cold and 2 parts of boiling water. The stain produced by potassium permanganate may be removed by a solution of oxalic acid, muriatic acid, or simple lemon juice.

Potassium permanganate readily gives up its available oxygen, and it is the free nascent oxygen that is the true disinfecting agent. Sternberg found a solution of 1 to 833 sufficient to kill pus cocci in two hours. Koch found that a 5 per cent solution killed spores in one day. Löffler found that the bacillus of glanders is destroyed in two minutes by a 1 per cent solution.

Water containing organic matter may be purified to a certain extent and rendered palatable by adding, drop by drop, a solution of permanganate until the pink color of the water ceases to be destroyed after the lapse of 24 hours. The clear liquid may then be decanted and used. Permanganate used in this way does not reach sufficient concentration to be a trustworthy germicide.

Hydrogen Peroxid and Other Peroxids.—Hydrogen peroxid is a rather feeble germicide, but has certain other qualities which render it useful in surgical practice. Blood, pus and muscle juice contain an enzyme "catalase" which rapidly brings about the decomposition of hydrogen peroxid with the liberation of gaseous oxygen. This action rapidly decomposes all the peroxid and its disinfecting action comes to a speedy end. The mechanical effect of the disengaged gas is often a valuable property, and is made use of in loosening sticky secretions, washing away pus, or loosening adherent dressings.

Other very active oxidizing agents used as germicidal agents are ozone (page 791) and hypochlorites (page 1027).

LIME

Lime is one of the best and cheapest disinfecting substances we have. It is usually used either as lime or chlorinated lime.

Lime, or quicklime, is a very caustic substance used for the destruction of organic matter as well as germ life. On account of its efficiency and cheapness it is a valuable addition to the list of practical disinfectants. Lime or calcium oxid (CaO) is one of the alkaline earths. It is not so caustic as the alkalies, having less affinity for water. It is obtained by calcining native calcium carbonate (CaCO_3), such as chalk, limestone, or marble, by which the carbon dioxid is driven off and the calcium oxid remains behind. Lime as such requires the addition of water for germicidal purposes.

Almost all laboratory experiments, while differing somewhat in certain unimportant particulars, confirm the conclusions of the early investigators as to the great practical value of lime as a germicide. A 1 per cent watery solution of the hydroxid kills non-spore-bearing bacteria within a few hours. A 3 per cent solution kills typhoid bacilli in one hour. A 20 per cent solution added to equal parts of feces or other filth and mixed with them will disinfect them within one hour.

Lime has been used since very early times in connection with the disposal

of the dead. The method is an admirable one for the burial and disinfection of bodies dead from a communicable disease. The body should be placed in a tight coffin with twice its weight of fresh, unslaked lime, without the addition of water or moisture in any form.

Slaked Lime.—Slaked lime or calcium hydroxid, $\text{Ca}(\text{OH})_2$, is prepared by adding one pint of water to two pounds of lime. The lime absorbs about half its weight of water. The mass becomes heated and the air escapes from the pores of the lime with a hissing noise. The result is calcium hydroxid or slaked lime. Upon exposure to the air the slaked lime will absorb still more water and also carbon dioxid, converting it into calcium carbonate, which is inert so far as its disinfecting power is concerned. Freshly slaked lime should therefore always be used.

Whitewash is slaked lime mixed with water. It is commonly used for the disinfection, sweetening, and brightening of the walls of cellars, rooms, barracks, barns, stables, poultry-houses, and outbuildings generally. Whitewash is a very satisfactory method of destroying spore-free bacteria that may have lodged upon such surfaces. It improves illumination and is an incentive to keep things clean. A mordant such as glue is usually added to whitewash to make it adhere; also a little bluing.

Milk of lime is slaked lime mixed with four to eight times its volume of water to the consistency of a thick cream. It is useful for the disinfection of excreta and privy vaults. Air-slaked lime containing the inert carbonate must not be used in the preparation of whitewash or milk of lime, freshly slaked lime containing calcium hydroxid being necessary to accomplish disinfection. Calcium hydroxid is mostly insoluble and settles to the bottom; the milk of lime must therefore be agitated to restore its homogeneous character before it is used. Milk of lime is most powerful when freshly prepared. It soon changes to the inert carbonate, and therefore should not be used if more than a few days old unless carefully protected from contact with the air.

Lime is particularly valuable in the disinfection of excreta. The lime in one form or another must be well incorporated with the mass and enough must always be added in order to make the reaction of the mixture distinctly alkaline. Sternberg recommends that freshly prepared milk of lime should contain about one part by weight of hydroxid of lime to eight parts of water. This should be used freshly prepared and added in quantity equal in amount to the material to be disinfected. The mixture should be allowed to stand at least two hours before final disposal. Fortunately, this valuable disinfecting agent is very cheap, so that it can be used with a liberal hand in excess of the amount which scientific tests find necessary. (See page 1383.)

Chlorinated Lime.—Chlorinated lime was used as a disinfectant and deodorant long before bacteriology was a science. The early work of Sternberg demonstrated that the confidence placed in this substance from an empiric standpoint is justified by scientific tests. Chlorinated lime under certain circumstances, in fact, is one of the most powerful germicides we possess, and

has been used particularly for the disinfection of water and sewage. (See page 1025.)

Chlorinated lime, popularly miscalled chlorid of lime, is a soft, white, friable substance, and is known also as bleaching powder. It has a peculiar chemical composition and is somewhat unstable. It is made by passing chlorin gas through lime. Owing to its affinity for moisture, which it slowly absorbs from the air, it soon becomes pasty and loses some of its chlorin; the hypochlorites are reduced to chlorids, which are inert as germicides. Freshly prepared chlorinated lime should have a very slight odor of free chlorin. A strong odor of this gas indicates that deterioration of the substance is taking place. It should therefore be kept in air-tight receptacles.

Chlorinated lime is made by passing nascent chlorin gas over very slightly moist calcium hydroxid. Concerning its exact chemical composition there is some disagreement. It is represented by the formula CaOCl_2 or ClCaOCl or $\text{Ca}(\text{ClO})\text{Cl}$. The dry powder contains calcium oxychlorid (CaOCl_2), which is at once converted into calcium hypochlorite ($\text{Ca}(\text{OCl})_2$) and calcium chlorid (CaCl_2) in watery solution; thus: $2\text{CaOCl}_2 = \text{Ca}(\text{OCl})_2 + \text{CaCl}_2$.

According to the U. S. Pharmacopeia it should contain not less than 35 per cent of available chlorin. The British standard is 33 per cent and the German 25 per cent. Chlorinated soda has almost the same germicidal value as chlorinated lime. Chlorinated soda is sold in solution, and is prepared by mixing a solution of chlorinated lime and sodium carbonate.

Chlorinated lime is only partially soluble in water or in alcohol. A solution in water of 0.5 to 1 per cent will kill most bacteria in from one to five minutes. A 5 per cent solution usually destroys spores within an hour.

Chlorinated lime not only bleaches but is destructive to fabrics. If the solution is employed for the disinfection of body linen and washable clothing these articles should be promptly and thoroughly washed in plenty of fresh water.

It should be remembered that the hypochlorites are decomposed and practically rendered inert by organic matter. They should therefore be used largely in excess. Thus a preparation containing 10 per cent of available chlorin has the high carbolic coefficient of 21.0, but on mixing an equal amount of this preparation with urine and allowing the mixture to stand one hour the coefficient falls to 0.8 per cent (Klein¹⁰). Gruber points out that the efficiency of chlorinated lime, when used to disinfect cattle wagons, is greatly increased by first thoroughly washing away the organic matter.

Chlorinated lime may be used either as a dry powder or in solution. As a dry powder it is very generally used by strewing it into damp corners of cellars, privies, and similar places, where it acts as a deodorant and desiccant and retards the growth of mold. The dry substance may also be used to disinfect excreta. For this purpose enough of the chlorinated lime must be added and well incorporated with the mass and sufficient water added to make

¹⁰ *Pub. Health*, Oct., 1906. Confirmed by Rideal, Sommerville, Moore, and others

a 4 or 5 per cent solution. Much more chlorinated lime is necessary to disinfect feces in a bedpan than for sewage.

In the United States Army a 4 per cent strength of chlorinated lime in solution is officially prescribed for use in the disinfection of the excreta of the sick, it being specifically stated that the chlorinated lime so used shall be of good quality and not have undergone decomposition. A solution known as the "American standard," containing 6 ounces of the powder to the gallon, is largely used for the disinfection of discharges and for the scrubbing of floors and other surfaces.

In recent years chlorinated lime or chlorinated soda has come into special prominence on account of its use for the disinfection of drinking water. A surprisingly minute amount will disinfect a large volume of water. The amount required depends upon the quantity of organic matter contained in the water. A clean water may be rendered safe by the addition of 0.1 of a part of chlorinated lime (estimated as available chlorin) to 1,000,000 parts of water. For waters containing organic matter as much as 1 to 5 parts per 1,000,000 may be required. Chlorinated lime is now being displaced by chlorin gas for the purification of water. (See page 1031.)

Chlorinated lime may also be used to advantage to disinfect the bath water in cases of typhoid fever, dysentery, cholera, or other communicable diseases. It may also be used for the disinfection of springs, wells, cisterns, tanks, and many other purposes.

Emergency Use for Travelers and Campers.—For campers and travelers a convenient method for using chlorinated lime to disinfect drinking water is to add 1 gram of chlorinated lime containing approximately 30 per cent of available chlorin to 1 liter of water. This should be mixed thoroughly and enough of the mixture added to the water in question to make 1 part of chlorinated lime to 200,000 parts of water, and then allowed to stand at least twenty minutes after having been thoroughly shaken. The water may then be regarded as safe, so far as typhoid, cholera, and similar infections are concerned. A solution may be prepared by adding half a teaspoonful of chlorinated lime to a pint of water. Use a teaspoonful of this to 10 gallons; 36 drops to 1 gallon; or 9 drops to 1 quart. Let stand at least fifteen minutes.

Javelle Water.—Javelle water consists of chlorinated potash, 65 grams; chlorinated lime, 90 grams; water sufficient to make 1,000 grams. It is used as a medicinal wash. The active principle is potassium hypochlorite.

Labarraque's Solution.—Labarraque's solution, or *Liquor Sodæ Chlorinatæ*, is an aqueous solution of several chlorin compounds, chiefly sodium hypochlorite (NaClO) and sodium chlorid (NaCl), and should contain at least 2.6 per cent by weight of available chlorin as determined by titration with thiosulphate. The active principle is potassium hypochlorite. The solution is clear and colorless when pure. If prepared with an excess of chlorin it is yellowish in color. It has a feeble odor of chlorin and bleaches indigo, litmus, and vegetable dyes. In practice this solution diluted with water 1 to 4 is mainly used for the disinfection of the person, and in surgery,

but as it is more expensive and somewhat less efficient than chlorinated lime it has no advantages over that substance.

Dakin's Solution.—Dakin's solution came to notice during the World War in the Carrell method of treating wounds. It may be looked upon as an improved Labarraque's solution. The active ingredient of Dakin's solution is sodium hypochlorite. It must be freshly prepared; must have an entire absence of caustic alkali; the concentration must be exactly between 0.4 and 0.5 per cent. Below 0.4 per cent of hypochlorite, the solution is not sufficiently active, and above 0.5 per cent it becomes irritating.

Dakin's solution is made from chlorinated lime, anhydrous sodium carbonate and sodium bicarbonate. The method of preparation and testing is given by Dakin and Dunham,¹¹ and also by Carrell.¹²

The Chlorin Group.—The chlorin group of disinfectants includes a number of important substances such as chlorin itself, hypochlorous acid and its sodium and calcium salts, and organic "chloramins," that is, substances containing chlorin attached to nitrogen in the form of NCl groups. They are all characterized by marked instability, since in disinfection they react not only with the cell constituents of microorganisms, but also with most other substances which are apt to accompany bacteria. In so reacting, the active chlorin of the antiseptic is eventually converted either into inert chlorids or into inert organic substances in which the chlorin has become united to carbon. Thus, in using the chlorin germicides, as with other readily oxidizable substances, the process of disinfection will go on only so long as some of the active substance remains undecomposed. The action is rapid and transitory.

Antiformin.—Antiformin is the patented name of a disinfectant which was introduced in 1900 by Victor Tornell and Axel Sjöo of Stockholm as a cleansing material for fermenting vats in breweries, but it is only since the investigations of Uhlenhuth and Xylander¹³ in 1908 that it has come into prominence in bacteriological and sanitary work.

Antiformin consists of equal parts of liquor sodæ chlorinatæ of the British Pharmacopeia and a 15 per cent solution of caustic soda. The formula for the liquor sodæ chlorinatæ is as follows:

Sodium carbonate	600
Chlorinated lime	400
Distilled water	4,000

Dissolve the sodium carbonate in 1,000 c.c. of the distilled water; triturate thoroughly the chlorinated lime in the remainder of the water; filter; mix the two and filter again.

Antiformin has a strong germicidal action in weak solutions (2 to 5 per cent), killing ordinary cocci and some bacilli rapidly, five minutes at most

¹¹ Dakin and Dunham, *Handbook of Antiseptics*. The Macmillan Co., 1917.

¹² *J. Am. M. Ass.*, 1916, 67: 1777.

¹³ *Berl. klin. Wchnschr.*, 1908, 65: 29.

being sufficient. In this respect antiformin acts more rapidly and surely than either of its component parts used alone. It has, however, very slight action upon the tubercle bacillus, the smegma bacillus, and other organisms belonging to the acid-fast group.

Antiformin is an almost colorless liquid, with a strong odor of chlorin, and is strongly alkaline. It keeps fairly well without particular precautions being taken. It has deep powers of penetration, owing to its ability to dissolve and render homogeneous the various substances in which bacteria are often found, such as sputum, feces, pus, urinary sediment, and even small pieces of tissue.

The germicidal action of antiformin is doubtless due to the energetic oxidizing properties of the chlorinated lime. The fact that it does not kill the tubercle bacillus and other acid-fast organisms seems to be due to the biochemical nature of these bacilli. The fatty or waxy capsule which is present and which gives them their acid-fast property acts as an impervious coat, resisting the dissolving action of the antiformin, and so protects the protoplasm of the bacilli from its germicidal action. The tubercle bacillus may be isolated in pure culture by exposing tuberculous sputum to a 20 per cent solution of antiformin for twenty-four hours at room temperature or four to six hours at incubator temperature. The bacilli may then be thrown down by centrifugalization, washed free of alkali, and then planted upon solidified egg or other suitable culture medium, or injected into susceptible animals.

While antiformin is therefore a very active germicide for the ordinary bacteria it cannot be depended upon for the acid-fast group.¹⁴

Bromin and Iodin.—Bromin and iodine are very potent germicides. They have about the same value as chlorin, both in their gaseous state and in solution. The tincture of iodine is now much used in surgery for the disinfection of the skin. The use of iodine as a skin disinfectant introduced by Stretton,¹⁵ in 1909, has marked value for this purpose. A 2½ per cent solution is usually strong enough and alcohol is the best solvent. Seventy per cent alcohol is preferable to stronger spirit, and it is important to use pure alcohol, as otherwise iodo-acetone and other products are apt to be formed, which are very irritating.

For *dichloramin-T*, *halozone* and other chlorin compounds, see page 1027.

FORMALDEHYD SOLUTION

Formaldehyd solution (*aqua formaldehydæ* of the United States Pharmacopeia) is known by the trade name of formalin. This is a very valuable disinfectant with a wide range of usefulness in general practice. It is superior to bichlorid of mercury for many purposes, especially as its action is not

¹⁴ *J. Med. Research*, 1910, 22: 315.

¹⁵ *Brit. M. J.*, Aug. 14, 1909; also May 22, 1915.

seriously retarded by the presence of albuminous matter. Formaldehyd is not injurious to most articles, and it is not very poisonous. It is a true deodorant.

Formalin consists of a 40 per cent solution of the gas formaldehyd (HC-HO) dissolved in water. The liquid is a clear solution, giving off an appreciable odor of the gas. It is exceedingly irritating, but not especially toxic. Formalin solutions are rather unstable. There is a constant loss by evaporation if the liquid is not kept well corked, and in cold weather the formaldehyd polymerizes and precipitates in one of its polymeric forms—trioxymethylene. For the description and uses of formaldehyd gas see page 1372.

The commercial solutions known as formalin are said to contain 40 per cent of formaldehyd gas. They are not always up to standard (average 36 per cent), and, being volatile, there is a certain loss if not well kept. In winter there is a decided deterioration, owing to the polymerization and precipitation of trioxymethylene. This substance is often found in abundance at the bottom of the bottle or carboy as a white precipitate. For these reasons it is well to use an excess of the liquid in practical work if the exact strength of the formalin has not recently been determined.

Formalin solutions of commerce are almost all acid in reaction, due in part to formic acid. Some of the commercial solutions also contain a certain amount of wood alcohol (about 10 per cent) which is added to increase their solubility and stability.

Formaldehyd does not attack copper, brass, nickel, zinc, and other metal substances. It causes no diminution in strength of textile fabrics and has no bleaching or other deleterious effects upon colors. Formalin renders leather, furs, and skins brittle as a result of the union that takes place between the formaldehyd and the organic matter of these articles, and they should therefore be disinfected by another process.

A 10 per cent solution of formalin in water is about the equivalent of a 1 to 500 solution of bichlorid of mercury, or superior to a 5 per cent solution of carbolic acid. It must be borne in mind that in speaking of a solution of formalin a solution is meant of the liquid containing 40 per cent formaldehyd; that is, a 1 per cent solution of formalin would contain that liquid in proportion to 1 to 100, but would contain the substance formaldehyd in the proportion of 1 to 250.

Fecal masses are deodorized almost instantly by a small quantity of formalin, and are disinfected in a short time when intimately and thoroughly mixed with a 10 per cent solution. It is advisable to continue the contact one hour to insure complete action.

There are discrepancies as to the percentage of formalin solution necessary to accomplish trustworthy disinfection in general practice. Taking into account the deterioration of the solution with age and allowing an excess as an element of safety, a 10 per cent solution is recommended. It may be used to disinfect urine, excreta, sputum, and other similar substances. It is also useful in public health work for the disinfection of precious articles, antiques, books and small objects of great value.

DYES

A large number of dyestuffs possess germicidal properties, although until recently they have been employed for the destruction of blood parasites, such as trypanosomes, rather than bacteria. Many germicidal dyes are selective or specific in action.

Malachite green, in conjunction with mercuric chlorid, is used extensively, especially in naval service. Certain other dyes chemically related to malachite green (triphenylmethanes) possess definite bactericidal action, as hexamethyl violet—also known as crystal violet—hexethyl violet and brilliant green.

Acriflavin, trypaflavin, or flavin, is diamino-10-methyl-acridinium chlorid. It was first prepared by Benda¹⁶ at Ehrlich's instigation in 1911, and was found to have marked germicidal action on trypanosomes. Acriflavin has been claimed by Browning and his associates¹⁷ to be a most powerful germicide. It is used in surgery and has also been employed in the disinfection of the nasopharynx of carriers of the meningococcus. Dakin and Dunham¹⁸ regard acriflavin as distinctly more active under most conditions than either malachite green or brilliant green, though its rate of disinfection is decidedly slow. One of the most remarkable properties of acriflavin is that its germicidal action is apparently enhanced by admixture with serum, though greatly diminished by pus.

ACIDS

Acids in sufficient concentration are very effective germicides. An amount of acid which equals 4 per cent of normal hydrochloric acid is sufficient to prevent the growth of all kinds of bacteria and to kill many.

The germicidal power of an acid depends upon the free hydrogen ion in solution. The disinfecting power of an acid is therefore proportional to the hydrogen-ion concentration, and this in turn is proportional to the dissociation of the acid. Thus, when we speak of a strong acid, we mean one that is highly dissociated, and, conversely, a weak acid one which is slightly dissociated. The mineral acids are more corrosive and also more germicidal than the organic acids because they have higher hydrogen-ion concentrations. A 1 to 500 solution of sulphuric acid kills typhoid bacilli within one hour. Hydrochloric acid is about one-third weaker, and acetic acid weaker still. Citric, tartaric, malic, formic, and salicylic acids are about equal to acetic acid. Salicylic acid and salicylates, benzoic acid and benzoates, boric acid and borates possess weak germicidal action and feeble antiseptic powers. They are used as mild disinfectants in medicinal washes and as antiseptics for preserving foods. Boric acid destroys the less resistant bacteria in a 2 per cent solution and inhibits the others. For medicinal purposes it is ordinarily used in saturated solution.

¹⁶ *Berl. Deutsch. Chem. Gesell.*, 1912, 45: 1787.

¹⁷ *Brit. M. J.*, 1917, 1: 73.

¹⁸ *Handbook of Antiseptics*, The Macmillan Co., 1917, p. 64.

ALCOHOL

Alcohol has both antiseptic and germicidal properties. In solutions of 1:1,000 the growth of some bacteria is somewhat delayed. Many microorganisms grow abundantly in 40 per cent alcohol and some in stronger solutions. Dry bacteria may be exposed to absolute alcohol for twenty-four hours without losing their vitality, while 60 to 70 per cent alcohol has definite germicidal power to both dry and moist microorganisms. The explanation of this curious phenomenon seems to be that alcohol fails to penetrate the microbe unless in the presence of water. Under 40 per cent the germicidal action is very slow so that the limits of alcohol as a disinfectant may be placed between 50 and 70 per cent. In this strength it is equivalent to about 3 per cent carbolic acid, provided there is little or no albuminous matter present.

Dakin and Dunham found that most vegetative forms of bacteria may be killed fairly readily by 50 per cent alcohol, but that much higher or lower strengths are less effective, while most spores are unaffected by alcohol of any strength.

Alcohol precipitates protein, which therefore seriously interferes with its germicidal property. Many germicidal substances which are potent when dissolved in water, have comparatively little effect when dissolved in strong alcohol. This is due to changes in dissociation constant.

Krönig and Paul found that phenol dissolved in 98 per cent alcohol was devoid of bactericidal action when tested against spores. It is therefore clear that alcohol is not a desirable solvent for phenolic disinfectants.

ETHER AND CHLOROFORM

The vapors of both ether and chloroform possess slight but definite action. An exposure to ether vapors of one to forty-eight hours was necessary to sterilize agar slants on which pyogenic organisms were growing.¹⁹ Ether is irregular in its action, for good contact with the organisms is difficult to secure. Chloroform is sometimes used as an antiseptic to preserve antitoxic serums.

SOAPS

Ordinary soaps have but limited disinfecting power. According to von Behring the germicidal power of soaps depends upon their alkalinity, but Serafini more correctly points out that the free alkali present, even in concentrated soap solutions, is so small in amount that it can exert no disinfecting action whatever, and that neither the alkali nor the fatty acid, nor the combination of the two is the effective agent.

Walker²⁰ tested chemically pure soaps prepared from pure fatty acids and

¹⁹ Topley, *Brit. M. J.*, 1915, 1: 237.

²⁰ *J. Infect. Dis.*, 1924, 35: 557.

found that, although they differed markedly, none of them killed *Staphylococcus aureus*, which limits the use of soaps as germicidal agents.

Unfortunately, the disinfecting power of soap solutions is not marked enough to make them trustworthy germicides, despite their great value as detergents. The common commercial soaps, especially the colored soaps, are frequently of very poor quality, containing rosin instead of fat, and are not to be depended upon. The soft soaps should also be avoided on account of the presence of all the impurities of the fat and alkali from which they are made. There are other conditions which render the use of soaps uncertain, the chief of which is the hardness of the water.

The action of soap solutions is much influenced by the temperature, which is easy to understand when we recall the powerful germicidal action of hot water alone. It has been shown that soap, even in strong solution and with prolonged exposure, cannot be trusted to destroy such frail organisms as typhoid, cholera, or the micrococci of suppuration. Therefore soaps alone cannot be depended upon for the disinfection of objects and clothing; but in conjunction with certain compatible chemicals, and also with the mechanical cleansing which always accompanies their application, soaps have a wide and varied usefulness in sanitation.

Soap solutions should always be made with soft water. The addition of one of the caustic alkalies, as lye, increases their germicidal and detergent value. The solution should be strong, containing not less than 10 per cent of soap, and the water should be as hot as possible and applied with mops or brushes.

Medicated soaps are for the most part a snare and delusion so far as any increased germicidal action is concerned. In fact, the addition of carbolic acid, bichlorid of mercury, and other substances which have the property of combining chemically with the soap seems actually to diminish the disinfecting value of the substance. As a rule a very small quantity of the disinfecting substance is added to the soap, and when we call to mind what an exceedingly small quantity of soap is generally used for the ordinary washing of the skin and the further dilution of this small amount by the water used it is easy to understand that medicated soaps as ordinarily applied cannot have an energetic disinfecting action.

Conover and Laird²¹ tested formaldehyd and cresol soaps and found they did not destroy test organisms in the length of time usually employed to wash surfaces by the ordinary procedures of employing soap and water followed by rinsing.

An exception seems to be the soap devised by McClintock, in which a mercury salt exists unchanged and active. He found that double iodid of mercury answers this purpose in the proportion of 0.05 to 2 per cent. A solution containing 1 per cent of the soap was found by him to be fatal to pus cocci, cholera, diphtheria, and typhoid bacilli in one minute. This soap

²¹ *Am. J. Pub. Health*, 1922, 12: 602.

does not attack nickel, silver, aluminum, steel instruments, or lead pipes, and does not coagulate albumin.

The value of soap consists in the removal rather than the killing of germs; that is, the cleansing properties are more important than the germicidal action. In hand washing, all the bacteria are not removed by soap and water, and the exposure is not long enough nor the concentration strong enough to disinfect. Therefore, the washing must be followed by the use of an effective germicide in order to render infected hands safe.

As bacteria and spores do not propagate on soap, the danger of infection from using bar soap is remote.

CONVENIENT FORMULÆ FOR DISINFECTING SOLUTIONS

Bichlorid of Mercury—Corrosive Sublimate

Bichlorid of mercury.....	1 dram	1 gram
Water	1 gallon	1 liter

Mix and dissolve. Label "*Poison!*" This is approximately a 1 to 1,000 solution. One ounce of this solution contains very nearly half a grain of corrosive sublimate. Useful for disinfecting clothing, the hands, the surfaces of walls, floors, furniture, etc. Not serviceable for feces or material containing much organic matter.

The addition of HCl or NaCl renders the solution more stable and more active.

Formaldehyd Solution

Formalin	13 ounces	100 c.c.
Water	1 gallon	1 liter

This solution, known by the trade name of formalin, contains 40 per cent formaldehyd. A 10 per cent solution, as above, is useful for the disinfection of clothing and a great variety of objects. It has no corrosive action and does not bleach pigments or rot fabrics. It is useful for the disinfection of feces.

Milk of Lime.—Slake a quart of freshly burnt lime, in small pieces, with three-fourths of a quart of water, or, more exactly, 50 parts of water by weight with 100 parts of lime. A dry powder of slaked lime (calcium hydroxid) results. Prepare the milk of lime shortly before it is to be used by mixing 1 quart of this dry calcium hydroxid with 4 quarts of water. Air-slaked lime is worthless. Slaked lime may be preserved some time if inclosed in an air-tight container. Milk of lime is especially useful for the disinfection of feces; an equal quantity should be added to the mass and thoroughly mixed.

Carbolic Acid

Crude carbolic acid	7 ounces	50 c.c.
Water	1 gallon	1 liter

The solution is facilitated by dissolving in hot water. This makes approximately a 5 per cent solution. The addition of from 12 to 14 ounces of common salt to each gallon increases its germicidal power, especially when used for the disinfection of excreta. The crude carbolic acid is more powerful than pure phenol, but can only be used for rough work, such as floors, feces, sputum, etc. For the disinfection of clothing phenol should be used in a 2½ per cent solution.

Chlorinated Lime ("Chlorid of Lime")

Chlorinated lime	3 ounces	30 grams
Water	1 gallon	1 liter

Mix. This is about a 3 per cent solution. It is exceedingly powerful and is useful for the disinfection of excreta, privy vaults, cesspools, and many other purposes. It is an active bleaching agent and destroys fabrics in this concentration.

GASES

A germicidal gas would be an ideal weapon for destroying such invisible foes as we have to deal with in public health work, especially for terminal disinfection. By reaching all portions of a room or confined space a gas lessens the risk of overlooking any surface upon which the infective agent may be lodged, but an efficient gas for this purpose is still to be discovered.

There is practically only one gas suitable for general application, viz., formaldehyd. It is not poisonous, does not injure fabrics, colors, metals, or objects of art and value. Formaldehyd, however, has distinct limitations. It is unstable and requires a certain temperature and humidity to be effective.

Sulphur dioxid is too destructive for fabrics, colors, and metals for general use. It is a better insecticide than germicide. It is very poisonous to all forms of animal life, which makes it valuable in fumigating against insect- and animal-borne diseases. It is used for the fumigation of the holds of ships, cellars, sewers, stables, and other rough structures infested with vermin.

Hydrocyanic acid gas is too poisonous to use in the household, and is limited in practice to the destruction of vermin on board ships, in warehouses, greenhouses, granaries, railroad cars, and other uninhabited or isolated structures. It has no germicidal power.

The very poisonous and destructive nature of chlorin gas contracts its usefulness to narrow limits.

None of the gaseous agents can be depended upon for more than a surface disinfection. They all lack the power of penetration. Practically all the gaseous agents are not disinfectants but fumigants.

For the distinction between fumigation and disinfection see page 1314, and for the use of gases for fumigation see page 1370.

Preparation of the Room.—The preparation of a room or space to be fumigated with a gas is a matter of some importance. A larger amount of gas

than is thought possible is lost through leaks, by diffusion, by absorption and in other ways; therefore the room should be made tight, all cracks and crevices should be well closed by pasting paper over them or by caulking with suitable material of some kind. Do not forget to close the registers, flues, hearths, and ventilators, and look carefully for openings in out-of-the-way places. Then expose the objects in the room so that the gas may have ready access to all the surfaces. Move bureaus, beds, and furniture away from the walls; open doors of closets, drawers of bureaus, lids of boxes, and the like so that the gas may freely enter and diffuse to all corners. *None of the gases can be depended upon to disinfect clothing, bedding and fabrics.*

While the articles in the room must be arranged so that the gas may freely gain access to all surfaces possible, the mistake must not be made of going to the opposite extreme of disarranging the contents of the room too much, for the same surfaces should be exposed to the gas that were exposed to the infection.

The conditions found in actual practice are so variable that we must allow for a liberal excess to make up for inevitable wastage. Wind pressure also seriously influences the efficiency of gaseous disinfectants in a confined space. Much more air than is commonly thought possible forces its way through cracks and through the walls themselves. The wind pressure may thus drive the fumigating gas entirely away from one side of the room. It is only necessary to stand upon the leeward side of a structure being fumigated with sulphur dioxide or formaldehyd to realize the great quantity of gas blown from the enclosure. (See page 268.)

Formaldehyd.—Formaldehyd is the most generally useful and one of the best disinfecting gases that we possess. Its superiority depends upon its high value as a germicide, its non-poisonous nature, and upon the fact that it is not destructive. The secret of successful disinfection with this substance is to obtain a large volume of the gas in a short time under correct conditions of temperature and moisture.

Formaldehyd (HCHO) exists in at least three well-recognized isomeric states:

1. Formaldehyd (formic aldehyd) is a gas at ordinary temperatures, colorless, and possessing slight odor, but having an extremely irritating effect upon the mucous membranes. At a temperature of about -20°C . the gas polymerizes into paraformaldehyd, known commercially as paraform.

2. Paraform is a white substance, unctuous to the touch, soluble in both water and alcohol. It consists chemically of two molecules of formaldehyd. It is this substance which is supposed to compose the commercial solutions of formaldehyd known as formalin, formol, etc.

3. Trioxymethylene is formed by the union of three molecules of formaldehyd. It is a white powder giving off a strong odor of the gas. It is but slightly soluble in alcohol and water.

Formaldehyd unites readily with the nitrogenous products of decomposition, forming new chemical compounds which are both odorless and sterile.

It is thus a true deodorizer in that it does not mask one odor by another still more powerful, but forms new chemical bodies which possess no odor.

Formaldehyd is rapidly absorbed from all parts of the gastro-intestinal tract and lungs, and may be excreted again by them. It is rapidly oxidized in the body to formic acid and carbonates. There is also a small amount of a dialyzable compound formed in the blood which is most probably hexamethylenamin since the latter is found in the urine. Small amounts of formaldehyd may pass through the body without causing apparent inflammation, while large amounts always cause some.

Formaldehyd Gas.—Formaldehyd gas possesses about the same specific gravity as air; it diffuses slowly, although somewhat better than sulphur dioxid. Formaldehyd combines with nitrogenous organic matter. A few drops added to the white of an egg will prevent its coagulation by heat. The formaldehyd unites with the albumin to form a totally new compound. Combined with gelatin it keeps that substance from liquefying. It is from this property of combining directly with the albumins forming the protoplasm of the micro-organisms that formaldehyd is supposed to derive its power as a germicide. It is perfectly plain, therefore, why there must be direct contact between the gas and the germ in order to accomplish disinfection.

Formaldehyd apparently has no detrimental effects upon silks, woolens, cotton and linen. It does not change colors, with the exception possibly of a slight effect upon some of the delicate anilin lavenders. An oil painting is not perceptibly altered after prolonged exposure to the gas. The metals are not attacked. It is this non-destructive property of the gas that renders it generally applicable. It is practically the only gaseous germicide which can be used in the richest apartments, containing objects of art and value, without fear of damage.

A certain amount of heat and moisture is necessary to obtain successful disinfection with formaldehyd gas. The exact amount of moisture necessary depends somewhat upon the temperature. As a general working rule it may be stated that *if the temperature is below 65° F. or if the relative humidity is below 60 per cent the results become irregular; much below these figures the results are unreliable, especially if the space is both cold and dry.* Formaldehyd polymerizes at low temperatures, therefore in cold weather it may be necessary artificially to warm the room to be disinfected. In dry weather moisture should be added to the room.

Formaldehyd gas cannot be depended upon to accomplish more than a surface disinfection. Under ordinary circumstances it possesses small powers of penetration. The gas polymerizes in the meshes of the fabric and is deposited as paraform upon surfaces. Large quantities of formaldehyd are lost by uniting chemically with the organic matter of fabrics, especially woolens, which further hinders its penetration. Therefore, formaldehyd gas cannot be relied upon to disinfect fabrics, especially quilted goods and materials requiring deep penetration.

Bacteria exposed directly to the action of a concentrated volume of for-

maldehyd gas are killed almost instantly, but in practical work it is necessary to prolong the time of exposure to six or twelve hours, as it takes considerable time for the gas to permeate to all the corners and dead spaces of a room. Bacteria are not always directly exposed upon the surface of objects, as they are in laboratory experiments, and, furthermore, they are frequently imbedded in albuminous matter or in dust, both of which retard the action of the gas.

Formaldehyd is a differential poison, being more toxic to bacteria than to higher forms of life. It is a feeble insecticide. The gas seems to have no effect whatever upon roaches and bedbugs, even after prolonged exposure to very high percentages. As a differential poison formaldehyd gas is a very remarkable substance. It destroys bacteria almost instantly, but, while it is irritating to the higher forms of animal life, it is not very toxic. I have repeatedly found that roaches and other insects with strong chitinous protection seem unharmed after twelve hours' exposure to very strong percentages of the gas in air-tight disinfecting chambers. Mosquitoes may live in a weak atmosphere of the gas over night. They are killed, however, if the gas is brought in direct contact with them in the full strength and time prescribed for bacterial disinfection.

When a weak insecticidal gas is used, it is much more difficult to obtain direct contact between the gas and the insects than between the gas and germs, because the sense of self-preservation aids the former in escaping from the effects of the irritating substance. Mosquitoes and other insects hide in the folds of towels, bed clothing, hangings, fabrics, and out-of-the-way places where the formaldehyd gas does not permeate in sufficient strength to kill them. Mosquitoes have a lively instinct in finding cracks or chinks where fresh air may enter a room or other places where the gas is so diluted that they escape destruction. Therefore, formaldehyd gas, as well as other culicides, cannot be trusted to kill all the mosquitoes in a room which cannot be tightly sealed. On account of its feeble action, formaldehyd should not be used as an insecticide.

Formaldehyd gas in watery solution, known as formalin, is useful for the destruction of flies. Small quantities of dilute formalin (1.25 to 2.5 per cent) placed in saucers about the room attract flies. They drink the fluid, which soon kills them.

It was formerly the custom to neutralize the gas with ammonia, but this is little practiced now. The ammonia neutralizes the formaldehyd by the production of hexamethylenetetramin.

Formaldehyd may be released from its watery solution by any actively oxidizing agent. Potassium permanganate is the best, for it liberates the largest volume of gas, and in the shortest time; but the following may be substituted: bleaching powder, unslaked lime, sodium dichromate, barium oxid. Permanganate liberates about 60 per cent of the gas; dichromate, about 30 per cent, and bleaching powder about 25 per cent. In the case of bleaching powder only about 3 per cent of the total gas set free is chlorin.

The Permanganate-Formalin Method.—Use 500 c.c. of formalin and 250

grams of potassium permanganate for each thousand cubic feet of air space. The permanganate is first placed in a bucket or basin and the formalin poured upon it. An active effervescence takes place and considerable heat is evolved; therefore a pail of sufficient capacity, and especially of sufficient height, should be used to prevent splashing or boiling over. In board of health work it is advisable to have galvanized iron pails made for this purpose with a flaring top. The floor should be protected against the heat by placing the bucket upon a brick, board, or other suitable device.

When the permanganate of potassium and formalin are brought in contact very active oxidation takes place, with the production of formic acid and heat. It is the heat that liberates the formaldehyd gas. Chemically, therefore, the method is a wasteful one, but practically a very serviceable one. It was first described by Johnson of Sioux City, Iowa, in 1904. In the same year Evans and Russell of Augusta, Maine, used the method.²²

The Dichromate Method.—The formula recommended by the Pennsylvania Department of Health is:

Sodium dichromate	10	ounces
Formalin	16	"
Commercial sulphuric acid.....	1½	"

The sulphuric acid can be added to the formalin and the mixture kept on hand for use; polymerization in cold water can be avoided by the addition of glycerin, 1½ ounces. The acid formalin is poured on the crystals of sodium dichromate and formaldehyd gas is at once liberated.

The Formalin-Lime and Aluminum-Sulphate Method.—This method was first described by Walker of the Department of Health, Brooklyn, New York. It is somewhat slower than the potassium permanganate method, but otherwise appears to be just as efficient.

The proportions for each 1,000 cubic feet are as follows:

<i>Sol. A.</i> —Aluminum sulphate	150	grams
Dissolved in hot water.....	300	c.c.
<i>Sol. B.</i> —Formalin (40 per cent CHOH).....	600	c.c.
<i>Lime.</i> —Unslaked lime	2,000	grams
Mix solutions A and B and pour upon the lime.		

In practical work 20 to 25 pounds of the commercial aluminum sulphate is dissolved in 5 gallons of hot water. This is sufficient to mix with 15 gallons of a 40 per cent formaldehyd solution and then used in the proportions as stated above. The lime should be freshly burned, broken into small particles, and should slake rapidly in cold water. The lime is placed in a large bucket. The formalin and aluminum sulphate solutions should be mixed and poured over the lime. In a few minutes the lime begins to slake and the heat evolved drives off the formaldehyd gas.

²² 14th Ann. Rep., Bd. Health, Maine, 1906.

The barium-formalin method was developed by Candall,²³ and consists of one pint of formalin (solution of formaldehyd U. S. P.) and one and one-half pounds barium oxid containing not less than 78 per cent barium dioxid for each 1,000 cubic feet to be disinfected.

Bleaching-Powder Method.—Place in a mixing pan 12 ounces of chlorinated lime, 4 ounces of water; stir to a paste. Then pour 1 pound formalin over the moistened lime. This is sufficient for 1,000 cubic feet.

The Spraying Method.—Spraying formalin is a satisfactory and simple method of disinfecting small inclosures, such as wardrobes, closets, and cabinets. It is not practical for larger rooms. If the formalin is sprayed directly upon the objects to be disinfected they enjoy the direct germicidal action of the substance in solution and, further, are bathed in the gas which is slowly evolved. The method is particularly serviceable for the disinfection of bureau drawers, closets, valuable books and manuscripts, jewelry, objects of art, etc. When used to disinfect small rooms suspend a bed sheet from a line stretched across the middle of the room. An ordinary bed sheet presenting a surface of about 2 by 2½ yards is required for every 1,000 cubic feet of space of the room. Properly sprinkled this will carry, without dipping, 8 ounces of formalin. The ordinary sprinkling pot used by florists can be used to spray the sheets and a liberal excess should be used. The room should remain closed not less than eight hours.

The other methods for disinfecting with formaldehyd gas are not described because most of them are unreliable, and none of them is as serviceable in practical work as the formalin-permanganate method or the formalin-lime method.

Sulphur Dioxid.—Sulphur dioxid (SO_2) is not an efficient germicide, but is exceedingly poisonous to mammalian and insect life. It is this property which makes it of especial value as a fumigant against diseases spread by rats, mice, flies, fleas, mosquitoes, etc.

The action of sulphur dioxid as a germicide depends upon the presence of moisture. The dry gas is practically inert against bacteria. Sulphur dioxid cannot be depended upon where penetration is required. Its action is merely upon the surface. It does not kill spores.

The disadvantages of sulphur dioxid as a disinfecting agent are such as to contract its application to rather narrow limits. It bleaches all coloring matter of vegetable origin and many anilin dyes. It attacks almost all metals; it acts upon cotton and linen fabrics so as seriously to weaken their tensile strength, especially if starched.

Cold water takes up more than thirty times its volume of sulphur dioxid. The solution contains sulphurous acid (H_2SO_3), and it is in reality this acid that is the disinfecting agent. The dry gas is therefore inert and moisture is essential in order to obtain any germicidal effect. It is also this acid and some sulphuric acid which has such a destructive effect upon fibers, colors, and metals. The corrosive action of these acids upon fabrics takes place

²³ U. S. Nav. M. Bull., Oct., 1917, p. 519.

slowly, and the damage may largely be obviated if they are washed at once. Metal work may be protected by coating it with a thin layer of vaselin or heavy-bodied oil.

Sulphur dioxid may readily be condensed into a clear liquid by either cold or pressure or a combination of both. At ordinary temperatures it liquefies if the pressure is raised to about four atmospheres, that is, 60 pounds. This liquid is a stable substance when kept well sealed and protected from the action of the air. It rapidly volatilizes when poured into an open vessel. It is now found in commerce and is one of the methods used for producing the gas for fumigating purposes.

One pound of sulphur when burned produces approximately 1 per cent of the gas. Five pounds will generate approximately 5 per cent, which is the maximum theoretical amount obtainable by burning sulphur in a confined space.

The amount of moisture necessary to convert sulphur dioxid into sulphurous acid is readily computed. It will be found that one-fifth of one pound of water should be volatilized for each pound of sulphur burned. The water may be added in the form of steam or in the form of a finely divided spray, or it may be vaporized by the heat generated by the combustion of the sulphur itself. The latter method is the one that commends itself in practical use, and is described under the "pot method."

While moisture is essential for the germicidal action of sulphur dioxid, it is not necessary in order to kill insects and small mammals. Dry sulphur dioxid is quite as efficacious against rats, mice, fleas, flies, mosquitoes, bedbugs, roaches, etc., as the moist gas.

In fumigating with sulphur dioxid it is necessary to seal the compartment tightly. The gas is disengaged so slowly that it may escape through cracks and crevices as fast as it is formed.

There are three well-recognized methods of fumigating with sulphur dioxid, viz., (1) the pot method, (2) liquid sulphur dioxid, (3) sulphur furnace.

The Pot Method.—The pot method is at once the easiest, cheapest, and probably most efficient method of using sulphur dioxid. The only materials required are iron pots and some sulphur. The best way to apply the method is by placing the sulphur in large, flat, iron pots known as Dutch ovens. Not more than 30 pounds of sulphur should be placed in each pot. The sulphur is preferably used in the form of flowers of sulphur. If it is in sticks or rolls it should be crushed into a powder, which may conveniently be done by placing the sulphur in a stout box and pounding the lumps with a heavy timber. The pot holding the sulphur should be placed in a tub of water, as shown in Figure 155. Although the specific gravity of sulphur dioxid is greater than that of air, when hot it rises, aided by the upward current produced by the burning sulphur. Hence the pots should not be on the floor, or at the bottom of the hold in the case of vessels, lest the cold gas settle and the flame, being deprived of oxygen, be extinguished before all the sulphur is burned. The

pots may therefore be placed upon a table or box or, in the holds of ships, upon piles of ballast or on the "tween decks."

Roberts and McDermott²⁴ suggest that the sulphur be burned upon pans arranged upon a rack, as shown in Figure 156, instead of pots. The advantages of this stack burner are that a large amount of sulphur may be more quickly burned in less time than is possible with the pot method. Further, the intense heat below each pan in the stack burner aids the complete and rapid burning of sulphur in the pans above it. A stack burner will burn sulphur of too poor a quality to give any satisfaction in the pots. The ground sulphur is placed in the pans, the surface of the sulphur is moistened with alcohol, and ignited.



FIG. 155.—THE POT METHOD OF BURNING SULPHUR.

The sulphur may be lighted by means of hot coals or a wood fire, but the most reliable way to get it well lighted is by alcohol, turpentine, or kerosene on a pledget of waste. Make a little crater of the sulphur, soak liberally with alcohol, and ignite. The sulphur then burns in the center, and as it melts runs down from the sides and forms a little lake at the bottom of the crater. If the sulphur is heaped up in a mound in the pot the flame is liable to go out.

Upon the principle of not putting all our eggs in one basket, it is best to have a number of pots when a large compartment is to be fumigated. A pot should contain not more than thirty pounds of sulphur, and the pots should be well distributed in various portions of the place to be disinfected.

The maximum amount of sulphur that can be burned in a given space is five pounds for each 1,000 cubic feet. Four pounds per 1,000 cubic feet and an exposure of six hours is necessary to kill lice; three pounds per 1,000 cubic feet and an exposure of six hours is sufficient for rats, mice and fleas; and two pounds per 1,000 cubic feet with an exposure of one hour will kill mosquitoes, flies and other frail insects.

Liquid Sulphur Dioxid.—Liquid sulphur dioxid, while efficient, is about ten times as expensive as burning sulphur by the pot method. It has the advantage of liberating a large volume of the gas rapidly, thereby facilitating its dispersion, and avoiding the danger of accidental fire.

One pound of burning sulphur will produce about two pounds of sulphur dioxid: $S(32) + O_2(32) = SO_2(64)$. Therefore two pounds of the liquid sulphur dioxid is necessary to produce the same volume of sulphur dioxid as is generated from one pound of the burning sulphur.

The method of using the liquid sulphur dioxid is very simple. If the substance is bought in small tins it is only necessary to cut the lead pipes in the tops of the necessary number of cans and invert the latter in an ordinary

²⁴U. S. Pub. Health Rep., 1911, 26: 403.

washbowl or iron pot, when volatilization rapidly occurs. All the cans must be cut simultaneously and the operator must act quickly and be prepared immediately to leave the room and shut the door. If the substance is contained in glass or metallic siphons the necessary amount of liquid sulphur dioxid can be projected from the outside through a pipe passed through the keyhole or other aperture. A suitable receptacle should be arranged on the inside to catch the drip and frozen mass which forms as a result of the expansion.

The Sulphur Furnace.—The sulphur may be burned in an apparatus of special construction known as a sulphur furnace, from which the resulting fumes are blown through a system of pipes into the room or hold of a vessel to be disinfected. Two forms of sulphur furnace are used: (1) the Kinyoun-Francis furnace, and (2) the Clayton furnace.

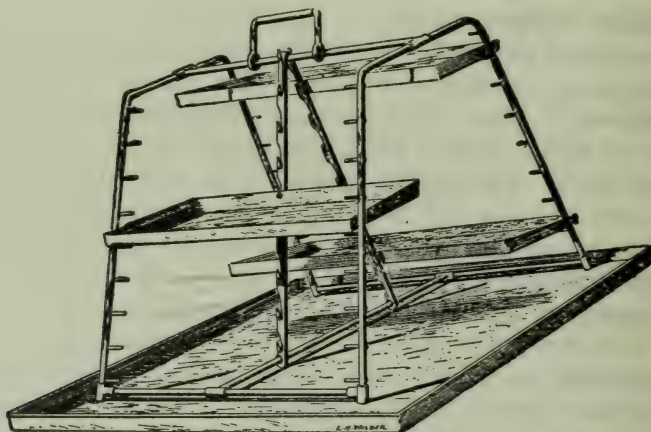


FIG. 156.—LARGE STACK BURNER FOR SULPHUR, WITH 15 OF THE 18 PANS REMOVED TO SHOW CONSTRUCTION.

This method requires expensive and cumbersome machinery and has little to recommend it over the simpler pot method except that a large percentage of the gas may be blown into a given space. The pot method at best cannot produce an atmosphere containing more than 4 per cent of sulphur dioxid, whereas it is theoretically possible to charge a confined space with a higher percentage of the gas by means of the furnace. In practice this is not possible without burning a great excess of sulphur and by expending a very long time, for the reason that the fumes first entering mix with the air and as the gas continues to flow into the space it displaces about an equal quantity of this mixture of sulphur dioxid and air, so that, as a matter of fact, in actual practice only about $2\frac{1}{2}$ to 6 per cent of the gas is usually obtained in the holds of vessels by the sulphur furnace.

It is advisable in using the sulphur furnace to arrange the pipe admitting the gas into the room as near the floor as possible. In disinfecting the holds

of vessels the pipe is usually let down the hatchway until it is near the bilge. The heavy gas collects at the bottom and gradually ascends, displacing the air, so that it is important to allow an opening of some sort for the exit of the air near the top of the compartment that is being fumigated. This opening should not be closed until the gas escapes freely.

The *Kinyoun-Francis furnace* consists of an iron pan upon which the sulphur is burned. The air enters through a valve arranged to regulate the amount of flow. It then passes over the burning sulphur in the direction shown by the course of the arrows to the fan. Fumes are compelled to take a devious course around the baffle plates and angle irons, as shown in the drawing, in order to insure complete combustion and to arrest sparks. The fumes are sucked to the fan, which is actuated by a steam engine or electric motor, and which forces the gas through the pipes to the place to be disinfected.

Running the fan at too high a speed may cause overheating of the pipes or the carrying over of sparks of burning sulphur. The proper amount of air should be carefully regulated so as to obtain complete combustion and the maximum amount of sulphur dioxide gas.

The *Clayton furnace* is a more compact apparatus than that just described. The sulphur dioxide is passed through a series of tubes surrounded by water, an arrangement corresponding in all respects to the tubular condenser of a low-pressure steam engine. The Clayton furnace is furnished with a Root blower, and has the advantage that a comparatively large volume of sulphur dioxide may be pumped rapidly through pipes of small caliber without fear of overheating or fire. These furnaces are being installed upon ships for the purpose of fumigation at port and during the voyage for the destruction of rats, mice, and vermin. It is also an efficient fire extinguisher.

The action of sulphur dioxide is discussed in detail on page 269.

Chlorin.—Chlorin is a germicide of considerable power. It has little practical usefulness as a fumigant, owing to its poisonous and destructive action. At best chlorin, like all gases, is but a surface disinfectant.

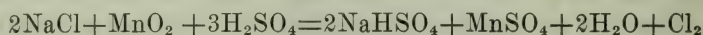
Chlorin is an extremely irritating gas, and great care must be observed in its employment, for the inhalation of very weak proportions of the gas produces serious irritation, resulting in spasm of the larynx, bronchitis, and even in death. Chlorin is heavier than air (sp. gr. 2.47) and tends to fall. Therefore the vessel generating the gas should be placed in an elevated position in order to obtain anything like effective diffusion. Carpets, curtains, and fabrics generally are injured by its action, and the element is a very active bleaching agent.

The germicidal action of chlorin depends upon its great affinity for hydrogen. So strong is this affinity that it combines with the hydrogen of water in the presence of light, liberating the oxygen in its nascent state. Its germicidal action is therefore more one of oxidation than chlorination. The value of chlorin as a deodorant depends upon its power of decomposing the offensive gases of decomposition such as sulphurated hydrogen and volatile ammoniacal compounds.

The compressed chlorin may now be obtained in cylinders from which the gas may be released for fumigation of holds, rooms and other spaces. Another convenient method of generating chlorin gas is by decomposing $1\frac{1}{2}$ pounds of calcium hypochlorite with 6 ounces of strong sulphuric acid. This produces sufficient gas for the fumigation of 1,000 cubic feet of air space, or the gas may be generated from:

Common salt	8 ounces (240 grams)
Manganese dioxid	2 " (60 ")
Sulphuric acid	2 " (60 ")
Water	2 " (60 ")

The following reaction takes place:



Mix the water and the acid together and then pour the mixture over the salt and manganese dioxid in a glazed earthenware basin. The basin should rest on sand or in water.

Fisher and Proskauer have shown that in ordinary dry air 5.38 parts of free chlorin per 1,000 cubic feet of air space appear to be necessary to kill microorganisms. If the air is moist only 0.3 per cent by volume in each 1,000 cubic feet of air is sufficient, disinfection being completed in 5 to 8 hours.

Free chlorin is much less useful than sulphur dioxid, since it is more difficult to control, more dangerous to manipulate, and more destructive in its effects.

Chlorin gas finds its greatest usefulness in the disinfection of water, for which purpose it is reliable. See page 1031.

Oxygen.—The disinfecting power of oxygen depends largely upon the physical state in which it exists. For instance, oxygen in the air has comparatively feeble germicidal properties when compared with nascent oxygen or ozone. The germicidal action of oxygen depends upon its very active property of combining chemically with the albuminous matter of the cell protoplasm. While most bacteria require the free oxygen of the air for their growth and multiplication, there is a large class of organisms (the anaërobes) to which the oxygen of the air acts like a poison or strong antiseptic.

Ozone.—Ozone is an allotropic form of oxygen containing three atoms of that element instead of two. In sufficient concentration it is a powerful germicide and has lately been found of practical use in the sterilization of water on a large scale for the use of cities and towns. It has also been used for the sterilization of bandages and other objects. There is not sufficient ozone in the air normally to exert any appreciable oxidizing or disinfecting properties. It requires at least 13 parts per million in the atmosphere to exert a definite effect upon bacteria; even then the action is not penetrating. Such quantities are harmful to man.

Ohlmüller²⁵ demonstrated that ozone in considerable strength was incapable of killing dry bacteria within the time limits of his tests. Jordan and Carlson²⁶ and also Konrich²⁷ found that ozone ranging from 3 to 4.6 parts per million exerts no surely germicidal action, and that the alleged effect of ozone on the ordinary air bacteria, if it occurs at all, is slight and irregular even when amounts of ozone far beyond the limit of human physiologic tolerance are employed. Parks and Kenwood²⁸ state that it requires 0.3 per cent of ozone to destroy *B. typhosus* in one hour.

Human beings are injuriously affected by amounts of ozone far less than are necessary to produce even a slight bactericidal effect. Ozone, therefore, has no place in practical disinfection of occupied places.

The exaggerated claims of the deodorizing properties of ozone are not justified. Ozone masks disagreeable odors without destroying them. In this way ozonizing machines can conceal faults in ventilation while not correcting them. These conclusions have been reached by Jordan and Carlson, Erlandsen and Schwarz, Hill and Flack, and Konrich, Sawyer, and others. The New York State Commission on Ventilation found that ozone failed to destroy body odors in the recirculated air of a school.

Recently ozonizers have been placed upon the market for the purpose of purifying the air of rooms; these must not be regarded as substitutes for ventilation. Not only may the ozone itself be harmful, but the higher oxides of nitrogen may be formed when the electric current acts upon moist air. See also pages 788 and 791.

For the use of hydrocyanic acid gas, pyrethrum and other fumigants, see Insecticides, page 267.

²⁵ *Arb. u. d. k. Gsndhtsamte.*, 1893, 8: 229.

²⁶ *J. Am. M. Ass.*, 1913, 51: 1007.

²⁷ *Ztschr. f. Hyg.*, 1913, 73: 443.

²⁸ *Hygiene and Public Health*, Blakiston's Sons & Co., 7th Ed., 1923, pp. 155, 215.

CHAPTER IV

METHODS OF DISINFECTION

In public health work the things most frequently needing disinfection are feces, sputum, and other discharges from the body; bed and body linen, and other fabrics; bedrooms and the hands. The disinfection of water and the pasteurization of milk have already been considered. The disinfection of ships is described under Quarantine. The following examples are selected.

Feces.—The disinfection of feces is most important because these discharges are most dangerous and at the same time most difficult to render safe.

From patients the discharges should be received in a glass or impervious vessel containing some of the germicidal substance, more of which is added afterwards, and the mass thoroughly disintegrated and mixed. The breaking up of the masses and mixing is best done with a little stick which is then dropped into the mess. The mixture should stand at least two hours before the contents are disposed of, kept well covered meanwhile, and the vessel given a thorough cleansing and disinfection before it is again used. At least an equal quantity of the germicidal solution should be used to the mass disinfected and enough should always be added entirely to submerge all particles. Excreta must always be protected from flies and other insects, even while undergoing disinfection.

It is necessary to emphasize the importance of breaking up all masses until they are completely disintegrated, and mixing thoroughly with the germicide. It is almost impossible for any of the ordinary germicides to penetrate particles of even moderate size, within a reasonable time; emulsions do not penetrate at all, and therefore should not be used to disinfect feces.

It is always desirable to use a generous excess of germicidal agent, both as to strength and amount, in disinfecting feces. The following methods are recommended:

Lime and Hot Water.—A simple and effective method for the disinfection of feces, such as typhoid stools, consists in adding enough hot water to cover the mass in the receptacle, and then adding about $\frac{1}{4}$ of the entire bulk of quicklime.¹ A large cup of lime is about enough for an average stool. The mass must be disintegrated and the receptacle covered and allowed to stand for two hours.

¹ H. Linenthal and H. N. Jones, *Month. Bull., Bd. Health Mass.*, 1914, 9: 50. *Boston M. & S. J.*, Jan. 8, 1914.

In addition to the germicidal action of the lime there is enough heat generated by the hydration of the lime to destroy typhoid and similar microorganisms. It is important to start with hot water from 50° to 60° C. and the mass will then be heated throughout to 80° or 90° C.

A bucket of boiling water (about 1 gallon) will disinfect a single stool when other germicidal agents are not obtainable. The vessel should be covered and allowed to stand until cool. Sufficient heat is thus had to destroy practically all bacteria except the spore bearers.

Milk of Lime.—Use freshly prepared milk of lime containing 1 part by weight of the freshly slaked lime to 4 parts of water. Add at least an equal quantity to the amount of material to be disinfected, disintegrate and allow the mixture to stand no less than two hours before final disposal. The perfunctory sprinkling of fecal matter with lime or milk of lime, as is often done, is not effective. Lime should not be thrown into the hoppers of water-closets for the disinfection of dejecta, for otherwise a thick mass may accumulate and obstruct the pipes. In disinfecting excreta with lime the reaction of the resulting mixture must be alkaline else the object will not be attained.

Lime or milk of lime is useful for the disinfection of privies, or trenches in camp, or in country practice. For its use under these circumstances the amount required may be arrived at as follows: The amount of fecal matter per person is reckoned at 400 grams a day. If the urine is also to be disinfected this may be counted as 1,500 to 2,000 c.c. per person daily. For the disinfection of the solid excrement alone 50 grams of lime, or 400 c.c. of the milk of lime (1 to 8), must be reckoned for each person per day. If the urine is included it will take four to five times as much. The mixture must have an alkaline reaction. Attention is again called to the fact that air-slaked lime is inert.

Chlorinated Lime.—This is one of the most useful and potent germicidal substances for the disinfection of feces. Use at least a 3 per cent, better 5 per cent, solution and an amount at least equal to the mass to be disinfected. Thoroughly mix and allow to stand at least two hours. Chlorinated lime is rendered inert by organic matter; therefore an excess should always be used. It is also converted to the inert carbonate upon exposure to the air (see

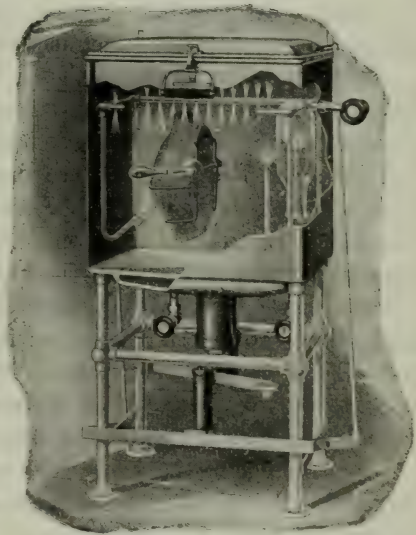


FIG. 157.—STEAM STERILIZER FOR BED PANS.

page 1026). Chlorinated lime may be liberally sprinkled on the fecal mass, water added, and the mixture then stirred.

Formaldehyd Solution.—A 10 per cent solution may be depended upon to disinfect feces if thoroughly incorporated with the mass and allowed to stand at least two hours. As a deodorant it acts almost instantly.

Carbolic Acid.—A 5 per cent solution of crude carbolic acid added to an equal bulk of excreta may be depended upon to disinfect in two hours, provided the germicide is thoroughly incorporated throughout the mass.

The *cresols* as “tricrosol” and liquor cresolis compositus or lysol are valuable agents for the disinfection of fecal matter in small amounts on account of their energetic action and because their efficiency is not greatly impaired by the presence of albuminous matter. They are used in strengths of 1, or better, 2 per cent.

Steam.—In hospital practice, bedpans and their contents are steamed and cleaned in special apparatus. See Fig. 157.

Sputum.—The discharges from the mouth and nose not alone of the sick, but of well persons, are often laden with infection. This is one of the frequent means by which disease is transferred. The proper disposal of sputum and its efficient disinfection are therefore important public health measures. It is a good rule to require the discharges from the mouth and nose of all patients to be received upon small pieces of gauze or paper, or in individual cups which may subsequently be burned.

The most trustworthy chemical disinfectants for sputum are carbolic acid, 5 per cent; formaldehyd solution, 10 per cent or stronger; chlorinated lime, 5 per cent. The methods for the disinfection of sputum correspond to those described for feces. Sputum offers special difficulties on account of the mucus which is readily coagulated and hard to penetrate.

Sputum should be kept well covered in suitable receptacles until it is disposed of. Germicidal solutions may be used in bedside cups or in cuspidors, but are not necessary.

The disinfection of the large amounts of sputum such as that collected in hospitals, public buildings, and other places is a difficult and disagreeable task. On account of its dense consistency it prevents the penetration of chemical solutions. A very good apparatus for the disinfection and disposal of sputum in hospitals, sanatoria, etc., consists of an autoclave in which the material is steamed under pressure and at a temperature of 120° C.; after the completion of the process the disinfected mass is washed through the drain into the sewer by water entering the autoclave. The entire operation can thus be conducted under cover. Wm. J. Manning² describes an ingenious and efficient method of handling spittoons and disposing of the sputum at the Government Printing Office in Washington. The cuspidors are self-draining. They are collected and handled by devices so that the attendants do not have to handle them directly.

² *J. Am. M. Ass.*, 1909, 52: 829.

Hands.—Hands soiled in nursing or in attendance upon a patient sick with a communicable disease should be immediately disinfected. They should first be soaked in liquor cresolis compositus, 1 per cent, or bichlorid of mercury, 1:1,000, in order to destroy surface infection. They should then be thoroughly washed in soap and water and finally again immersed in the germicidal solution. Special attention must be given to the spaces about the finger nails. It is not possible to sterilize the deeper portions of the skin, and ordinarily this is not called for. The skin may be disinfected with tincture of iodine, using 7.5 per cent for adults and 3 per cent for children. It is painted on the skin and left until it dries, and then washed off with 75 per cent alcohol.

Surgical purposes require a much more thorough process. After washing with soap and hot water for five minutes with vigorous use of the nail brush, the hands are soaked in 85 per cent alcohol for one minute, scrubbed with a sterile brush, then soaked in a 1:1,000 bichlorid of mercury solution for two minutes. This is the well-known Furbinger's method. Care must be taken not to allow the bichlorid solution to come in contact with soap, as the two form a nearly inert compound.

The hands should always be washed after a visit to the toilet and always before eating or handling food. Typhoid carriers must be especially careful.

Bed and Body Linen.—Fabrics, such as towels, napkins, handkerchiefs, sheets, pillowslips, underwear and similar articles, should always be disinfected after contact with any of the communicable diseases, for they are very apt to become infected. They may be steamed or boiled or immersed in a germicidal solution such as phenol 2.5 to 5 per cent; liquor cresolis compositus, 1 per cent; formaldehyd solution, 10 per cent; or bichlorid of mercury, 1 to 1,000.

Special care is necessary in washing or disinfecting towels, sheets, underwear, and other fabrics soiled with such discharges as pus, blood, or excreta. If they are heated or boiled without special precautions they will become indelibly stained by the coagulation of the albuminous matter, which becomes fixed in the fiber.

Soiled wash may be treated as follows: It is wrapped in a sheet wet with sublimate solution, and this is placed in a sack likewise moistened with a germicidal liquid. The sack is placed unopened in a solution containing 3 per cent of soft soap and heated to 50° C. for three hours and left in the same solution for forty-eight hours after it cools. If not soiled with albuminous matter the wash may be immersed in a solution of bichlorid of mercury, 1 to 1,000, with the addition of common salt. After this preliminary disinfection the articles are boiled half an hour in a water containing:

Petroleum	10 grams
Soft soap	250 grams
Water	30 liters

Rooms.—The disinfection of a living-room calls for all the resources of the disinfectors' art, and requires the ingenuity and often the vigilance of the operator.

Certain articles commonly found in living-rooms, such as bedding, carpets, rugs, cuspidors, upholstered furniture, and other objects liable to become infected must be treated separately by some process applicable to each article. It is usually best to remove fabrics and articles for special treatment. Then wash the floor and surfaces with a strong hot germicidal solution. Finally, give the room a house-cleaning, sunning and airing. It may then be renovated with paper and paint.

None of the gaseous disinfectants can be depended upon. Formaldehyd gas is sometimes used as a preliminary. The surest method of handling carpets, rugs and other bulky fabrics is by steam sterilization, after which they can be gone over with a vacuum cleaner and finally hung in the sun for a day or two. If carpets, rugs, upholstered furniture, mattresses, pillows, quilts, or other articles have become badly contaminated with infected discharges the soiled areas should be thoroughly saturated with a strong formaldehyd solution. Bed and body linen may be boiled, steamed, or immersed in one of the germicidal solutions. Such articles removed from the room should be wrapped in a bag or in a sheet wet with bichlorid of mercury. Rubbish that is collected in the room should be gathered and burned. The cuspidors and their contents should be steamed or soaked in strong carbolic solution or chlorinated lime. Door knobs, bed rails and other surfaces and objects handled by the patient or soiled with discharges should be wiped with bichlorid or carbolic solution.

The method used for the purification of a room will vary somewhat with the infection for which the disinfection is done. Attention should be focused upon the places, objects and surfaces liable to infection.

For the preparation of a room for fumigation, see pages 268 and 1370.

Stables.—The disinfection of a stable requires a particularly thorough application of all the resources at the hand of the disinfectors. The conditions met with in a stable render its disinfection doubly hard, not only on account of the accumulation of organic filth which has worked into the many crevices, but on account of the high resistance of anthrax and tetanus spores, for which stables are sometimes disinfected. In addition to these diseases stables require disinfection on account of tuberculosis, glanders, pleuropneumonia, foot-and-mouth disease, and various diseases of man as well as those of the domestic animals.

It is advisable to give the stable a preliminary fumigation, preferably with sulphur, in order to destroy surface infection and the vermin which always infest these places. The preliminary fumigation is especially important in the case of plague and glanders, not only to prevent the spread of the infection, but as a safeguard for the disinfectors. Then remove all small articles that need disinfection. The blankets should be wrapped in moist bichlorid sheets and boiled, steamed, or immersed in a strong germicidal

solution. Buckets, currycombs, brushes, stall tools, and other equipments that have been in contact with the sick animals or with infectious materials should be mechanically cleaned with a hot carbolic solution in which they may be allowed to soak over night. Metallic and wooden objects or utensils should be given a thorough preliminary cleansing with a stiff brush and hot water and soap, and then boiled or immersed in a 5 per cent solution of carbolic acid or 2 per cent solution of cresol for several hours. Leather articles, as harness or equipment, should receive a similar preliminary cleansing and be scrubbed with either a strong solution of bichlorid of mercury or carbolic acid.

All hay and grain should be removed from the racks and mangers and all bedding from the floors. After its careful collection at some designated point this refuse should be saturated with petroleum and destroyed by fire.

The stable must now be soaked with a strong germicidal solution applied with a hose or splashed on all surfaces by means of mops. The floors, corners, and stalls must be saturated with the solution. On account of the presence of so much albuminous matter carbolic acid or one of its derivatives is preferred for this purpose to chlorinated lime or sublimate solutions. Chlorinated lime is useful if used in sufficient concentration and generous amounts. Now scrape out the débris from all the cracks in the floors and walls; collect it for burning. Then clean the woodwork with hot lye or a strong alkaline soap solution and follow with another general hosing with the germicidal liquid. Follow with washing, airing and sunning.

After several days' exposure to air and sunshine the interior of the stable should receive a fresh coat of whitewash, applied quickly, and prepared from freshly burnt lime.

The watering troughs are very apt to be infected, especially in dealing with glanders. In all instances not only the troughs and watering buckets should be disinfected but the water remaining in them, for often there is no drain or sewer, and this water poured on the ground may be a source of subsequent infection. The water may first be disinfected by the addition of a suitable amount of chlorinated lime or any of the standard germicides. The troughs are then to be mechanically cleaned, thoroughly removing all organic matter, and then applying a strong germicidal solution to both the inside and outside. For metal-lined troughs the use of bichlorid of mercury is, of course, inapplicable, and for such carbolic acid, alkaline creosotes, bleaching powder or formaldehyd solution is recommended. Most germicides are poisonous, and must therefore be finally washed out of the troughs or buckets by flushing with fresh water and then airing in the sunlight before they are again used.

Sometimes the ground in the immediate vicinity of the stable will need attention. Lime or the gasoline torch will generally be found most useful for this purpose. Carcasses and excreta are to be disinfected and disposed of according to the methods given under these titles.

Books.—With the exception of their exposed surface, books cannot be disinfected in the bookcase or on the shelves of houses and libraries. However, if the books have not been handled or exposed to infection in any way except by their presence in the sick room there is no reason for considering any part of the book, except the exposed surface, as infected.

Books which have been handled by the patient or which have been otherwise exposed to infection require particular care in their disinfection on account of the difficulty of penetrating between the leaves. Books used in public libraries are often regarded with suspicion, and many librarians require that they should be sunned, aired, or disinfected before they are again issued. The danger from this source has doubtless been exaggerated. Books, however, which have been handled by persons suffering with one of the readily communicable diseases should always be disinfected before they are again used. Patients sick with acute communicable diseases should be given magazines and cheap prints which may be burned.

Books may be disinfected in a specially constructed chamber by means of heat and formaldehyd gas. They must be arranged to stand as widely open as possible upon perforated wire trays. Under these conditions the exposure should be continued twelve hours with high percentage of formaldehyd and a moist-air temperature of 80° C., a partial vacuum having first been introduced. The binding, illustrations, and print of books are not injured by this process.

When only a few books are to be treated in the absence of a special apparatus they may be disinfected by placing 2 or 3 drops of a 40 per cent formaldehyd solution on every second page, taking care to distribute the drops well. The book is then laid in a closed box or drawer in which more formalin has been sprinkled, and left in a warm place for not less than twenty-four hours.

Pamphlets and unbound volumes may be steamed without serious harm. Steam is not applicable to the disinfection of bound books on account of the glue and leather.

Beebe³ recommends dipping the books in a solution of carbolic acid and gasoline. After immersion the books should be placed before an electric fan, which rapidly drives off the gasoline.

Nice⁴ recommends the use of moist, hot air at 80° C. and 30 or 40 per cent humidity for thirty-two hours for the disinfection of books. This is said to destroy all non-spore-bearing bacteria in closed books, even tubercle bacilli in thick layers, without injuring the most delicate bindings.

Surgical Instruments.—The disinfection of surgical instruments varies with the nature and use of the instrument and the character and region of the operation. Ordinarily, boiling in water containing 1 per cent sodium carbonate for one minute is sufficient. This removes fat and prevents rusting.

³ *Am. J. Pub. Health*, 1911, 1: 54.

⁴ *J. Am. M. Ass.*, 1912, 58: 1201.

A good method of disinfecting and preserving surgical instruments is to keep them in liquor cresolis compositus. They are taken out of this, washed in alcohol and then in sterile water just before use, after which they are carefully cleansed and replaced. Alcohol, about 70 per cent, is often used to disinfect delicate instruments. Fortunately, a clean surgical instrument is comparatively easy to disinfect.

Thermometers.—A thermometer may be the source of conveying disease from one person to another, and it behooves the physician to exercise special care concerning its cleanliness and disinfection. The best practice is to keep a pure formaldehyd solution or 70 per cent alcohol in the thermometer case in which the instrument is kept constantly bathed.

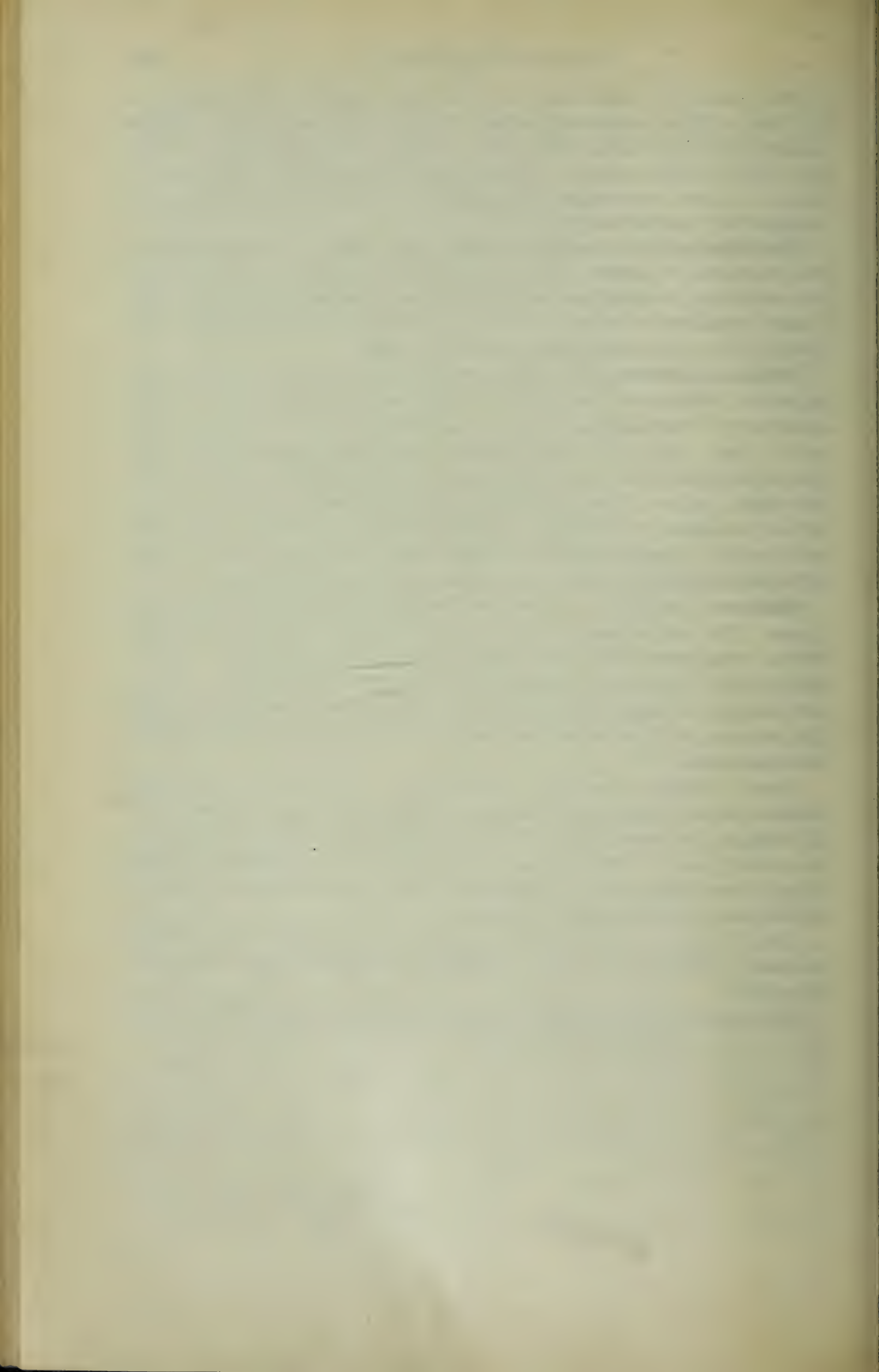
Wells and Cisterns.—The disinfection of a well may be accomplished by the use of freshly burnt lime. About half a barrel is thrown into the well, stirred up with the water, and the walls are scrubbed down with the resulting milk of lime. The well is then pumped out, cleaned, allowed to refill, and a second supply of lime added, after which the well is allowed to stand twenty-four hours. After a thorough stirring the solution is then pumped out and the well is allowed to refill and is reëmptied until the water is practically free from lime. Instead of lime chlorinated lime may be used for this purpose, sufficient being added to make approximately a 1 per cent solution.

Cadavers.—Dead bodies are seldom the cause of spreading communicable diseases. The body without previous washing should be wrapped in a sheet wet with a strong germicidal solution, such as bichlorid of mercury, 1 to 500, carbolic acid, 5 per cent, or cresol, 1 per cent, until it is disposed of. Should it be desirable to wash the body it should be done with formaldehyd solution (10 per cent) or Labarraque's solution, or one of the germicidal solutions above mentioned.

From a sanitary standpoint bodies dead of one of the communicable diseases are best disposed of by burning. When cremation is not practicable the body may be surrounded by twice its weight of freshly burnt lime in an hermetically sealed coffin and buried at least six feet underground. There is much less danger from the spread of disease from bodies buried in the ordinary way than is commonly supposed.

Embalming with strong solutions of formaldehyd and arsenic that are commonly used for this purpose is effective in destroying all but the surface infection.

The disposal of bodies dead of anthrax is an important and difficult matter and has been discussed on page 1272.



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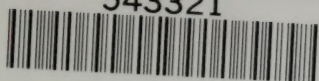
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